TEXT BOOK

OF

VETERINARY MEDICINE

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VETERINARY MEDICINE.

CONTAGIOUS (ZYMOTIC) DISEASES.

Contagious or Infectious Diseases are such as are transferred from animal to animal by an infinitesimal microbe or a parasite, which propagates itself in or on the body of the victim. This volume is devoted to the microbian diseases in which the essential cause is an infinitesimal microscopic organism introduced from without and propagating itself in the body as similar organisms do in fermenting liquids or solids. The parallel with fermentation has given rise to the Greek name *zymotic*. As there can be no fermentation without the living organized ferment, so there can be no zymotic disease without the pathogenic microbe. The pathogenic microbes are divided into those belonging to the vegetable kingdom—**bacteria**, and those belonging to the animal—**protozoa**. All however pertain to the very lowest forms of their respective kingdoms, nearing the line on which the two kingdoms meet.

The microbes establish their pathogenic action in a variety of ways of which one or two examples may be named. Some, like the *bacillus anthracis* are so avid of oxygen that they rob the blood of that essential element and unfit it for the functions of life. Some like the *trypanosoma*, or the *pyroplasma bigeminum* liquefy and destroy the red globules of the blood, quickly reducing it to a dilute condition unfit for its normal uses. Some like bacillus tetani produce toxins which unite with body tissues or cells or their constituents, and cause disorder or loss of their normal functions and consequent disease.

Infection Atria.

Pathogenic germs may enter the system through the mucous membrane of the air-passages, eyes, alimentary canal, urino-generative organs, mammary glands, etc., and especially through the glandular structures in such membranes, or opening through them. The follicles of the tonsils, solitary and agminated glands

are especially favorable to the reception and culture of such microbes. Again, some may enter through the injured and partly devitalized skin, and even in exceptional cases through the sound integument. Very commonly however admission is gained through wounds, abrasions, or other breaches of surface of either mucosa or skin. These are very varied, but include cuts, bruises, pricks, scratches, chaps, cracks, vesicles, pustules, burns, frost bites, eschars, cauterizations, sloughs, the open umbilicus of the new-born, the wounds made by biting or stinging insects, etc., the wounds made by external parasites or internal worms, and those due to the poisons of venomous animals. The action of insects in carrying infection from a diseased animal or product to a sound one, and the conveyance of germs on or in the bodies of worms, which hook themselves to vascular surfaces, or migrate inward through the membrane, deserve especial mention as these factors have been so much forgotten and overlooked.

Certain microbes pass from the circulation of the dam to that of the fœtus, with or without colonization and disease of the chorion. Others fail to pass through the placenta to the fœtus.

Some microbes (bacillus tetani, bacillus of emphysematous anthrax) are confined to the seat of inoculation and perish in the blood.

Anærobes grow only in the depth of the tissues, where exudation has diminished or arrested the circulation of oxygenated blood. They die in the blood. *Ærobes* grow on or near the surface, in the oxygenated blood, or where uncombined oxygen is present. Some therefore enter most easily by surface wounds, and some by deep wounds only.

Again some genera and species are immune to certain microbes so that an inoculation is harmless to them; the microbes cannot live and form colonies. Animals of susceptible species which have passed through a self-limiting microbian disease show a similar immunity. Some may carry the infecting germ on the surface of the skin or mucosa, and transmit it to other (susceptible) animals, though themselves immune.

Colonization of Different Tissues.

Certain microbes and their products show a particular affinity for given tissues which they colonize to the exclusion of others,

or their toxins unite with the constituents of such tissues so as to abolish or derange their functions. The action may be likened to the election of aloes for the large intestines, jalap for the small, saltpeter for the kidneys, opium or alcohol for the brain. So the microbes of tetanus, dourine and rabies affect the nervous system, those of aphthous fever the mouth, feet and udder, those of glanders, tuberculosis and strangles the lymph system, and those of cowpox the skin.

Elimination of Pathogenic Microbes.

Different microbes may leave the system by any of the natural excreting surfaces. The mucosæ of the bronchia, bowels, or kidneys are the most usual outlets. The kidneys especially, receiving so much blood relatively to their size; habitually pass out microbes which find a home in the blood and thus the kidneys are notoriously subject to secondary local infection and pathologic processes.

Susceptibility: Vulnerability.

Just as certain tissues are receptive of given microbian poisons and succumb to them, so certain animals and species, suffer from particular microbes and their products, when other tissues of the same, or other species of closely allied animals, escape, The latter are said to be immune, and this immunity is racial, or innate on the one hand, or acquired on the other. The recognized and assumed conditions of susceptibility will be more easily dealt with under the head of immunity.

Immunity: Serum Therapy.

Immunity may be defined as the successful resistance, by the animal body, of invading microbes and their products. *Congenital or racial immunity* to given infections which prove pathogenic to others is so common that a large number of infectious diseases have been long recognized as peculiar to a given genus or species. Thus the *horse* has his strangles, influenza and contagious pneumonia; the *ox* his lung plague and Texas fever: the *sheep* its sheep-pox caseous lymph adenitis and braxy; the *pig* its erysipelas (rothlauf) and hog cholera; the *dog* his distemper;

and the *chicken* its apoplectiform septicæmia. *Acquired immunity* is equally familiar as shown in all cases of recovery from a microbian disease, and in the fact that after a first attack of many such diseases there is an acquired resistance to a second attack for a greater or less length of time. This antagonism is in degree only, and too often an overdose, or a specially virulent or potent germ, or a particular condition of the system exposed, will cause the latter to succumb to a microbe, to which it would otherwise have proved immune. In general terms it may be said that, as the microbe and its toxins are the main vulnerant agents in establishing the disease, so the body cells (leucocytes, erythrocytes, tissue cells) and their toxins (antitoxins) are the resistant or immunizing agents. It is an antagonizing of specific pathogenic agents by defensive substances derived from the normal body cells. The attack and the repulse are each made through the soluble chemical products of the living organism (microbe or body cell), and such products are diffused through the serum or lymph. In this may be included the liquid contents of the body cells for when these englobe the invading microbe, the latter, until it perishes, continues to produce its pathogenic products, which are the more concentrated and effective because they are in the main confined within the narrow limits of the cell. Hence many cells are destroyed in the clash of forces, and the result wavers in the balance until decided by the relative numbers or potency of the contending bands of microbes and cells.

Immunization, by products of the disease to be protected against, is initiated by one of two methods: 1st by the use of *enfeebled germs* which will no longer prove fatal to the animal (*protective infection*): and 2nd, by employing the *soluble toxic products* of the pathogenic germ in the absence of that organism itself (*protective intoxication*). Since however the real immediate factor, which operates on the animal cell to cause the formation of the defensive material, is the soluble chemical product of the microbe, it follows that the success in both cases is due to the same cause. The practical difference is that whereas the enfeebled microbes are still vital, and capable of reproduction, they may, under given favorable conditions, start up a new infection which may propagate itself indefinitely as an epizoötic; the same

is not possible with the simple chemical product in solution, which has neither life nor power of self propagation. When, therefore, we aim at the complete extinction of a disease and its infection, and when this can be effected equally well by the use of the simple chemical products, this is much to be preferred to the use of live microbes however much they may be enfeebled.

In seeking a scientific explanation of immunization, many subsidiary factors and complex conditions have been discovered, and on these have been based ingenious theories which must be mentioned.

Phagocytosis (*phagein*, to eat, *cytos*, a cell) is the process in which cells possessed of amœboid movement, lymphoid and neutrophile leucocytes, etc., flow out into arms (pseudopodia) which enclose solids that they digest as food, and among these the invading pathogenic microbes (Metchnikoff). When the englobed microbes are not too numerous in a cell or too toxic, they are themselves dissolved and digested and their destructive powers arrested. On the other hand, the cells often perish and even become for a time centres for the culture and encrease of the microbes within them. This is well shown in the great destruction of the blood globules in Texas fever and other affections.

The *defensive products* of the *phagocytes* include an *enzyme* (ferment) or *cytase* which dissolves the microbe or other body on which it operates: and a *cytotoxin* (*leucotoxin*) which poisons or enfeebles the invading organism so that it may be less able to produce defensive secretions and protect itself. These defensive products of the cell are especially attributable to the lymphocytes whether in the blood or the great lymphatic system, to the amœboid epithelium of the capillaries, and to the wandering lymphocytes, but also in part to the red blood globules, and it is presumed the tissue cells when set free in inflammation, and endowed anew with amœboid movement. The defensive products pass out of the cells into the surrounding serum endowing that with defensive qualities similar in kind. Hence the principle of *serum therapy*.

Hæmolysins are enzymes that cause solution and destruction of blood globules. Injection intravenously, intraperitoneally, or subcutaneously, into a guineapig, horse or other animal of the defibrinated blood or blood serum of the rabbit, determines in

the blood of the injected animal, the formation of hæmolysins which act on the red globules of the blood of the rabbit but not on that of other animals. This operates when the blood serum from the injected horse or guineapig is injected into the circulating blood of the rabbit, or mixed with the rabbit blood *in vitro*, (Bordet). Here we have a suggestion of immunization. The alien and inimical blood of the rabbit in the circulating blood of the horse (or other animal) educes the protective hæmolysin which quickly destroys the alien red blood globules of the rabbit if brought in contact with them. The hæmolysin, however, is composed of two bodies *one* of which (*alexin*, *complement*) is rendered inactive when heated for half an hour to 55° C., and a second (*immune body*, *sensitizing body*, *ambocepter*, *copula*, *fixative substance*, *desmon*, *preparator*) only loses its power at a heat of 60° to 65° C. The serum robbed of its hæmolytic power by heating to 55°, resumes this power if mixed with the blood serum of a healthy guineapig, which would not of itself have affected the rabbits' blood. The *alexin* shows no affinity for the red globule, to which it proves harmless except in presence of the *ambocepter*. Again, *alexin* and *amboceptor* show no affinity at a low temperature but may lie side by side at 0°C. without uniting, and can only do so at a higher temperature up to 40°C. The *alexin* shows no affinity for the cell unless the *amboceptor* is present, but, given the two conditions, affinity between the *alexin* and *ambocepter* and a corresponding affinity between the *ambocepter* and the *cell*, and the microbian toxin can reach the cell and exercise its baneful influence upon it.

Ehrlich carries the subdivision still farther. The *amboceptor* has its *haptophore* group, (*haptein*, to touch, *pherein*, to bring), intermediate between it and the animal cell as an essential element to the completion of the mutual affinity of the two, and the alexin or complement has its *toxophore* group, (*toxos*, poison, *pherein*, to bring), which on that side carries the toxins. Then on the part of the cell he invokes projections of the cell wall in the form of pseudopodia (*receptors*) which must have a special affinity for the *haptophores* in order to establish an effective union with the *amboceptor* and *complement*, and finally with the *toxin* and *microbe*.

The relation of one of these bodies to its fellow is of the nature

of chemical affinity and each must find its proper affinity to become effective, as a halogen or acid must meet with a base in order to form a salt. But just as a given acid and a given base may have no mutual chemical affinity, so a potent toxin may be present and yet prove harmless for lack of the intermediate bodies which are necessary to establish a true affinity with the cell. The simile has been offered of a lock with its wards, the key fitting those wards, and the hand that turns the key, and if the reader will eliminate the mechanical elements of the picture and replace them by chemical affinities, the interdependence of each element on the adjacent one in order to establish a toxic action in the cell, will be fairly represented. The current diagram in which each element is represented as having on one end a mechanical conformation which will fit a corresponding structure on the adjacent element, and can unite with no other, and at the other end a special form which will fit that of another element and no other, is liable to mislead certain minds. A special element in the chain is fitted to connect with two other elements differing from itself and from each other, but, the connection is established through chemical attraction and not by gross mechanical adaptation.

The absence of these affinities may in part explain why certain animals and certain genera are invulnerable by certain microbes and their toxins,—why given diseases are peculiar to given species of animals.

Detachment of Receptors. Under many infections by the affinity for the microbe or its toxins, the leucocytes (the natural defensive cells) accumulate in the seat of the microbian invasion and even in the infected blood, the encrease being due not only to their congregating, but no less to direct multiplication. Their nuclei enlarge at the expense of the surrounding protoplasm and nuclein escapes into the surrounding liquid. According to Ehrlich multitudes of receptors are cast off from the surface of each cell and meeting in the serum with the haptophores of the toxins, unite with these, satisfying their affinity and thus ward off their union with those receptors which are still attached to the cell, and in this way they protect the life and integrity of the cell. This contributes to abort or cut short the disease, and the overproduction of these receptors, once started, tends to continue

for a greater or less length of time and thus establishes an acquired immunity.

It must be assumed that these receptors, before their detachment from the cell have acquired the special affinity which makes them available for engaging with the haptophores and toxins of the invading disease, and of that only, as the acquired immunity is from the one affection only.

Isolysins or *Autolysins* (*isos*, similar, *autos*, one's self) are solvents of the red globules produced by injecting excess of the blood of the same species of animal. They do not act the same on the blood of all animals of the same species, showing a difference in the globules in animals of the same species. This suggests a reason why different individuals are not all equally susceptible to the same microbe, and why the action of antitoxins should be more effective in one animal than another of the same breed.

Agglutinins: Hæmagglutinins. These are elements in the normal and diseased blood-serum, which cause *clumping in masses of the blood globules* of another animal, *or of bacteria.* The action bears an apparent relation to that of hæmolysis and the two conditions usually coexist. Normal goat serum agglutinates the red corpuscles of man, pigeon and rabbit: normal rabbit serum agglutinates typhoid and cholera bacilli. The serum of a person with a well developed case of typhoid fever clumps the bacilli in an artificial culture of the typhoid germ. The same holds with the blood serum of hog cholera, and the hog cholera bacillus. In these cases and in others this is made a mode of diagnosis. The union of the agglutinin and the bacillus is shown by the following: To a serum which agglutinates both typhoid and cholera bacilli, some typhoid bacilli are added and the whole centrifuged when the clear fluid will no longer clump the typhoid bacilli, but still clumps the cholera ones. Conversely if the cholera bacilli are first added and the whole centrifuged the clear fluid no longer acts on the cholera bacilli, but will still act on the typhoid. The agglutinins of the bacilli introduced adhere to the surface of these bacilli and are precipitated with them, while those that would clump the bacilli of the other disease remain in the clear liquid. The same applies to the agglutinins in their action on blood cells. To a normal goat serum, which agglutinates the

red cells of man, pigeon and rabbit, red cells of man were added and the whole centrifuged. The clear liquid left will no longer clump the red cells of man, but will still clump those of the pigeon and rabbit. The agglutinins in the blood of one animal are thus shown to be many, one acting on a given microbe or on the red globules of one animal and another on those of another.

The power of the agglutinins survives a temperature of 60° C. but is lost at 65° C. Thus by heating a serum to 55° C. the hæmolytic action is destroyed while the action of the agglutinins is retained.

Precipitins. If a rabbit is injected with the serum of horse, chicken or eel, and the serum of this rabbit is later mixed with the serum of a horse, chicken or eel, as the case may be, the latter becomes cloudy by reason of the precipitation of a portion of the albumen. The substances in the rabbit's serum which determine the precipitation of a portion of the albumins are known as *precipitins*. Leblanc finds that in the majority of cases the precipitate is made up in part of albumin from the serum operated on, but mainly of pseudo-globulin from the specific serum. The specific serum may operate equally on the serum of two nearly related animals. Thus the serum taken from the rabbit which had been first injected with chicken serum, precipitates the normal serum of both chicken and pigeon. These two animals are supposed to have common receptors.

Without going further in the elucidation of precipitins and their relations, it may be said that in them as in the agglutinins we have to a certain extent a parallelism with the production of antitoxins by the introduction into the living body of the toxins of a given disease. No method for the utilization of the precipitins for protective purposes has been successfully worked out, yet they have been availed of to differentiate the flesh of one animal from that of another for which it has been substituted :—the flesh of the horse from that of the ox, or that of the dog from that of the sheep.

Antibodies. These are protective agents produced by the animal cells, when they are submitted to successive, encreasing doses of the inimical material of microbian or other origin. Thus treatment with *hæmolysins* develops *antihæmolysins*, *isolysins* develop *anti-isolysins*, *agglutinins* develop *anti-agglutinins*, *precipitins*

produce *antiprecipitins*, *immune body* secures *anti-immune body*, *toxins* produce *antitoxins*, *cytotoxins* produce *anticytotoxins*, *spermotoxin* invokes *antispermotoxin*, *neurotoxtin* produces *antineurotoxine*. The operation of each poison rouses the leucocytes and body cells to elaborate its special antidote. The *antibody* for one species of animal, however, or for one poison, is not therefore effective in another animal species nor for another poison. The antibodies cannot be used interchangeably for different diseases or animals.

Immunization by *toxins* and *ptomaines* is a means of rousing the animal cells of the subject injected to the production of the requisite *toxins*, *ptomaines*, etc., (*antibodies*) to prevent the disease from which the poison was drawn. The production of the *antibodies* may be more or less abundant and permanent, and thus immunization is likely to be more or less perfect and enduring. These antibodies may often be cultivated in the bodies of animals which are not naturally susceptible to the disease (which are already innately charged with antibodies) and which can therefore bear much larger doses of the poison, and in this way the most potent defensive serum may be secured. They may, on the other hand, be rendered ineffective on the animal injected, by reason of the absence of the intermediate bodies by which their connection is established, by reason of a special susceptibility to the pathogenic microbes or their toxins, or by reason of the presence of acids or other bodies which increase susceptibility to microbes or toxins.

Two other considerations enter into the question. If the unaltered serum of an infected animal is used containing the living microbe, there is some danger of the survival of the latter, and of its propagation to other susceptible animals, thus starting an epizoötic. Again if the serum is devitalized by heat, oxygen under pressure, or otherwise, there is the danger of destruction of the antibodies, or of some of the intermediate bodies by which their preventive action is established. The devitalized serum is the safe agent to use, but it is not in every disease that it can be secured by available methods, and therefore sterile sera cannot always be availed of.

Protection by *antibodies* produced in the system of a normally immune animal which has been made still more resistant, or

taken from an animal normally susceptible but which has been rendered artificially immune, and long enough after the induced immunity to avoid all risk of left-over microbes, cannot fairly be called *immunization*, seeing that it only lasts so long as these *antibodies* remain in the system of the animal injected. It does not rouse the cells of this animal to themselves produce the required *antibodies* and to continue such production for a length of time. It is comparable rather to a dose of chloral or other antispasmodic in tetanus, which will suppress the spasms so long as the chloral remains in the system, but has its action limited by the amount of the dose and the period up to its elimination. This partakes of the nature of therapeutic medication rather than immunization.

For *immunization* the following conditions may be laid down as most uniformly applicable :

1. The active pathogenic products of the invading micro-organism must be employed.

2. A nonlethal dose must be employed or a succession of such doses.

3. These must be used on an appropriate subject. The injected animal must not be temporarily insusceptible like the meat fed rat to anthrax, nor morbidly susceptible to the same disease like the ox charged with lactic acid.

4. The immunizing product is best injected into the connective tissue. In intravenous injection the toxin may kill at once by undue potency, or may be eliminated so rapidly that it has no tangible or enduring effect.

5. In case of very deadly toxins (Rinderpest) the danger may be lessened or obviated by mixing the antitoxins with the toxins before injection, or by injecting first the antitoxins and later the toxins.

For *serum therapy* the following conditions are important :

1. Use a suitable animal for producing the antitoxins, and raise its resistance to the specific infection by successsive nonlethal injections of the products of the microbes of the disease in hand.

2. Use the resistant serum of the highly immunized animal in appropriate doses for the animal to be treated.

3. The hypodermic use of the serum is usually to be preferred, though in certain cases intravenous injection may be employed, or even injection into cavities containing the organs that have an elective affinity for the toxins.

4. Use the therapeutic serum as early as possible after infection. A short delay means the demand for an enormous encrease of the antitoxic serum to be used, and even then in some diseases (tetanus) it has little or no effect.

5. The introduction of the antitoxin just before the infection, or at least during the incubation stage, when as yet the toxins have not united with the susceptible tissues, promises the best results.

Modifying Factors :—Physiological and Pathological.

Many different conditions of the system disturb, modify or annul the habitual operation of the agencies relied upon for immunization and serum therapy, and although such interference is exceptional, yet its occasional occurrence will interfere in such cases with the full success of the sanitary work based on these methods.

Both immunization and serum-therapy are largely influenced by the supply of oxygen in the freely circulating blood, and, when this has been interfered with by excessive or defective functional activity of the thyroid, anterior pituitary body, or, above all the adrenals, the protective or curative process is materially affected. Sajous, who ascribes the bacteriolytic action mainly to a tripsin body formed in the pancreas and spleen and sent in the blood to the comparatively immune liver, or in the wandering or tissue cells, claims that the abundance of this and other antibodies is determined largely by the supply of adrenalin, which controls the dilatation and contraction of the capillaries and the quantity of blood and oxygen supplied. If the stimulation of the adrenals by toxins is insufficient he advises to add to this alkaline salts, strychnine, digitalis, etc., but there is again the danger of complete arrest of adrenal function by overstimulation. Other examples might be adduced to show that the immunization is at times an uncertain quantity and to enforce the truth that the absolute extinction of the microbe by segregation

and disinfection is to be preferred when it can be availed of at reasonable expense.

Habits of Microbes as affecting Suppression.

As bearing on the suppression and extinction of contagious diseases, the life history of each pathogenic microbe, and particularly its survival as a saprophyte outside the body, or its strict confinement to a life in the animal body (pathogenic career) offers most valuable indications. The microbe which is restricted to an intracorporeal existence in the animal body can be certainly and definitely circumscribed and exterminated, and a disease which has prevailed and decimated a race of animals from the dawn of their existence can be wiped out from the face of the earth. A microbe which can live and multiply not only in the body of an animal host, but also outside in a decomposing organic matter (a saprophyte), can not be met and exterminated on the same easy and certain terms, but must be dealt with in all parts of the outside world wherever there is a concurrence of the suitable dead organic matter, and the pathogenic microbe. It may or may not be possible to utterly exterminate a microbe which possesses this habit of life, but we can usually circumscribe its prevalence and shut it out of given areas, by eliminating it from the animal system within such areas, and putting an end within the protected field to the conditions that favor its saprophytic life. Again, a microbe which is pathogenic when introduced into the animal body, and which may live outside that body as a saprophyte, under conditions which are unusual and which can be successfully altered, stands midway between the two first named as regards its amenability to suppression and extinction. Again, certain microbes, though deadly to their animal host, habitually employ ticks or predatory insects to convey them from one animal host to another. These can be cut short in their pathogenic career by the simple expedient of destroying or excluding the invertebrate host which acts as a bearer between the two higher animals.

The following tables are interesting in this connection :

Diseases due to Pathogenic Microbes : Nonsaprophytic.

Microbes habitually live in the Animal Body only.

Variola.	Strangles.
Glanders.	Contagious Pneumonia.
Dourine	Influenza.
Lung-plague.	Canine Distemper.
Rinderpest.	Rabies.
Rothlauf : Pig Erysipelas.	Foot and Mouth Disease.
Hog Cholera.	Vesicular Exanthema.
Swine Plague.	Tuberculosis.
Swine Fever with Invisible Microbe.	Entero-hepatitis in Turkeys.
Braxy.	Apoplectiform Septicæmia in
Fowl Cholera.	Chickens.

Diseases due to Microbes which can Live as Saprophytes, outside the Body.

Pyæmia.	Hæmorrhagic Septicæmia.
Septicæmia.	Tetanus.
Malignant Œdema.	Actinomycosis.
Emphysematous Anthrax.	Coccidiosis.
Anthrax.	Thrush (Muguet).
	Contagious Ulcerous Stomatitis.

Diseases with Microbes that are Conditionally Saprophytic.

Milk Sickness.	S. African Horse Sickness.

Diseases caused by Microbes borne by Invertebrate Animal.

Texas Fever.	Trypanosomiasis.
Trembling (Louping-Ill).	Paludism.

CYCLES OF POTENCY: PERIODS OF MALIGNANCY AND BENIGNANCY.

Nothing is more certain than the varying potency of a microbe and its toxins for a year or a series of years at a time. For a number of years just past smallpox has been so mild in the United States that physicians often differed in opinion as to whether the prevailing affection was variola or a less dangerous skin disease. Some years strangles are almost uniformly mild and simple and in others almost constantly irregular and dangerous. The same holds true of canine distemper, hog cholera, influenza, contagious pneumonia, lung-plague, Rinderpest, and other pestilences. Even locality and season influence largely. Lung-plague is a less vioent affection in the Northern United States than in Great Britain, and Glanders largely escapes recognition in Wyoming and Montana. Tuberculosis at one time, or in one locality, shows a very low virulence and mortality, and at another time or place an unwontedly dangerous and fatal prevalence. Again, the food, or indoor life of an animal, or other variation of management o environment often modifies the susceptibility to a microbe and therefore its virulence and the fatality it causes. Any such condition prevailing for a length of time determines a special cycle of the disease for evil or good, so that agencies which appear to operate in the way of prevention or cure at one time are comparatively useless at others. This has often led to confident claims for given methods, based on a series of experiments, or on observations made during a given epizöotic which break down completely under a larger experience.

This should never be forgotten in estimating the value of measures which come short of the complete and final extinction of the microbe of a disease. All restriction by special diet, medicine, open air; all attempts at immunization by enfeebled microbes (misnamed vaccine), by sterilized toxin or cytase drawn from such microbes; any use of antidotal agents in the form of antiseptics, antitoxins, etc.; any resort to sero-therapy or organotherapy will perhaps succeed in a long series of cases, and then signally fail under new conditions. But when the one essential

cause of the disease is a particular microbe the complete extinction of that microbe, not only at once cuts short the existing epizöotic, but absolutely excludes the possibility of that disease arising anew. The present losses are promptly cut short, and all future losses, which would otherwise have gone on to the end of time, are absolutely prevented.

And yet such absolute extinction is objected to because of the cost. Which is greater—the present outlay to pay for even 10 per cent. of a susceptible race when this will put an end to all such losses for all future time ;—or the temporizing to save a fraction of this 10 per cent. at the certain cost of the maintenance of the disease and losses year by year for hundreds or thousands of years, together with the equally galling tax for the sanitary police restrictions through all these centuries ?

In spite of the present claims for immunization and other half-way measures, the only truly ideal and economical method is that which destroys every germ of the infection, so that no future cases can possibly occur. This is, however, restricted largely to acute infectious diseases, the germs of which have no saprophytic life outside the body of the host.

For some affections, like foot and mouth disease, the object may be accomplished by stopping all intercourse between animals and herds, the exclusion of man, beast, vermin, birds and insects, and accompanied and followed by an exhaustive disinfection of the premises and every object that may have been exposed even remotely to infection. In all cases the exposure of susceptible animals, or those that have been near them or their products or any unfenced place or within reach of other animals, must be made a penal offence.

For other more fatal affections of this kind, the removal (usually the slaughter) of the infected, the disinfection of the carcases, premises, products and contaminated things, and the expert control and daily testing of the survivors, are important additional measures.

When the cost of expert control for a long period would approach the actual value of the herd, it is often better to buy them out and kill the whole at once so as to purify the premises at once and allow the owner to continue his business and earn a living.

In the absence of violations of the law, or recent importation indemnity for all animals taken, is essential to success; it is a matter of bare justice, and its omission is an invariable source of failure in veterinary sanitary police. In some countries the cost is met by a tax on live stock, but it is much simpler to let such an outlay for the public good be paid out of the public exchequer.

When the germ of a disease is saprophytic it may be impossible to deal with it as above, and then such measures as immunization, serum-therapy, organo-therapy, may be resorted to in suitable eases. But in all such cases any step that would risk the extension of the area of infection must be prevented so that an expert supervision of the stock and their products is de. manded.

Some epizöotics must be prevented by doing away with the intermediate invertebrate bearers of the microbes, and some by removing the conditions of environment by which the microbes are preserved. These and other precautions must be referred to the chapters on individual diseases.

PYÆMIA AND SEPTICÆMIA.

Multiplicity of Septicæmias. Pyæmia and Septicæmia, distinction between. Multiple secondary abscesses in pyæmia; no secondary abscesses in uncomplicated septicæmia. Septico-pyæmia. *Pyæmia.* Causes: bacteria, deep wound infection, susceptibility, debility, shock, illness, narcotic ptomaines, partial immunization, complexity of infection, mutual decomposition of toxins, dose, lesions of intima of blood vessels, thrombosis, embolism, action of microbes on hæmatoblasts and blood globules, viscidity of cells, adhesion to endothelium, coagula, solution of clot, escape of microbes. Lesions: wound, abscess, thrombus, emboli, infarction, abscesses, ulceration, endocarditis. Spleen. Blood coagulates. Symptoms: rigor, hyperthermia, with remissions, pulse weak, soft, rapid, perspiration, diarrhœa, sweet breath, fæces, urine acid, dulness, prostration, mucosæ dusky or yellow, blood shows leucocytosis, hæmolysis, cardiac murmur, arthritis, stupor, coma, palsy, dry, puffy sore, source of infection. Prognosis grave. Prevention: largely surgical, antisepsis, asepsis, excision of infected vein. Treatment: antiseptics, locally and internally, stimulants, tonics, nourishment. *Septicæmia:* microbes, toxins, septic intoxication, septic infection, fermentation fever, sapræmic fever. Lesions: blood dark, incoagulable, spleen enlarged—gorged, petechiæ, cloudy swellings, coagulation necrosis, organs as if parboiled, congested glands, kidneys. Symptoms: chill slight,

hyperthermia without marked remissions, weak, rapid pulse, hurried breathing, anorexia, emesis, yellow mucosæ, nervous disorder—dulness, apathy, stupor, paraplegia. Value of precursory conditions. Prognosis grave. Prevention, Treatment: remove source of poison, antiseptics, drainage, internal antisepsis, strychnia, quinine, iron chloride, stimulants, supporting, easily digestible food, sponging.

At first introduced to indicate the supposed results of pus and septic material respectively in the blood, these terms have come to represent the clinical phenomena which come from the introduction into a susceptible system of pyogenic and necrogenic microbes and their toxic products. Gradually different affections, which would have been included under the same general terms, came to be identified under specific names, and a number of these will be described as individual diseases—strangles, mouse and rabbit septicæmias, metritis, phlebitis, omphalitis, rouget, barbone, chicken cholera, septicæmia hæmorrhagica, etc.—yet a certain number have been left to be referred to under the generic terms, though respectively due to different microbes.

Distinctions between Pyæmia and Septicæmia. *Pyæmia* is a morbid condition characterized by the formation in different organs of multiple metastatic abscesses, dependent on the transference, in the blood stream, of infected clots, or particles containing pus microbes, and their arrest at distant points, so as to cause foci of suppuration commencing with the intima of the vessels.

Septicæmia indicates a general infection often by the same microbes, but showing its results in enlargement and blood engorgement of the spleen and lymph glands and necrotic foci of the liver, kidneys and other organs, but without the formation of multiple abscesses. The presence of the microbes in the different organs affected, shows that it is not due to the diffusion of the toxic chemical products alone, as at one time supposed, and the lack of abscesses appears to be due to the absence of clots or of modified and adhesive leucocytes or hæmatoblasts, which adhering to the epithelium of the vessels predispose to suppuration.

The two conditions are, however, often combined, constituting what is known as *septico-pyæmia*.

As in the occurrence of other infecting diseases, the condition of varying susceptibility must be taken into account, one individual, or one species resisting an inoculation which would be deadly to another.

Pyæmia. *Causes.* The causative microbes are most commonly staphylococcus pyogenes aureus, or albus, the streptococcus pyogenes, and less frequently bacillus pyocyaneus, bacterium coli commune, and probably any pus producing microbe. Many conditions must however be accepted as contributing to the general infection.

Inoculation on a mere abrasion or surface sore is not to be dreaded so much as if the virus is lodged *subcutaneously or in a deep wound.* The ready escape of the toxic products, the active leucocytosis which takes place in the granulations, and the action of the oxygen of the air are more or less protective in the exposed sores.

The native *susceptibility of the subject*,—horse, ox,—conduces to the disease, while the insusceptibility,—bird—tends to obviate it.

The *debility of the system or of individual tissues attacked*, lowers the resistant power, and especially that of leucocytosis, and thus favors survival and encrease of the microbes and their chemical products. Thus the *shock* succeeding a serious operation, the general depression attendant upon severe illness, or the poisoning by narcotizing ptomaines and toxins, may easily become the extra weight which causes the system to succumb. On the contrary, pre-existing or long standing disease, with consequent general debility appears at times to prove to some extent a protective factor, the previous exposure to the invading germ having educated the leucocytes to resist the toxins and to produce the defensive sera which neutralize the latter or keep the invading microbes in check. A measure of immunization has been secured.

The resulting immunization cannot be looked on as very perfect, nor permanent, as a specially strong inoculation by a virulent microbe, or large dose, or different conditions of life, will entirely overcome it, and the pyæmic fever appears. Yet in chronic cases of secondary abscesses from a deep source of infection, the resistance is often such as to ward off febrile pyæmia. In a horse with primary abscess situated deeply under the humerus, free evacuation and healing of the wound, have, in my experience, been followed by the formation of abscesses in distant points for a period of seven years, but without any marked febrile reaction.

The *complex nature of the infection* appears at times to overcome the vital resistance more effectively than will the presence

of even a potent microbe alone. Some of the worst cases follow on a wound, the seat of complex infection, and even saprophytes are to be dreaded in this connection. This may operate in various ways, either by mutual combinations or decompositions of the toxic products of associated germs producing more deleterious products, or by the individual action of one ptomaine or toxin on leucocytes, hæmatoblasts, sera, or tissue, laying it more open to the attacks of those of another microbe which by itself would have been comparatively harmless.

Koch's experiments showed that the *attack is violent in ratio with the size of the dose :* one thousandth part of a drop of pyæmic blood was harmless to the rabbit, while one tenth of a drop killed in one hundred and twenty-five hours, and a syringe full in forty hours.

In ordinary cases of pyæmia the occurence of internal *phlebitis* or *arteritis* with the inevitable **thrombosis** is an important step in causation. Any inflammation of the inner coat of the vessel leads promptly to the formation of a coagulation of the contained blood, and blocking of the lumen. Beginning on the diseased or abraded surface, the clot forms backward along the line from which the blood normally comes (proximal in the arteries ; distal, in the veins), until it reaches the next considerable colateral branch. The clot is firmly adherent to the intima except at the free end, which is conical and projects into the blood current.

If small portions are detached from the thrombus and washed on in the blood stream they become arrested when they reach a vessel too small to admit them, it may be a smaller artery, or it may be a capillary, and always in the line of the circulation,— from the systemic circulation to the lungs, or from the lungs to the system at large. This is **embolism**. Wherever arrested, the contact of the leucocytes and hæmatoblasts with the inner coat of the vessel, leads to metabolic changes and firm adhesion, and the pus microbes in the clot determine suppuration and abscess.

Eberth and Schimmelbusch have shown that the hæmatoblasts, even more than the other blood elements, when acted on by the pus microbes become viscous and stick not only to each other, but to any floating body, and to the inner serous coat of the vessel, particularly when the latter has been abraded or injured. This clumping together of the hæmatoblasts forms white clots which block the smaller vessels, but in the viscous condition they

further the coagulation of the fibrine, and again when they come in contact with the intima, instead of passing through, or moving on, they remain adherent and start the formation of thrombi. This is above all common in given tissues, and the medulla of bone has in this respect a bad preëminence, so that acute suppurative osteomyelitis, is a familiar lesion and is liable to become chronic, and determine distant abscesses and general infection long after.

The thrombus thus formed is an infective coagulum, tending to constant encrease, as the clot is a favorable culture-field for the microbes, and the tendency is to coagulate more and more of the adjacent blood. It tends further to disintegration, as the action of the microbes and their toxins on the leucocytes, transforms these into pus cells, inducing softening of the mass, and the washing on of individual infective pus cells and minute portions of the clot to form infective centres and abscesses at distant parts.

If the pathogenesis of the invading microbe is weak and the resistance of the leucocytes potent, such clots may remain circumscribed or may even be absorbed, but in the opposite conditions with potent and numerous microbes and abundant and effective toxins, the tendency is not only to a continuance of infection, but to an acute febrile pyæmia.

Pyæmia does not supervene at once upon a trauma as may septicæmia, but only after a variable number of days, (3 to 8), a peculiarity which is explained by the temporary protection of the clot. By the constant accretions on its exterior, of the new layers of hæmatoblasts and fibrin, the microbes are at first imprisoned, and it is only when softening has taken place, or when the coagulum has extended into the free flowing current passing into a colateral trunk, that the infection is liable to be washed on in dangerous amount.

The mere presence of pus microbes or their toxins in the blood, does not determine pyæmia: a modification of the intima of the vessels leading to local infections with thrombosis or embolism and abscess is an essential condition. This lesion of the vessels may be a trauma, as from bruise, puncture, operation, ligature, or it may be the extension of a disease process as in arteritis, phlebitis, atheroma, the growth of a tumor from adjacent tissues, or parasitism. The seat of the secondary abscess depends primarily on the location of the original suppurating centre. As such centres

are most commonly in the systemic circulation (osteomyelitis, omphalitis, wounds, traumas) the lungs are most commonly attacked, the pulmonary capillaries acting as a sieve and arresting the floating infective coagula. When the primary infection comes from the chylo-poietic viscera, the liver is likely to show the first crop of secondary abscesses. When, on the other hand, the primary abscess is in the lungs, the great flow of blood through the kidney renders it especially subject to secondary suppurating foci, though these may form in any part of the body.

Lesions. Pyæmia may result from a wound or abscess in connection with which will usually be found a vein containing a thrombus more or less softened or liquefied. If from a deep seated injury or from osteomyelitis, the same condition is met with. The thrombus and circulating blood furnish abundance of the infective microbes, and at distant points, in the complimentary circulation, most commonly in the lungs the arteries are found to be the seats of *embolism* from arrested clots. The arrest always takes place where the vessels are diminished by bifurcation, or the giving off of a considerable colateral trunk, and the appearances will depend on the duration of the embolism. If quite recent, a wedge-shaped mass of tissue supplied by the vessel is ischæmic and pale, its blood passing on into the veins without further arterial supply; if later, this tissue forms an *infarct* being gorged with deep red or black blood which has filtered in from adjacent anostomosing capillaries and distended those of the exsanguine area. This area becomes of a deep red or black color, consolidated by an exudate of lymph, and rapidly invaded by suppuration. The microbes determine suppuration and softening, first in the clot and intima, and next in the outer coats of the vessel and the surrounding exudate, so that an abscess of variable size may result. Abscesses are usually smaller and more numerous in the acute forms of the disease, and larger and less numerous in the more chronic.

Ulcerative endocarditis with coagula on the valves is not uncommon. The spleen is often the seat of small abscesses in the centre of solid exudates, with, in many cases, softening and enlargement of the organ. The blood tends to retain its normal bright red color, and clots firmly, contrary to the usual condition in septicæmia.

Symptoms. The formation of emboli and secondary abscess is usually marked by a violent rigor, lasting from a few minutes to

an hour and which may be repeated at irregular intervals, serving, in some measure, to distinguish pyæmia from septicæmia. The temperature rises with the rigor, (102° to 105°) but shows marked remissions especially in the morning, when it may not exceed the normal, and rising again with the recurrence of chill or staring coat. The pulse is usually encreased in frequency even during the remissions and is soft and compressible. Remissions may be attended by profuse perspirations or even, in the advanced stages, by fœtid diarrhœa. The breath has a peculiar sweetish or mawkish odor. Blood passed with the fæces may indicate intestinal abscess, and albumen or pus in the acid urine, bespeaks suppurating foci in the kidney. The cloudy mucus from the pelvis of the kidney in the horse must not be mistaken for this. Cough or dyspnœa will indicate abscess of the lungs, and intercostal tenderness, pleurisy.

The buccal mucosa may be dry and cracked, and the tongue coated. From the first the animal is dull, and prostrate, and the visible mucosæ become dusky brown or even yellowish from the liberated hæmatin. Blood abstracted will show the microörganism, an excess of leucocytes and diminution of the red globules. The poison determines hæmolysis. A cardiac murmur, usually with the first sound, betrays endocarditis. This is especially characteristic of chronic pyæmia. Again multiple suppurating arthritis may appear. Stupor, coma or paralysis will indicate cerebral or meningeal lesions.

In pyæmia following trauma there is drying up of the pus which becomes serous or bloody, a puffy condition of the granulations, and the evidence of a thrombus in one or more veins leading out from the wound.

In other cases the occurrence of pyæmic symptoms, consequent on parturition, metritis, omphalitis, bone-abscess or osteomyelitis, on suppurating internal inflammations, ulcerative endocarditis, or infective fevers like strangles, influenza, contagious pneumonia, cattle plague, distemper, rouget, hog cholera, etc., serves to identify the disease.

The *prognosis* of pyæmia is always grave, and death may be expected in six to fourteen days in acute cases. Chronic forms last much longer.

Prevention is the great object in regard to surgical cases, and this means the prevention of suppuration in the wound. As far

as possible, however, this is to be sought by asepsis, or the use of weak non-caustic antiseptics only, as cauterized tissues form favorable culture media, when the action of the antiseptic is spent, there being no longer any living and resistant leucocytes present. The early excision of veins, the seat of thrombosis, has proven successful.

In purely medical cases, the seat of the primary suppuration is not always obvious and one is thrown back on medical treatment which is rarely satisfactory in severe cases.

Treatment. When accessible even the secondary abscesses may be opened, washed out with a weak antiseptic (3 per cent. carbolic acid solution), and covered with antiseptic dressing. Antipyretics are worse than useless, because of the resulting depression of the vital powers, and the reduction of the natural powers of resistance. Calomel in small and repeated doses tends to assist in elimination, and to counteract complications through sepsis of the contents of the bowels. Quinine and chloride of iron continued in large doses have been especially relied on as antiseptic tonics. Liberal feeding, if the appetite will admit, is all important to tide the patient over the period of depression. In the chronic cases tepid bathing is of great value (Senn). Senn has great confidence in the stimulating and supporting action of alcoholic liquors—beer, ale, porter and even whisky, and in human beings accustomed to the daily use of these beverages they are more imperative than in the lower animals.

Septicæmia. The micro-organisms causing septicæmia are the same as those of pyæmia, but they differ somewhat in activity, and act upon a system with a modified susceptibility, and above all one void of lesions in the internal membrane of the vascular system. The symptoms can be developed by the introduction of the ptomaines and toxins alone, which hypothetical condition has been named *septic-intoxication.* In case of excessive doses of septic material, death occurs so early as to indicate simple narcosis. If, as is usually the case, the microbes also gain access to the blood and multiply there, the condition has been known as *septic infection.* In any prolonged case of septicæmia, the tendency is to the formation of suppurating foci (*septico-pyæmia*), so that the two affections may be looked upon as probably the same, with modifications of the earlier phenomena.

In connection with septicæmia must be mentioned the *fermen-*

tation fever of Bergmann, (*aseptic* or *resorption fever*) which follows on extensive wounds, even if aseptic, on the intravenous injection of the blood of healthy animals or even of fine foreign particles (charcoal, flour), of a normal salt solution, or of well water, or of pancreatin, pepsin or trypsin. It has been attributed to the introduction and metabolism of fibrine and other elements, but manifestly arises also from the solution of blood globules, (hæmolysis). It comes on within a few hours after a severe operation or other cause and lasts from one to three days, terminating in recovery, unless complicated by some intercurrent infection.

The *sapræmic fever*, of Mathews Duncan (sapros—putrid, haima —blood) may also be named in this connection. It is associated with one or more of the common saprophytes (Bacillus saprogenes 1—2 and 3 of Rosenbach, Proteus Vulgaris, Proteus Zenkeri, Proteus mirabile, etc.) These are propagated with difficulty in the blood, but grow readily in pus or necrotic tissue from which their toxic products can pass into the blood.

Again the observations of Brieger and Maas, Ruine, Vaughan, Bourget and others show that the isolated toxins from putrefactive fermentation of animal matters, apart from the living bacteria are capable of producing the characteristic symptoms of septicæmia.

It is now generally concluded that the septicæmic phenomena can be produced by the introduction of such poisons, whether they are the product of septic fermentations outside the animal poisoned, or of fermentation in dead matter in the economy of such animal.

Lesions. In *fermentation fever* no tissue lesions are known. In *septicæmia* gross lesions are usually lacking unless the case has been prolonged to allow of secondary abscesses (septico-pyæmia). The blood however is dark and coagulates feebly if at all. The spleen is enlarged, softened, dark in color and gorged with blood. There are petechial hæmorrhages into the serosæ and mucosæ, and the solid organs; cloudy swelling of internal organs from coagulation necrosis; a parboiled appearance of heart, liver, kidneys and voluntary muscles; congestion of the lymph glands and usually the presence of the specific microbes in the blood and local lesions. The kidneys are always congested, and their epithelia granular and swollen, and there may be exudation between the glomeruli and their capsules.

Symptoms. Septic intoxication or septic infection may be ushered in by a staring coat or slight chill, but it rarely shows a violent rigor, such as inaugurates pyæmia. There is a rapid rise of temperature (102° to 104°), which persists for three to seven days without the marked remissions of pyæmia ; weak, compressible pulse ; great muscular debility ; hurried, shallow breathing, usually without cough ; anorexia ; emesis in vomiting animals ; dusky or yellow mucosæ from dissolved hæmoglobin ; scanty, high colored urine, rarely albuminous ; dulness, sometimes nervous twitching, delirium, apathy, stupor or paraplegia ; and either constipation or, later, diarrhœa. When such symptoms supervene on a gangrenous sore, septic abscess or fistula, retained placenta, blood clot in the uterus or elsewhere, suppurating tubercle, or other morbid product, gangrenous lung or other internal organ, purulent pericarditis, pleuritis or peritonitis, or any febrile affection which is complicated by necrosis, septicæmia is to be suspected. "Septicæmia should always be suspected during the course of any disorder the lesions of which afford an opportunity for the growth and development of septic microörganisms, when the symptoms of that disorder depart from the usual type and an elevated temperature continues beyond the usual duration." (Atkinson). "The final diagnosis of septic infection must be based on the existence of an infection atrium, through which pus microbes have entered the tissues, and from which they have reached the general circulation." (Senn).

Prognosis is always grave. A slight infection, overcome by the leucocytes or a simple septic intoxication may get well in two or three days, but an *acute progressive septic infection* will usually prove fatal in from one to seven days.

Prevention does not differ from that recommended for pyæmia.

Treatment is virtually hopeless unless it can secure the removal of the necrotic tissue or fermenting material from which the poison is derived. When the poisoning is due to the absorption of septic products only, with little or no introduction of microbes (septic intoxication) the removal of their source of supply may bring about a speedy and permanent improvement. The removal of a putrid placenta, or liquid from the womb, followed by irrigation with an antiseptic lotion, the evacuation of a putrid abscess, empyema, or ascites, followed by a similar disinfection, or indeed the extirpation of a sloughing and putrid mass of any kind may

be followed by a lowering of temperature within a few hours, and a steady improvement in the general symptoms. The antiseptic agents employed must be sufficiently potent, and persistently applied to render the surface sterile and yet not so caustic as to destroy more tissue to become a future culture-medium for the septic microbes. Mercuric chloride (1 : 2000) aluminum acetate (1 : 100) powdered iodoform, or aristol will often serve a good purpose, to be followed, when necessary, by efficient drainage and a covering of antiseptic gauze. When the primary source of infection is in the intestinal canal, calomel, napthalin or B. napthol may be tried.

For the weak heart strychnine is the most safe and reliable agent. Quinia in large doses acts as an antipyretic, without the attendant dangers of the coal tar products. It may be advantageously combined with tincture of chloride of iron.

Ammoniacal and alcoholic stimulants are largely resorted to to tide the patient over the period of depression, and nourishing and easily digested food should be given so far as the stomach can make use of it. Skim milk, eggs, and beef tea may be given even to the herbivorous patient.

The thirst should be met by plenty of pure water to favor elimination of the toxins, and the surface frequently sponged with tepid water, not only on the ground of cleanliness and disinfection, but also as calculated to lower the febrile temperature.

MALIGNANT ŒDEMA.

Definition. **Causes. Bacillus septicæmia gangrenosa, anærobic, rarely in living blood. Source of germ in soils. Pathogenic to man and domestic animals except cattle. First attack immunizes. Infects deep wound, exudates, dropsical and gangrenous parts, womb, intestine, debilitated parts, large dose intravenously. Lesions and symptoms ; excess of exudate, boggy swelling, watery discharge, fœtid gas bubbles, œdema of lungs and bowels. Complex infection. Minimum dose—abscess. Diagnosis : from black quarter and anthrax. Treatment : free incisions, hydrogen peroxide. Prevention : disinfection of skin and wounds. Immunity.**

Definition. An acute bacteridian disease of domestic and wild mammals, and of man, manifested by doughy, painful and often crepitating swelling in the vicinity of the affected part, and proving fatal in many cases in twenty-four to forty-eight hours.

Cause. The essential cause is the bacillus of malignant œdema, the septic vibrio of Pasteur, bacillus septicus gangrenæ of Arloing, the bacillus of septicæmia gangrenosa of others.

Morphology. This is a staff-shaped microbe 3–4 μ. long by 1 μ. broad, often united in chains of three or more to form long flexuous filaments. They are furnished with numerous flagella by which they are rendered very actively motile. The movements are tardy or simply flexous in the filaments. Spores form in the isolated bacilli (not in the filaments) in suitable culture media and at a temperature of from 20° to 38° C. They occupy a place near the center of the bacilli, not the ends as in the bacillus of emphysematous anthrax. The bacilli are anærobic and die quickly in air, but the spores are unaffected by oxygen. The spores are similarly resistant to most disinfectants. They will grow readily in ordinary culture media if oxygen is excluded, for example under an atmosphere of hydrogen, nitrogen or carbon dioxide, and liquefy gelatine. Even the oxygen present in the circulating blood is highly inimical to them, so that they are rarely found in the blood during life but rapidly invade both it and the tissues after death, and the suspension of respiration. In peptonized and glucosed gelatine the colonies are characterized as globules of liquefaction usually combined with gas. The germ is widely distributed in soils in general and not confined to limited areas like the bacilli of anthrax and black quarter.

Animals susceptible. The bacillus attacks man, horse, ass, goat, sheep, pig, mouse, Guinea pig, rabbit, white rat, cat, dog, chicken, pigeon, and duck. The mature ox is immune, but calves suffer. Dogs are often immune having already suffered from the disease. The first attack gives immunity from a second.

Infection Channels. Inoculation on an abrasion of the skin or surface sore is not usually infecting, the oxygen of the air destroying the germ. If, however, it is inserted deeply in the connective tissue, subcutem, it grows readily in a susceptible animal. Hence the danger of infection in deep wounds the recesses of which are not exposed to the air, and in such it becomes a most redoubtable surgical complication. If such wounds are the seat of active inflammation, with abundant exudate and more or less exclusion of the air-bearing blood, and in cases of blood stasis the field is specially inviting to the bacillus.

The debility of the injured tissue is a further invitation to the

attack. Chauveau injected 4 to 5 cc. of virulent liquid of malignant œdema into the veins of a ram and then practised bistournage, with the result that an invasion of malignant œdema of the scrotum and tunica vaginalis followed immediately. Pure cultures may be harmless, whilst an admixture of proteus vulgaris or micrococcus prodigiosus renders them most deadly (Penzo). Granulating wounds are even less favorable to invasion than simple abrasions. In these the bacillus cannot enter at once into the lymph channels and is exposed to destruction by the combined influence of the air and leucocytes.

Wounds in dropsical or gangrenous parts are equally favorable, to the development of the bacillus. Under such conditions the tissues are wanting in oxygen and resemble the condition of the entire body after death, when the bacillus of malignant œdema quickly penetrates its whole substance. Petri has traced the infection through the genital passages of newly delivered rabbits, producing a fatal metro-peritonitis and cutaneous œdema. A similar invasion may take place in other susceptible parturient animals. Lustig in a certain number of cases satisfied himself that he had traced the invasion through the intestine of the living horse. Invasion by the lungs, even by spores, is usually rendered impossible by reason of the presence of the inspired air.

A large dose of the virus is most likely to effect a successful invasion, since the toxins tend to debilitate and lower the defensive powers of the tissues and leucocytes. The effect of the toxins is shown under injections into the arteries, veins or trachea. A certain amount of hyperthermia follows, but there is rarely any colonization and reproduction of the bacillus in the connective tissue. In dogs and rabbits large doses given in this way induce short inspiration and broken or double expirations. In fatal doses death is preceded by extreme dyspnœa and convulsions. (Rodet and Courmont).

Lesions and Symptoms. The tissues where invasion occurs, become the seat of an abundant œdematous exudation, which feels boggy and painful and may even crepitate when pressed or manipulated. In case of an open wound, there is a profuse liquid discharge of a yellowish watery or serous aspect, and bubbles of gas or froth having a somewhat fœtid odor. The center of the swelling may become soft and flaccid while the

peripheral parts where the disease is advancing are tense and resistant.

In fatal cases the mucosæ of the small intestine and lungs are usually the seats of œdema in which the bacillus is found. The bacilli may also be found in the liver. It is noticeable that gross lesions of the spleen and kidneys are usually absent, in marked contrast with anthrax. The microbes found in the tissues may be in the form of bacilli, micrococci (spores, or m. prodigiosus), and sometimes filaments.

Inoculation with a minimum dose usually results in local abscess only.

Diagnosis. From *emphysematous anthrax*, with which malignant œdema is most likely to be confounded, it is to be distinguished by its appearance anywhere outside the black quarter areas, by the immunity of cattle which are so obnoxious to emphysematous anthrax, by the susceptibility of man, who does not contract black quarter, by the formation of the spore near the middle of the bacillus in place of at one end, by its resistance to the action of ordinary disinfectants, and by the greater tendency to form filaments.

From *anthrax* it is differentiated by its appearance outside the anthrax localities, by the absence of the bacillus from the blood and from the surface layers of the skin, by the normal size of the spleen, and by the active motility of the bacillus. It cannot be cultivated like anthrax in the free air or on the surface of culture media, and unlike anthrax bacillus, its cultures produce bubbles or gas.

Treatment. This is essentially surgical and consists in free incisions to admit air freely to all parts of the œdematous tissue, perfect drainage and a liberal use of peroxide of hydrogen. Other disinfectants may be employed but are much less promising. The free disinfection of the adjacent skin is an important element in treatment.

Prevention. This consists essentially in the thorough disinfection of all accidental and surgical wounds, the careful cleansing and antisepsis of the skin before an operation, the exclusion of earth, manure, or water from driven wells or fountains, from all wounds, and above all the exclusion of proteus vulgaris, and micrococcus prodigiosus.

Immunity may be secured by a first, non-fatal attack of the disease.

INFECTIOUS FEVERS OF SWINE.

One name for several affections. Differentiation, swine erysipelas, hog cholera and swine plague. Complex infections. Effects of large, medium and small doses, of more or less potent germ, of greater or less susceptibility.

Until comparatively recent years the various infectious fevers of swine have been confounded and described as a single disease, the name varying in the different countries in which they were observed. In America it was *Hog Cholera;* in England, *Swine Fever;* in France, *Rouget;* and in Germany, *Schweineseuche.* A closer study showed a marked tendency to a particular class of lesions in different epizoötics, and bacteriological research associated plagues in given localities with different microbes, so that progress has been made in differentiating one from another to a certain extent.

The first clear distinction was made in setting aside the swine erysipelas (rouget, rothlauf), from the rest as distinguished at once by its small, delicate bacillus, differing notably from the others in its staining and cultural peculiarities, as well as in the predominance of the cutaneous lesions.

What remains after eliminating erysipelas, constitutes a group having so much in common that attempts at further differentiation have led to much disputation, and not even to-day is there such accord in different countries as the writer of a text-book would find desirable. One class of pathologists claims but one common disease with many varieties under different conditions, just as the term *septicæmia* or *blood poisoning* has been made to designate a whole class of local and general infections, irrespective of the particular microbes that cause them. Others with greater precision give the disease a name according to the causation by one particular microörganism, or by another, which may be closely related to it in many respects, but which in successive subjects and outbreaks, maintains its own individual characteristics as regards morphology, cultural and staining habits, pathogenesis, etc. The question has been rendered all the more trying, by the occasional association in the same animal system, or in the same

outbreak of two distinct varities of microörganisms, in place of one, giving rise, of course, to modifications in the symptoms, lesions, progress, mortality, etc.

Apart from the microörganisms the whole class tends to show a close family relationship in their pathological phenomena shown under different conditions:

1st. Under a large dose, or specially virulent germ, in a particularly susceptible animal, all tend to a manifestation of an acute septicæmia, with generally diffused petechiæ of the skin, mucosæ, serosæ and internal organs, blood extravasations, and an early high mortality.

2d. Under a smaller dose, or a less potent germ, or in a less susceptible animal the tendency is to necrotic processes in the seat of inoculation or the point of election of local lesions. Necrotic ulcers are especially common in cases that survive one, two or three weeks, or that develop in a subacute or chronic form.

3d. With a minimum dose of a germ of little potency, and in a very resistant subject even the necrotic lesions may be absent, and there may be suppuration only or ulceration of serosæ and joints.

The question of the primary identity, or disparity of the whole class of germs, causing the septicæmic swine plagues, may be practically ignored in this work; it is important rather with our present knowledge to note the diseases associated with particular germs, or varieties of germs, and to describe these as far as possible as independent affections. This is as permissible as it is to describe small-pox, sheep-pox, and cow-pox as distinct affections, no matter what may be the truth or falsity as to their alleged original identity.

Rouget in Europe and *Hog Cholera* and *Swine Plague*, as the best established types in the United States deserve primary mention, to be followed by references to additional types which have been found to be associated with other distinct microörganisms.

ROUGET, ROTHLAUF. RED FEVER OF SWINE. SWINE ERYSIPELAS.

Definition. Comparative immunity of sucking pigs. Disease unknown in America. Causes: Bacillus erysipelatos suis, mature age, infection through yards, buildings, troughs, dust, mice, rabbits, pigeons, men, dogs, vermin, birds, butcher's and kitchen scraps, swill, hot weather, damp seasons, close pens, movement of swine, stockyards, fairs, public conveyances, public highways. Symptoms: incubation three days, chill, violet mucosæ, hyperthermia, recumbency under litter, muscular weakness especially behind, inappetence, thirst, costiveness, later diarrhœa, tenderness to touch, lymph glands swollen, red, blue, violet or black discoloration of skin, cutaneous swelling and pitting. Course: death in 12 hours to 6 days, or convalescence prompt. Mortality 20 to 80 per cent. Lesions: congestion of capillary vessels, blood extravasations, petechiæ, affecting cutis and subcutaneous fat, lymph glands congested, discolored; lungs engorged; spleen enlarged, liver and kidneys congested, petechiæ general, blood little altered. Bacillus 1.5 μ, anaerobic, easily destroyed in pens, in pork. Pathogenesis: swine, rabbits, mice, rats, pigeons and sparrows suffer. Rabbit germ less fatal to pigs. Immunization, advantages and drawbacks. Technique.

Definition. A microbian disease of swine manifested by high fever, great prostration and muscular weakness, a violet tint of the visible mucosæ, red or violet discoloration of the skin in spots and patches or universally, enlarged lymph glands, encreased size of the spleen, and general congestion of the capillary plexus.

Contrary to the habit of hog cholera and swine plague, rouget attacks mature swine mainly, the sucking pig showing a remarkable power of resistance. It does not appear whether this is due to the animal (milk) diet or to the absence of infection from feeding in the trough used by the adult animals. Up to the present this disease has not been recognized in America.

Causes. The one essential cause of rouget is the presence of the bacillus. The other conditions are either such as predispose the animal to receive it, for example, mature age: or they are such as favor diffusion of the poison, such as the introduction of an infected animal, the feeding of the healthy from the same manger with the infected, the introduction into the manger of the feet or snout which have become soiled with the infected manure or urine, the distribution of the infection in dust, the introduction of

the bacillus in the bodies of mice, rabbits, or pigeons, or on the feet of those animals, of men, dogs, birds, and vermin. We may add the distribution of infection in dried butcher's scraps used in pig feeding, and in uncooked scraps from the kitchen or in hotel swill.

It has been noted that the highest mortality prevails in hot summer weather, in damp seasons, and in narrow, confined, badly ventilated pens. Under such circumstances the introduction of a diseased pig will lead to the infection of most of the others in a few hours. Infection is quite as prompt through public pens in stock yards and fairs, and in public conveyances (cars, stock wagons, steamboats, ferry boats, etc.) and public highways.

Symptoms. After a period of incubation of three days or more the subject is seized with shivering, the limbs are hot and cold alternately, respiration and heart beats are accelerated, the mucous membranes assume a dark violet tint and the rectal temperature rises to 104° to 108° F. From the first the pig tends to bury itself under the litter, and refuses to move unless absolutely forced to do so, and then only with painful grunts, swaying and staggering limbs (especially the hind ones), and straight drooping tail. There is inappetence, but thirst remains, and the bowels are at first costive, the manure being covered with a film of mucous or even streaks of blood; later they become relaxed and diarrhœa becomes often a prominent symptom. The pig seems to suffer and often squeals when handled, and he may give a weak, dry cough. The external inguinal glands may often be felt perceptibly enlarged. The red discoloration of the skin appears early and extends and deepens to the end in fatal cases. It may be of a bright red, or of a bluish red, violet or black. The first indications appear as spots, by preference around the roots of the ears, on the breast and abdomen, inside the arms and thighs, and in the perineum. These isolated spots run together into great patches, which extend over the whole ventral aspect of the body, and may cover the entire dorsal aspect as well. In some instances the skin is swollen and retains an impression made by the finger.

Course. The disease may reach a fatal termination in twelve hours: more commonly it endures for forty-eight hours, and at times it will last for four, five or six days. In the most rapidly

fatal cases, the violet discoloration of the skin may be absent or only a little marked, while in the protracted cases it acquires its greatest extensions and its darkest shades. In the protracted cases too the prostration becomes extreme, the animal may find it impossible to raise himself on his hind limbs, the diarrhœa becomes profuse, liquid and fœtid, the respiration labored, cyanosis sets in and the temperature is reduced below the normal standard.

In case of recovery, convalescence is usually prompt and complete, differing in this from cases of swine plague and hog cholera. The more favorable issue in rouget probably depends on the comparative integrity of the intestinal mucosa and mesenteric glands, which are subject to slow-healing lesions in swine plague and hog cholera. Slow convalescence is however not uncommon, yet in such cases, the concurrent, speedy and complete recoveries in other animals in the same herd serve to identify the disease as rouget.

Mortality. The mortality among grown hogs averages eighty per cent.

Morbid Anatomy. The most prominent lesion is the general congestion of the capillary blood vessels, and the numerous minute extravasations or petechiæ. The skin shows in the red patches a general dilatation of the capillaries which have become at the same time elongated and tortuous, with minute, often microscopic, ruptures and extravasations at frequent intervals. This usually extends to the whole thickness of the cutis, and to a considerable depth in the subcutaneous fat, Where swelling occurred or pitting on pressure, a serous infiltration of the tissues is found. The lymph glands are uniformly enlarged and discolored, of a dark red, almost black, color, the congestion and extravasation being extreme in the cortical substance, while the medullary is paler, soft and cellular. The lungs are usually gorged with black blood suggesting death by asphyxia. In tardy cases there may, though rarely, be centres of broncho-pneumonia. The spleen is enlarged, with dark color and uneven surface from rounded swellings, and is filled by a soft black, bloody pulp. The liver is congested, the kidneys congested, enlarged and petechiated, and the gastric and intestinal mucosa congested and thickened, with desquamating epithelium, and swollen solitary and agminated glands, the degree of alteration usually bearing a ratio to the duration of the disease. The serosæ are usually extensively

petechiated and serous effusions occur into the serous cavities. The muscular substance of the heart and the endocardium are also the seats of petechial extravasations. Unless in some protracted cases the blood appears to be unaltered as regards its power of taking up oxygen, or coagulating.

Bacillus of Rouget. The germ of this disease is found in small numbers only, in the blood and vascular tissues, but very abundantly in the lymph glands, the spleen, the kidneys, and the red marrow of the bone. It is also present in enormous quantities in the urine and the bowel dejections, the former (urine) offering a ready means of diagnosing the disease microscopically.

The bacillus is 1 μ to 1.5 μ long by 0.1 μ to 0.15 μ broad, is non-motile, and stains readily even in Gram's solution. They occur either solitary or in pairs tending to unite at an angle. In old artificial culture, chains of considerable length may be formed. In the blood the bacillus is usually found in the leucocytes, as many as 20 or more being often present in a single cell. In the lymph networks of organs they also invade the leucocytes but are found in free masses as well. The bacillus is anærobic, but facultative ærobic, its preference being manifestly for the absence of oxygen. It is nonliquefying. In gelatine cultures no development takes place on the surface, but along the line of puncture a delicate cloud-like branching growth takes place which extends horizontally in parallel masses from the central puncture. This resembles but is not quite so delicate as that formed by the bacillus of mouse septicæmia with which it is supposed to be identical. It grows scantily on the surface of nutrient agar or blood serum, but not at all on bouillon, in the bottom of which, however, it forms a slight grayish white deposit. It does not grow on potato. The bacillus sometimes shows refrangent granules which have been supposed to be spores, but this idea appears to be negatived by the ease with which its vitality is destroyed by heat and disinfectants. The thermal death point is 68°C. (137° F.) maintained for 10 minutes (Sternberg). Boulton found that it was killed in 2 hours by mercuric chloride (1 : 10000), by carbolic acid solution (1 : 100) and by sulphate of copper solution (1 : 100).

It is killed by desiccation, by quick lime and by chloride of lime. At a temperature of 18° to 27°F. it perished in 13 days. In salted pork it lost vitality in one month.

Pathogenesis. The bacillus is pathogenic to swine, rabbits, white mice, house mice, white rats, pigeons and sparrows. Field mice, guinea pigs and chickens are immune.

Mice and pigeons take the disease most certainly, and die in three days to five, the whole body swarming with bacilli. Rabbits take the disease less certainly or rapidly, inoculation in the ear causing first an erysipelatoid inflammation, and recovery with immunity often takes place.

Immunization. When inoculated continuously from rabbit to rabbit it encreases its potency for that animal, which it comes to kill in 24 to 48 hours, but in the same ratio it loses its virulence for swine upon which it can then be inoculated without danger to their life.

It was on this basis that Pasteur and Thuillier established in 1883, their preventive inoculation for rouget. The method has been most extensively employed in Europe, and where intelligently employed has prevented this disease. From the laboratory at Buda-Pest alone, there was sent out in one year material for 249,816 swine.

The objections to the method are: the danger of mistaking hog cholera and swine plague respectively for rouget, as the rouget mitigated germ would be in no sense protective against these; and the danger of spreading the germs of rouget in fresh localities and thus introducing a new plague instead of controlling and preventing an old one. In the Baden experiments 5.4 per cent. of inoculated pigs died, and of 118 unprotected pigs exposed to them 62 per cent. contracted the disease and one died. In France and Hungary, on the other hand, 1 to 1.45 per cent. died of the operation, instead of 20 per cent. when the disease was contracted in the ordinary way.

It is held that the danger lies largely in the inoculation of very young pigs, and Nocard advises to operate only on those of four months and upward.

The danger of spreading the germ by inoculation may be the more easily guarded against, considering that it is very destructible by disinfectant agents (heat, dryness, cold, chloride of lime, quick lime), and that it does not readily survive in a locality, where it cannot find a constant succession of victims. Yet the practice ought to be confined to herds exposed to infection, and under special precautions, as regards the exposure of other herds.

The *technique* of the Pasteurian inoculation is to inject, subcutem, on the inside of the thigh, 0.1 cc. of the weaker preparation (premier vaccin), and twelve days after a similar dose of the stronger one (deuxieme vaccin).

This produces a mild attack of the disease from which the great majority recover, and though they still react somewhat to a second and third inoculation, yet the disease so produced is rarely fatal.

CHOLERA SUIS; HOG CHOLERA.

Definition, Synonyms, History, Losses. Bacillus choleræ suis, 1.2—2μ., ærobic, biology, table of germs; accessory causes, roaming pigs, railways, car litter and manure, boats, trucks, loading banks, chutes, runways, stockyards, pens. fairs, watershed, butchers, dealers, etc., wagons, dogs, birds, vermin, insects, offal of abattoirs, butcher's and kitchen scraps, unburied carcasses of dead hogs, convalescent and immune hogs, susceptibility, parasites and infection atria, putrid food, infection from ground carried into feeding trough by snout and feet, large herds, rapid carriage of swine for long distances. Lesions; hæmorrhagic spots and petechiæ on skin, mucosæ and serosæ, circumscribed capillary congestions, congestion of spleen, lymph glands, stomach, intestines, necrotic processes. Button like ulcers on intestinal mucosæ. Incubation, 6 to 14 days. Symptoms: fulminant cases. Acute Cases: dulness, anorexia, recumbency on belly, weakness, paresis behind, thirst, tenderness of skin and abdomen, hyperthermia, easily blown, blush on skin, dark red spots and patches, enlarged inguinal glands, cutaneous exudate—greasy or drying black, bowels costive, later pultaceous and finally diarrhœaic, petechiæ on mucosæ, emaciation. Chronic Cases: symptoms more slight, but great loss of condition. Diagnosis: from swine erysipelas, swine plague, Widal test, table of differential symptoms. Prevention: expense of extinction prevents effective measures; removal of accessory causes, comfort, air, light, food, salt, powered soaps, mouldy bread, cotton seed, space, green food, precautions against introduction of bacillus, special shipping provisions for fat hogs, exclusion of stock hogs from infected localities, precautions by purchasers. Immunization. Disinfection. Certificates. Extinction in herds and districts. Treatment: chronic cases, food, antiseptic medication, antithermics stimulants, tonics. Serum therapy, method, merits, demerits.

Definition. A contagious bacteridian disease of swine, acute or subacute, and characterized by hyperthermia and other febrile disorders,—congestion, exudation, ecchymosis and necrotic ulcera-

tion of the intestinal mucosa and of that of the stomach and of other parts,—by a profuse foul, liquid diarrhœa, by enlargement of the lymph glands with congestion and blood extravasation,—by effaceable blotches, and petechiæ (ineffaceable) of the skin, snout and visible mucosæ, with a tendency to necrotic changes—less frequently by pulmonary congestions, and degeneration,—and by a high mortality.

Synonyms. The earlier designations were mostly drawn from the red or black discoloration of the skin and mucosæ and applied indiscriminately to the other forms of hæmorrhagic septicæmia which we now differentiate as erysipelas (rouget, Rothlauf) and swine plague. They included *measles*, *erysipelas*, *scarlatina*, *red soldier*, *purples*, *blue sickness*, *carbuncular fever*, etc. Others basing their nomenclature on the prominent intestinal lesions, etc., designated it *typhoid fever*, *pig typhoid*, *typhus*, *carbuncular gastro-enteritis*, *pneumo-enteritis*, and *diphtheria*. Even in Europe while the *pig erysipelas* (*rouget*, *Rothlauf*) is now recognized as a distinct disease there is no clear distinction made between *hog cholera*, and *swine plague*. In England we find these more or less confounded under the names of *swine fever*, *swine plague* and *hog cholera*, and on the continent of Europe under those of *schweineseuche* and *schweinepest*, or *pneumo-enteritis infectieuses*. Differences in different epizootics or outbreaks are recognized, and the field is left open for the future identification of different forms of this common group of swine fevers, but the existence of constant bacteriological distinctions are not always insisted on, as we do in the United States in the case of the two great leading types *swine-plague* and *hog cholera*.

History. Definite history of this disease may be said to begin with the discovery and demonstration of the actively motile hog cholera bacillus by the U. S. Bureau of Animal Industry in 1885. Yet in the history of animal plagues, even in early times, deadly epizootics are described which undoubtedly represented one or other of the contagious affections of modern times. Among the more definite may be named a destructive gastro-enteritis (magen seuche) in Germany in 1817, a pleuro-pneumonia in France and Bavaria in 1821, a cholera with blotching of the skin (morbus niger) in Ireland, and an erysipelas in pigs in France and Switzerland in 1836, and in Ohio in 1833, there was a fatal

affection afterward recognized as hog cholera. Writers conjecture that it was imported into America from Europe in improved pigs, and from one European country to another in the same way, but we have no absolute proof of times and shipments and their immediate effects, so that these theories are but more or less reasonable deductions from the familiar extensions of the disease in more recent cases. Under the great commercial activity of the latter half of the 19th century, the active movements of animals by canal, steamboat and rail, and the massing together in one market of many animals drawn from widely different sources, hog cholera has made extraordinary extensions on both sides of the Atlantic, until Friedberger and Fröhner pronounce the schweineseuche and schweinepest the most widely disseminated and dangerous of swine epizootics, and Dr. Salmon estimates the losses in the United States at $10,000,000 to $25,000,000, per annum.

Bacteriology. Prior to 1885 bacteria had been found in the different outbreaks of contagious fevers in swine, and the bacillus of swine erysipelas had been demonstrated in 1882, but it was only two years later (1884) that the motile bacillus choleræ suis was first described by Klein, and in 1885 that Salmon and Smith described it as the essential cause of the disease, together with its biological and cultural peculiarities.

It is a short bacillus, 1.2 to 2μ × 0.5 to 0.8μ, but varying considerably in size according to the stage of its growth and the genera of animal or culture medium in which it is grown. It has rounded ends and is usually in pairs connected by an invisible band. It stains promptly in all the aqueous aniline colors, but loses the stain in a solution of iodine (Gram's). Prolonged exposure of artificial cultures produces an uniform stain, while a transient exposure, and especially of bacilli obtained from the tissues, stains them most deeply at the ends (polar) and periphery, while the centre remains somewhat clear. This is less marked than in the bacillus of swine plague, yet serves to show the relation between this microbe and the colon group.

It is *ærobic* (facultative anærobic), non-liquefying, and, in fluids, very actively motile, the movements lasting for months in preserved specimens (Smith). It grows luxuriantly in various culture media, and especially in alkaline ones, at the room

temperature, and most actively at 85° to 100° F. It may grow as low as 60° to 70° F. and as high as 104°. (Swine plague bacillus grows at 55.4°).

On *peptonized gelatine* the surface colonies are usually round and flattened, those in its substance globular and smaller, and those at the bottom expanded next the glass and rising in the center into the gelatine like a knob. At 48 hours they appear as opaque whitish points and slowly increase to ½ to 2 mm. They may be brown by transmitted light, the depth of color increasing with age. On *agar* the colonies are grayish, shining and translucent and may reach the size of 4 to 6 mm. On *potato* (alkaline) a straw yellow film is formed, darkening with growth. In *bouillon* a turbidity appears in 24 hours, and in 1 or 2 weeks a precipitate and surface film.

The bacillus is usually larger in the gelatine and smaller in the bouillon than it is in the tissues. It seems to produce neither phenol nor indol.

Its behavior with sugars is significant. It ferments glucose producing acid and gas; does not ferment saccharose nor lactose, but turns the saccharose solution alkaline (no gas). In bouillon containing muscle glucose, it may without additional glucose form a little gas. The swine plague bacillus ferments saccharose producing acids but no gas: it ferments neither glucose nor lactose but turns the former acid.

Milk is neither coagulated nor soured by the hog cholera bacillus, but in 3 to 4 weeks it undergoes a change, becoming saponified.

Cultures have no special nor offensive odor. Some varieties in close tubes may cause a faint acid odor.

Oxygen is not essential to the success of a culture. The colonies form as promptly and as large in the depths of gelatine, or in a vacuum, as if in free air.

The following table will serve to show differences between the hog cholera bacilli, and related pathogenic microbes:

Hog Cholera.	McFadyean's.	Swine Plague.
B. Choleræ Suis	Swine fever B	B Pestis Suis
1.2 to 2μ × 5 to 0.8μ	1 to 2μ × 0.6μ.	0.8 to 1.5μ × 0.6 to 0.8μ
Ends rounded	Ends rounded	Ends rounded
Involution forms		Involution forms
Actively motile in liquids	Actively motile	Nonmotile
Flagella		No Flagella
Stains throughout, lighter in center	Polar or uniform faint stain	From fresh organs polar stain, from old cultures uniform
Bleaches in Gram's (I Sol.	Bleaches in Gram's (I) Sol.	Bleaches in Gram's (I) Sol.
Ærobic (Fac. Anaerobic)	Ærobic	Ærobic, (Fac. Anærobic)
Vigorous growth in alkaline nutrient fluids	Slight growth in alkaline nutrient fluids.	Weak growth in alkaline nutrient fluids.
Active growth on potato yellowish, becoming darker	No growth on potato	On potato at 37° C. a slight, thin gray, waxy layer
On gelatine, small, round brownish colonies	On gelatine light bluish colonies shading off insensibly at edges	On gelatine feeble growth or none
Nonliquefying	Nonliquefying	Nonliquefyiug
On agar conical colonies, grayish white, semitranslucent. shining	On agar slight, transpaent, almost invisible growth	On agar, grayish translucent, or brown, knobbed, waving edges
In milk grows freely, no acid, no clot: Saponifies in 3 or 4 weeks	Grows in milk, no clot	Grows in milk, no acid, no clot
Forms no indol in pancreatic bouillon		Forms no indol in pancreatic bouillon
Ferments glucose, forming acid and gas		Does not ferment glucose, forms acid, no gas
Lactose not fermented		Lactose not fermented
Saccharose not fermented; alkalinity; no gas		Saccharose fermented, forms acid, no gas
Thermal death point (moist) 58° C. in 15 minutes	Thermal death point 58° C. in 10 minutes	Thermal death point (moist) 58°C. in 7 minutes
Desiccated it dies according to bulk in 7 to 49 days	Dies quickly, if dried at body temperature	Dies in 3 days if dried
Dies in water in 3 to 4 months		Dies in water in 10 to 15 days
Dies in soil in 2 to 3 months		Dies in soil in 4 to 6 days
Pathogenic to swine, rabbits, guinea pigs, mice, pigeons	Pathogenic to swine and rabbits	Pathogenic to swine, hens, pigeons, pheasants, sparrows, mice, rabbits, cattle, deer, guinea pigs; etc.
	Guinea pigs immune	

Swine Erysipela,.	B.Coli Commune.	Typhoid Fever.
B. of S. Erysipelas		B. Typhi Abdominalis
1 to 1.5μ × 0.1 to 0.2μ	2 to 3μ × 0.4 to 0 6μ	1 to 3μ × 0.6 to 0.8μ
Ends rounded	Ends rounded	Ends rounded
	Involution forms	Involution forms
Nonmotile in liquids	Nonmotile or very slightly so	Motile
No flagella	No flagella	Flagella
Stains readily and uniformly	Stains uniformly or polar	Stains uniformly (points clear)
Stains in Gram's (I) Sol.		Bleaches in Gram's
Anærobic (F. Ærobic)	Ærobic (F. Anærobic)	Ærobic (F. Anærobic)
Active growth in common nutrient liquids at 37°C	Grows well in usual nutrient liquids, even if acids	Grows well in usual nutrient liquids
Usually no growth on potato; variable	Yellowish thick white growth on potato	Grows on potato; transparent glistening surface
In gelatine stab culture delicate feathery, branching growth	In gelatine amber colonies, becoming brown; may be bubbles of gas	ON gelatine clear colonies with radiating and encircling lines
Nonliquefying	Nonliquefying	Nonliquefying
Grows in milk; acidifies, often coagulates it	Acidifies and clots milk in 8 to 10 days	Acidfies milk growing freely
Thermal death point (moist) 58°C. in 10 minutes	Thermal death point (moist) 60°C in 10 minutes	Thermal death point (moist) 56°C. in 10 minutes
Loses virulence rapidly when dried		
Dies in water in 18 to 20 days		
Lives and even multiplies in rich soils, manures, etc.		Lives and grows in fæces
Loses virulence slowly in light and air		
Pathogenic to swine, pigeons, sparrows, rabbits, white and house mice, white rats		
Guinea pigs, field mice and hens immune		

Hog Cholera.	McFadyean's.	Swine Plague.
Hen or pigeon has slough where inoculated, diarrhœa, ruffled plumage, somnolence		Hen or pigeon dies in 48 hours, after drowsiness, drooping wings, sunken head, ruffled plumage, liquid stools, soft, black comb and wattles, prostration
Rabbits getting 0 1cc. virulent culture subcutem die in 5 to 7 days with eularged spleen and necrotic liver foci.	Rabbits getting 0.5 to 1cc. culture subcutem had tumor like walnut but recovered	Rabbits getting 0.01cc. culture subcutem die in 16 to 20 hours, with inflamed serosæ and lung; Petechiæ
Weaker culture kills in 10 to 20 days with enlarged spleen, or recovery ensues		Weak cultures kill in 4 to 10 days, with inflamed serosæ and suppuration
Guinea pigs die in 7 to 12 days.	Not pathogenic to Guineapig	Guineapigs die in 1 to 4 days
Swine inoculated subcutem have often local lesions and bacilli, also in lymph glands, only exceptionally fatal		Swine inoculated have local lesions only, only exceptionally fatal
Ingestion of virulent cultures by fasting pig causes bowel lesions and death	Ingestion of 30 cc. by pigs proved always fatal	Ingestion of virulent cultures by pigs is usually harmless
Intravenous inoculation in pig causes septicæmic lesions and death, or chronic diseases and typical bowel ulcers		Intravenous inoculation causes septicæmia and death in 1 or 2 days
		Intrapulmonary infection causes pleuropneumonia

Swine erysipelas kills inoculated pigeon in 3 to 8 days, and rabbit in 4 to 8 days.

Invisible, Infinitesimal Hog Cholera Microbe.

The later research of Dorset, Bolton and McBryde, of the Bureau of Animal Industry, shows clearly that the *Bacillus Pestes Suis* (B. Suisepticus) and the *B. Cholerœ Suis* do not exhaust the list of pathogenic microbes in the familiar swine fevers. An infinitesimal microbe which passes readily through the Chamberland and Berkefield filters, and the existence of which has not been demonstrated by staining and microscope, produces symptoms and lesions as characteristic as those due to the pure cultures of the hog cholera bacillus and is incomparably more infecting. It is also much more deadly, but the few animals that recover from the disease show an immunity as strong as that which follows an attack of the disease caught by simple exposure.

Recovered cases after the disease caused by a pure culture of hog cholera bacillus do not show such immunity. It would appear, therefore, as if the hog cholera bacillus were an attendant complication, though by no means a harmless one, whilst the primary infecting factor is the, as yet unseen, infinitesimal organism.

Of 21 pigs herded with pigs that had been injected subcutem with *pure cultures of Bacillus Choleræ Suis*, only one showed any sickness, and that very slight. The blood of the injected pig to which this was exposed, though injected intravenously into a healthy pig, had no ill effect, showing the absence of both the *B. Choleræ Suis* and the unseen infinitesimal organism. The one which had slight sickness, when afterward penned with casual cases of hog cholera, proved immune.

Eighteen of the above 21 pigs, exposed to swine injected with pure cultures of the Bacillus Choleræ Suis, were afterward put in a pen with cases of casual hog cholera and 16 sickened. The rate of mortality in these 16 is not mentioned. The two other pigs were evidently immune. One had shown slight illness as named above, when exposed to hogs injected with *pure cultures of Bacillus Choleræ Suis*, and both resisted infection when penned with hogs suffering from *casual hog cholera*.

Of 11 pigs, injected subcutem, with blood from hogs sick from administration of *pure Cultures of B. Choleræ Suis*, none sickened, and of 10 of these, exposed to infection in a pen with *casual cases*, 9 sickened and 7 died.

Of 19 pigs injected subcutem with *pure Cultures of B. Choleræ Suis*, 3 only died and 8 showed no illness. Of 2 fed with the same *B. Choleræ Suis* in pure cultures, both sickened and recovered. Of the 18 survivors, placed in a pen with *casual hog cholera cases*, 16 sickened and died. Six of these had already shown sickness from injections of pure cultures and 2 from being fed on them, while 8 had shown no illness as the result of these cultures. The 2 survivors had both sickened from injections of the pure cultures.

Two sound pigs injected subcutem with blood from a case of casual hog cholera, and the blood taken at intervals from the tail, and in each instance disposed of, 1st by injecting a sound hog subcutem, and 2d by culture in neutral bouillon, it transpired that blood proved infecting to the two hogs on the *second day*, while the bouillon was not successfully inoculated with the *B. Choleræ*

Suis until the blood of the *third* and *fifth day* (in different cases) was reached. The blood of the inoculated pig was therefore invaded by the infinitesimal microbe from one to three days before it was invaded by the *B. Cholerœ Suis.*

Of 36 pigs injected subcutem with unfiltered serum from casual cases of hog cholera, all sickened, 24 died, 10 were killed when moribund, and 2 recovered. Of 61 pigs injected subcutem with an equal amount of serum from casual hog cholera cases, but which had been passed through a Chamberland or Berkefield filter, 58 sickened, 39 died, 10 were killed when moribund, 9 recovered, and 3 showed no illness. Though there is here a slight reduction in the mortality from the filtered product, and though sickness often occurred slightly earlier from the unfiltered material, this is of little account in solving the question before us. That the filtered serum was free from *B. Cholerœ Suis* was shown by its inability to produce these in culture tests as well as by its harmlessness when inoculated on rabbits and Guineapigs. The 9 hogs that recovered proved immune, for all resisted when placed in a pen with casually infected hogs, one died but without evident lesions of hog cholera. Finally, the blood of the pigs injected with the filtered hog cholera blood proved infecting to other pigs when inoculated or fed to them.

Assuming that due care was exercised to prevent the casual introduction of *B. Cholerœ Suis* the following conclusions seem warranted :

The infinitesimal filterable hog cholera microbe.	*Bacillus Cholerœ Suis.*
Very contagious from pig to pig by simple exposure.	Not contagious by exposure.
Filtered injections subcutem constantly virulent, usually fatal.	Pure cultures injected subcutem, usually harmless to pig; deadly to rabbit and Guineapig.
Injections of blood subcutem, constantly virulent and fatal.	Injections of blood subcutem, usually harmless.
Intravenous injections of blood constantly virulent and fatal.	Blood intravenously virulent and fatal.
Cases that recover strongly immune.	Recovered cases show little immunity.

Accessory Causes. These are especially those conditions which favor the transmission of the germ from animal to animal. They include the reprehensible habit of allowing swine to run at large

so that herd mingles with herd ; the freedom to wander along the lines of railroad by which hogs are carried, and where the infected excretions fall on the ground ; the scattering of infected litter or manure from a car or boat ; the use of the same cars, boats or trucks for the conveyance of infected and sound pigs in succession, without intermediate disinfection ; the use of the same loading banks, chutes, runways, yards, pens and feeding and watering troughs by strange pigs from all sources in succession, without constant disinfection ; the purchase of stock swine at public markets ; the return of swine from public fairs and exhibitions ; the feeding and watering of pigs on the line of streams that have drained pig pens or pastures higher up ; the use for pigs of premises that have harbored infected ones at an earlier (even distant) date : the supply of food or litter from barns where pigs have recently died ; the admission to the pens or yards of butchers, dealers or others who are likely to carry infection on their persons ; the admission even of wagons, dogs or other animals, including birds, tame and wild, which are liable to carry infection. Of all birds, the buzzard is the most to be shunned as having presumably just come from infected carrion, but barnyard fowl and small birds that feed from the same trough with the pig are to be feared as well. The same remark applies to rats and mice, squirrels, skunks, woodchucks and rabbits which may easily carry the infection on their paws. If the infection is near, flies and other insects, in the warm season, will convey it for some distance from herd to herd. A common cause is the feeding of swine about abattoirs where they devour the offal and waste in a raw condition. Another is the feeding of boarding house, hotel and other kitchen slops, raw, or without the most exhaustive precautions in the way of cooking. Many outbreaks can be traced in this way to the consumption by the animals of the products of infected swine. Some indeed are fostered by the utter neglect of the parties in charge of an infected herd, in leaving the infected carcasses exposed so that they are eaten by wandering hogs. or portions are carried away by buzzards, carrion crows, dogs and other animals. In some cases a strong wind will carry the infection on dust, straw or other light object into sound herds at a distance. The introduction into a hitherto healthy herd of an apparently sound pig may be the occasion of a deadly outbreak. The strange new pig may have already had

the disease, and in a condition of immunity, may without hurt to itself, carry the germ which becomes so fatal to the susceptible.

This susceptibility is one of the most important factors. It may be inherent in a given family or strain of blood. It may be enhanced by a constitutional weakness, engendered by too close breeding, by breeding from the young and immature, or from the old and worn out. It may be favored by a general debility from starvation, faulty or injudicious feeding, as exclusive feeding on corn (maize), an unbalanced ration, feeding cotton seed, irregular feeding, etc. It may result from parasitism, as round worms in the lungs, bowels, muscles, fat, kidneys or liver, from trichinosis, from cysticerci, echinococci, or from distomatosis. These not only lessen the force of constitutional and phagocytic resistance, but they also in many cases open the way for the entrance of the microbe by the wounds which they inflict. Perhaps nothing operates more effectively in this way than the attacks of other pathogenic microbes. The treatment of the domestic hog is often such that it would almost appear as if it were designed to destroy health and vitality. He is used to clear up the soiled and spoiled provender which has been rejected by other animals. Decayed vegetable and flesh of all kinds, which is no longer fit for other use, is supposed to be good for him and is furnished raw. Worse still, this is conveyed in barrels that are never washed, but are sent for each new supply reeking with abominations which render them a nuisance on the highway. It is left standing till wanted in these barrels, or in still larger receptacles, which are never emptied or cleaned, but are allowed in the hottest weather, to continue a hotbed of the foulest fermentations. On a smaller scale the kitchen swill barrel becomes a similar centre of decomposition. Even at the creamery and cheese factory the surplus or waste products often remain in a common tank breeding larvæ, toxins and ptomaines, before they are fed to the hogs. In the hog pen, or yard, corn in the ear is thrown on the ground, already filthy with the solid and liquid excretions and is eaten with the rotting, if not infecting, filth in which it has been rolled. From grubbing in this filth with his snout, the pig plunges the latter in the liquid food in his trough and too often he gets his feet into the food as well, and further charges it with the injurious ferments. Again the kitchen swill is liable to contain various inorganic poisons and notably the carbonates and bicarbonates of

potash and soda which are used to excess in the form of *powdered soaps* and, as shown by experiment, are deadly poisons to pigs.

The gastro-intestinal disorders caused by these poisons; (it may be botulism from stale or decomposing flesh, fish or fowl, the poisoning by mouldy bread or musty grain, or meal, or by the toxins of the many and varied saprophytic fermentations), often prove as deadly as outbreaks of genuine hog cholera, and are habitually mistaken for them. They do not, however, as a rule extend beyond the particular herd which has been exposed to the faulty management, and introduce no risk of a general spreading infection. The careless owner suffers and adjacent herds escape, unless exposed to similar causes. But if the hog cholera germ is present these pave the way for its destructive advance and tend to enhance the mortality. It may even be that the combination of the two factors is a condition of the eruption of a severe attack. The faulty feeding or food or poison by itself could be resisted, and the comparatively non-virulent hog cholera bacillus might have been resisted, but with weakened system and digestive apparatus, the microbe finds a specially inviting field in which it can multiply destructively, and where it can gather a virulence which will enable it to invade and sweep away herd after herd in a deadly epizootic.

I may add, as a prominent factor in the great modern extensions of hog cholera, the habitual aggregation of swine in large herds. This with the rapid steam transit of modern times, and the great aggregations of hogs in one common market, probably contributes more than anything else to the extraordinary diffusion of the infection. By accident, purchase or otherwise, a large herd becomes infected, and the owner, knowing that delay is ruin, at once ships the apparently healthy animals to market; these infect anything they or their excretions come in contact with; if sold in smaller lots they carry infection into every locality where they go, and along the route; if sold for slaughter, they still diffuse infection through the herds that receive their butcher and kitchen trimmings.

Finally other domestic animals may bring in an infection which becomes manifested by symptoms similar to those of hog cholera, and which if really different, yet serves to pave the way for such an outbreak. Galtier's remarkable experience with a *pneumo-enteritis* in sheep, introduced into five separate flocks by infected

pigs from the same market, is significant in this respect. It is further significant that the hog cholera bacillus is a very protean microbe. Th. Smith, to whom we owe more than to any one else the identification of the germ, gives seven varieties, which showed well-marked distinctions in their morphology, in their modes of growth on culture media, in the amount of gas they respectively produced in a glucose bouillon, or in their pathogenesis for rabbits. One of these modified germs which has largely parted with its virulence for pigs and some other animals, may under specially favorable conditions, resume its former potency and proceed on a new career of infection.

Lesions in the Acute or Septicæmic Form. The skin and subcutaneous fat are the seats of diffuse blotches or spots of a deep red varying from dark purple to light red, confined it may be to the inner sides of the arms and thighs, the belly, the ears, eyelids, and muzzle, or it may be all but uniformly diffused over the body. When pressed so as to expel the blood, the greater part of the surface may be momentarily whitened, but red points remain representing the minute extravasations. Under the microscope the red points show tortuous and enlarged capillaries with here and there a rupture and minute clot. The visible mucosæ may show similiar petechiæ, as may also the serosæ of the chest, cranium and abdomen. In the latter, blood extravasations are liable to be more extensive. The spleen and lymph glands (particularly those of the bowels and omentum, the sublumbar and subdorsal regions) are usually enlarged, gorged with blood and softened. Many of the lymph glands may escape, and in others the congestion is largely confined to the cortical portion. The lungs may show petechiæ and even extensive hæmorrhages into their substance. The kidneys may show petechiæ in the glomeruli, the medullary substance, the papillæ or the pelvic mucosa, or there may be larger circumscribed hæmorrhages.

The stomach in its greater curvature especially is usually deeply congested and petechiated, with small submucous extravasations, and these conditions are liable to be still more marked in the small intestines and especially in the large, which may have a dark red or port wine hue. Blood may be present in clots among the contents. Necrotic ulcers are absent.

Lesions in the Protracted and Chronic Forms. The lesions of the skin are usually less extensive than in the acute type, and may be almost entirely absent. The lymph glands are enlarged

and congested, though the discoloration may be largely confined to the cortical layer. The spleen is as a rule normal in size. The liver is firm, but it may show softening of the secreting acini and encrease of the fibrous framework. Petechiæ or circumscribed hæmorrhages may or may not be present on or under the serosæ or in the tissues.

The characteristic lesions belong to the gastric-intestinal organs. Congestions and ulcers may be found on the gastric mucosa, on that of the small intestine, and rectum, but they are above all common on the ileocæcal valve, cæcum and first half of the colon. In the earlier stages of these lesions mucosa and submucosa are the seat of a congestion and exudation, but later the round button-like ulcer usually stands out prominently with its necrotic centre dirty-white, brown or black, and composed of superposed layers, the whole resting on a congested and thickened submucosa. This contains small round and giant cells and may show considerable encrease in connective tissue. The ulcers may be seated on the agminated or solitary glands but do not show the same predilection for these parts which is seen in typhoid fever.

Incubation. This varies, according to the dose and susceptibility, from two or three to as many as thirty days. With the short incubation the disease tends to assume its most acute and deadly type, while the prolonged incubation bespeaks a milder form. During ordinary outbreaks from six to fourteen days represent the average interval between exposure and the onset of active symptoms. During the extreme heats of summer and the excessive cold of midwinter in our northern states incubation tends to be shortened.

Symptoms in Fulminant Type. In violent outbreaks some pigs are found dead without observed preliminary symptoms, and have been set down as fulminant examples of the disease. When these occur during very hot weather, in open yards or fields, there is reason to believe that insolation, acting on a system rendered specially susceptible by the toxic fever, has much to do with the early death. Though seldom observed during life, it has been said that such cases, show extreme dulness, prostration, stupor, weakness, unsteady gait, thirst, hyperthermia, persistent recumbency, and at times red blotching of the skin and even convulsions.

Symptoms in Acute Type. In contrast with erysipelas these may advance slowly and insidiously, there is a lack of the customary life and vivacity, the tail droops, appetite is impaired, the pig creeps

under the litter and lies there, preferably on its belly, a great part of its time, there may even be tremors suggestive of slight chill, when moved it shows weakness, may stagger, or it may have difficulty in rising on its hind limbs and there is encreased thirst and heat of the skin. Even in the absence of shivering or chill, the skin is usually tender to the touch, calling out plaintive grunting or squealing, and the same is often true of manipulation of the belly. The temperature is raised, yet this must be compared with the previous temperature under the conditions in which the pig has been kept. That may have been anywhere from 100° F. in a confined, cold, draughty pen, to 104° F. in a warm, dry pen and with plenty of exercise. In hog cholera it may rise 1° to 3°. The patient is breathless under exertion, the circulation is accelerated and the mucosæ congested.

Sooner or later, (usually by the second or third day) the skin shows an erythematous blush, especially on the ears, breast, belly and inner sides of the thighs and forearms, in greater part effaceable by pressure but promptly reappearing and complicated by darker spots of extravasation which retain their color under pressure. The blush may appear in spots of $\frac{1}{10}$ to $\frac{1}{8}$ inch in diameter, or it may cover the region, or indeed the whole body uniformly. At first of a brighter red it tends to pass in succession through the different shades of purple and violet. Appetite becomes more and more impaired, and in exceptional cases vomiting may occur, but often the pig will drink liquid food to the last. A marked symptom is the enlargement of the inguinal lymph glands, which may even be tender. An early symptom is watering of the eyes, and later a muco-purulent exudate may form, and drying, gum the lids together. An abundant exudate appears on the skin as the disease advances, most abundantly about the eyelids, roots of the ears, axillæ and groins, but often covering the whole body, forming a foul greasy inunction, and later a black scaly covering.

The bowels may be costive at first, with fæces, firm, moulded, and covered with mucous, and this may continue to the end. In most cases, however, about the second or third day they become soft, pultaceous and finally liquid, profuse, fœtid, and mixed with abundance of mucous or even blood. The color varies, they may be whitish, yellowish (on maize diet), red, or black (on swill).

Petechiæ usually form on the mucosæ and small sloughs and ulcers may be found on the lips, tongue or elsewhere on the buccal mucosa.

A cough may be present but is by no means a marked symptom.

Emaciation advances with great rapidity, the patient arches the back, tucks up the abdomen, moves weakly and unsteadily or is unable to rise, and dies in one or several weeks, it may be quietly or in a state of coma, but usually without convulsions.

Symptoms in Subacute and Chronic Forms. In this type the disease may be obscure, and even overlooked, so that infected animals carry the microörganism into fresh herds, without rousing a suspicion as to its true source. In other cases, after a slow and progressive development, it takes on such a distinct pathognomonic character that its diagnosis becomes more easy.

In the slightest cases there may be only a capricious or irregular appetite, drooping tail, enlarged inguinal glands, and a progressive emaciation, with loss of life and strength and occasiona irregularity of the bowels. The greasy exudation on the skin and black scaly encrustation is not uncommon. Such patients usually survive but they are liable to prove unthrifty and unprofitable.

In other cases the pig becomes dull and listless, leaves its fellows, creeps and lies much under the litter, has impaired or irregular appetite, some costiveness followed by a fœtid diarrhœa, abdominal tenderness, enlarged inguinal glands, progressive emaciation, arched loins, hollow flanks, skin exudation, and oftentimes in the end erythematous eruption with petechiæ and black scaly exudate on the skin. It is in these protracted cases especially that the formation and detachment of the necrotic intestinal sloughs take place and these may pass in the fæces as flattened rounded masses or more extensive plaques. Necrotic ulcers are also liable to show on the buccal mucous membrane or skin. The patient may finally die of colliquative diarrhœa, of exhaustion and marasmus, in a state of coma as in the more acute cases. The mortality may be high and the survivors are liable to prove unthrifty and unprofitable.

Diagnosis. With a group of plagues in swine, bearing a strong family resemblance, and maintained by microörganisms, which, though maintaining distinct characters, yet show so much in common that it seems not impossible that they may have been originally derived from a common ancestor, and in face of the not infrequent complication of two of these microbes in one patient, it becomes a task of great difficulty to diagnose at once

the particular outbreak that is met with in the field. In some outbreaks, however, the differential features are clear enough to allow the veterinarian to pronounce at once on the true nature of the disease. In others he must withhold his diagnosis until he can put it to the test of microscopic examination, bacteriological culture, the Widal test, or inoculation.

Hog Cholera may be decided upon, when upon wholesome food, in healthy environment, without any change of food, and in six to fourteen days after the introduction of pigs from outside, or the arrival of strange pigs in the near vicinity, or higher up on the watershed, sickness appears tardily, taking one or two daily, with or without a sudden hyperthermia, petechiæ on nose, eyes, belly, axilla, or groin, a general soreness of the skin and abdomen, stiffness or weakness, hiding much under the litter, enlargement of the lymph glands, costiveness with dark red rectum and glazed dung, followed by a profuse, watery, fœtid, bloody, black or yellow diarrhœa, and death mostly after one or two weeks or more. The absence of cough, and the presence of ulcers bearing necrotic sloughs on the lips, mouth or skin, and above all the presence of the button-like necrotic ulcers on the mucosæ of the cæcum, colon or ileum may be accepted as conclusive evidence on this point. So also its prompt fatality to rodents but not to pigeons.

Swine erysipelas has a much shorter incubation, more rapid and violent onset, deeper, darker congestion of visible mucosæ, more extensive petechiæ of skin, mucosæ, serosæ and tissues generally, a comparative absence of inflammatory and necrotic lesions of bowels, a very early and high mortality in swine, rabbits and pigeons, and a harmlessness toward the inoculated Guinea pig.

Swine plague also shows a shorter incubation, a speedy elevation of temperature, more mucous congestion, less indication of abdominal tenderness or of diarrahœa, more cough, dyspnœa, wheezing and objective symptoms of pulmonary consolidation, less congestion or engorgement of the spleen, or ulceration of the bowels, and finally is very much more fatal to pigeons, and spares neither rabbits nor Guinea pigs.

Widal test. The cessation of movements and the agglutination of the bacilli of hog cholera, noted by Dawson, is a valuable test, but as in the case of typhoid fever in man is not to be implicitly relied on in all cases. Some of the forms of bacillus coli commune and other allied microbes act in a similiar way. It necessi-

tates the maintenance of fresh (24 hours) active, artificial, agar cultures of the hog cholera bacillus and is thus virtually reserved for the bacteriological laboratory. A drop of blood is drawn from the suspected pig smeared very thinly on the cover glass and about ten times the amount of sterile water added. Then the smallest possible addition of the agar culture of the bacillus is made. Immediately, or in ½ hour the bacilli cease their active motility and mass together in clumps in which they can be seen individually clear and distinct but absolutely still and crossing each other in all directions forming a kind of network. A few isolated bacilli remaining in the intervals between the clumps and even showing a slight motility are not to be considered as invalidating the reaction.

The table on the next page will serve to place in contrast the differential phenomena of the diseases caused by bacilli of hog cholera, swine plague and swine erysipelas in uncomplicated infections.

Prevention. As in all other contagious diseases, effective preventive measures imply the destruction of the pathogenic germ and all sanitary measures should aim at the early and final extinction of this organism and its subsequent exclusion from the country. This, however, entails an outlay and governmental control which it seems idle to expect in the very near future, so that palliative measures, and those looking toward success over limited areas must still be resorted to. It should be here distinctly stated, however, that the extinction of a plague, though often the most expensive at the start, is in the end by far the most economical resort.

Boiling all food such as scraps from slaughter houses, butcher shops, and other places using raw hog products, and including kitchen swill, under the strictest precautions against subsequent contamination would all but exterminate the infection and should be compulsory wherever hogs are bred or kept in herds.

Removal of accessory causes. The *health* and *vigor* of the animal exposed is not without its influence in case of attempted invasion by a virus of diminished potency.

Dry, warm beds with plenty of *air* and *light* are essential to vigorous health and the usual damp, filthy, dark pens are depressors of the vital forces and virtually invitations to hog cholera as to other diseases. The close packing of swine under manure or under rotten piles of straw where they often suffocate each other is to be carefully guarded against. It is a sufficient com-

DIFFERENTIAL SYMPTOMS AND PHENOMENA.

Hog Cholera.	Swine Plague.	Rouget.
Incubation 6 days +	Incubation 1 day to +	Incubation 1 day
Mucosæ not necessarily congested	Mucosæ congested	Mucosæ deeply congested, dark red, violet
Petechiæ on snout, eyes, mouth, etc	Petechiæ on snout, eyes, mouth, etc	Petechiæ extensive
Necrotic ulcers on snout, mouth, skin, etc	Necrotic ulcers rare	Necrotic ulcers less frequent
Furred tongue, vomiting common	Vomiting less likely	Vomiting not uncommon
Temperature high in acute cases (104° to 108°)	Temperature high in acute cases	Temperature very high, 107° to 109°
Lies on belly mostly, Abdomen hot, tender	May lie on side	May lie on side
Moves stiffly, feebly, unsteadily with grunting, may be paraplegic	Stiff but less so, Paretic	Stiff, weak, paraplegic
Bowels 1st costive, fæces molded, glazed; 2d or 3d day, or before death, diarrhœa, profuse, watery, fœtid, bloody, black on slops,—yellow on corn (maize)	Diarrhœa less marked, may be entirely absent	Diarrhœa usually sets in
Everted anus dark red	Anus may not be deep red, less everted	Anus may be less red, less everted
Cough often present, hurried breathing	Cough, hard, frequent; wheezing breathing. Auscultation and percussion may show lung consolidation toward lower border. May bleed from nose	Cough absent, save in latter stages with pulmonary consolidation
Spleen slightly enlarged	Spleen usually little altered	Spleen enlarged, soft, grumous
When death is deferred 1 to 2 weeks, necrotic button-like ulcers on ilio-cæcal valve, cæcum, colon, or ileum	Necrotic, button-like ulcers on cæcum rarely marked	Necrotic ulcers on bowels rare
Lobar pneumonia uncommon	Lobar or lobular pneumonia a marked lesion	Pulmonary and enteric inflammation rare
Kills rabbits (5 to 7 days) and Guinea pigs (7 to 12 days) Pigeons sicken but survive	Kills rabbits (1 to 12 days), Guinea pigs (1 to 4 days), pigeon (48 hours)	Kills pigeon (3 to 8 days), rabbit (4 to 8 days), Guinea pig resists
Blood serum of hog cholera patient causes agglutination of bacilli in cultures; (not constant, occurs with other bacilli)		

mentary on this to say that for every killogramme of its body weight, the horse consumes daily 13,272 grammes oxygen, the cow, 11,040 grammes, and the pig, 29,698 grammes. This is in perfect keeping with the high normal temperature maintained by the latter animal. In the interest of health the pig requires twice the breathing space for every 100 lbs. of his weight that is demanded by either ox or horse. What violence is done to this demand of nature in the daily treatment of the hog!

Fresh, sound, wholesome food is no less a *desideratum*. Yet the omnivorous pig is condemned to become the scavenger for the kitchen, the stable, the feeding pen, the slaughter house, the creamery, the sugar works, the brewery and even the rendering works. Whatever is considered unfit for human use is thrown into a swill barrel, and as this is never emptied it becomes the field of endless decompositions with the production of the most varied toxins, ptomaines and enzymes. Many of these chemical toxic products cause gastro-intestinal inflammation with vomiting, bloody diarrhœa and tenesmus, and derangement of the nervous and other functions as manifested in weakness, staggering, dulness, stupor, etc. Death may follow in a few hours and the cases are set down as acute forms of hog cholera, rather than the simple poisoning that they are. All the same they pave the way for the attack of hog cholera if its germ is present even in a form of little potency. All such foods should, on the contrary, be fed fresh and after boiling.

Salt in excess, the *brine of salt meats or fish* (containing toxins,) the *powdered soaps* used in kitchens and added to swill, *mouldy bread, cotton seed meal* fed in any considerable proportion in the food, and even an exclusive diet of *corn* (maize), must be guarded against.

The crowding of many pigs in a small yard where they root continually in each others' droppings and their own, should be avoided. Individual pens, or pens holding two or three only and kept clean are to be preferred, and still more a wide grassy range where they may escape from their own filth. The long feeding trough should be discarded in favor of one into which the pig can introduce his nose only. The nose itself will introduce filth ferments, but, where there are no specific plague-germs, it is the quantity that tells and the exclusion of the foul feet is

an important consideration. To these various poisonous products of saprophytic ferments it often happens that the older swine have by continuous exposure, acquired a comparative immunity, while the young growing pigs perish in large numbers.

Feeding pigs in confinement, without green or animal food is very liable to induce costiveness and indigestion which pave the way for the inroad of the hog cholera germ. A certain allowance of green food, slops, and, above all, a variety of food constituting a well-balanced ration are always desirable.

Again, the constitution of the pig is often material. On the continent of Europe it is the high bred English pigs that suffer most, and in all cases a lack of the rugged vigor attained through an active, open air life lays the system more open to a violent attack. Too close breeding must be similiarly avoided, together with breeding from the immature, the weak and the debilitated. In this connection it is important to rid the herd of parasitisms which not only weaken the system and lessen the power of resistance, but by the bites or the inflammation induced, open channels for the introduction of the hog-cholera bacillus.

Prevent the Introduction of the Bacillus. The above precautions are important in obviating infection and favoring a milder type of the disease when the germ has been introduced, but they are but palliatives at best, and will not hinder the development of a plague in the presence of an active and potent virus. Adopted alone they are worse than useless as a means of extinction of the germ: they tend to preserve it. The exclusion of the hog-cholera germ is the one essential thing in prevention and whatever comes short of this must have at best but a partial effect.

Avoid pens, pastures or streams that drain swine enclosures higher up, Discard all provender or litter that has come in contact with other pigs or their products. Allow no visitors to the herd such as butchers, dealers, drovers, that have habitually come in contact with other herds. Exclude as far as possible domestic animals (dogs, sheep, cattle, fowls, pigeons), and even vehicles coming from places where hogs are kept. Wild animals such as buzzards, and other carrion feeders, must be especially guarded against. Wild rabbits and hares (jack rabbits), skunks, wood chucks minks, rats, and mice should be exterminated. Small birds and flies are difficult to deal with but the breeding of

the latter may be checked by acids, copperas, or sulphites on the manure and the former may even be exterminated when hog-cholera exists in the vicinity.

Sows should not be sent from herd to herd for service or otherwise, and any swine that have been hired out, or sent to an exhibition, and all that are acquired in any manner, should, on arrival, be excluded from the herd and held in quarantine, well apart for three or four weeks, and finally washed with carbolic acid soap before they are admitted.

The pestilential prevalence of hog cholera and other swine plagues to-day is largely the result of the great industrial and commercial activity of modern times. In America the disease was comparatively unknown until after 1830, and in Europe even later. But with the advent of steamboat and railroad, the few pigs raised in separate pens, or secluded localities, and killed and cured near by, gave place to the large herds, sent when fattened to great markets where pigs were collected from distances of many hundreds of miles, the stock animals and the fat occupied in succession the same boats, cars and yards, and, as a matter of course, the virulent germs were concentrated and diffused through the infected places and things. We cannot go back to the antiquated safer methods, but it would be possible to so regulate our commerce, that the evil could be reduced to a minimum. Separate cars, loading banks, chutes, alleyways, and yards can be reserved for fat swine going to immediate slaughter and no animal having passed through any of these should be allowed to be taken out for stock purposes, unless it has been passed through a rigid quarantine. The places and things used for such fat swine should be disinfected at intervals, and the manure and offal should be disinfected, or exposed to a boiling temperature for a sufficient length of time before removal from the premises. Stock swine on their part should be shipped only on a certificate of the complete immunity of the herd and locality from which they come from swine epizootics, and of the roads or vehicles by which they reached the shipping point. They should be debarred from all yards, loading banks and cars or boats used for fat hogs, and admitted only to such as have just passed through a thorough disinfection. They should be sent directly to their destination, or if to a market, for purposes of sale, it

should be well apart from that used for fat swine, and the loading banks, chutes and yards should be entirely distinct and should be thoroughly disinfected on every occasion after use. The millions now lost yearly from swine epizootics might well warrant the inconvenience and expense entailed by such precautions. Heavy penalties should be imposed on those shipping pigs from infected localities, on those making false certificate, and on all who in any way violate the law.

Independently of State or local authorities the stock owner can do much to protect himself. He can make a number of pens large enough to hold 2 or 3 pigs each, safely fenced off from one another and so constructed that no drainage can take place from pen to pen. Then in winter in the absence of flies, and with rats, mice, and birds excluded the opportunity for the extension of infection from pen to pen can be kept at its minimum. All pigs must be kept apart from the manure heap, and in summer the manure must be so treated as to destroy the larvæ of flies. All food and water that might convey infection must be guarded against. Then if one pig is attacked it will only be necessary to destroy it and its two fellows in the same pen, and even if those in adjacent pens are killed or quarantined the loss will be a trifle as compared to the ruin of the whole herd, as usually happens. Prompt disinfection of the pens and manure is imperative, and the same would apply to the person and clothes of the attendant, and to all stable utensils.

Immunization by injection of *sterilized products* of the bacillus, has not proved satisfactory. In 1880 I applied this to two pigs, causing a transient fever, after recovery from which, the subjects resisted exposure to infected pens and pigs, and even virulent inoculations. But they failed to thrive well. Later experiments by Drs. Salmon and DeSchweinitz respectively, also proved unsatisfactory. The latter separated and injected the enzymes, but lost 50 per cent of his cases, the survivors proving immune, with the drawback of troublesome local lesions. The enzymes obtained from cultures in milk could be used safely on guinea pigs in the dose of 0.01 grams and in some cases even up to 0.04 securing immunity. But the great risk of an overdose, the frequent local lesions, and the subsequent unthrift, have prevented the adoption of the method.

Disinfection. The experiments of the Bureau of Animal Industry show that, apart from freezing *four months in the soil*, serves to render the bacillus harmless.

From .75 to 1 per cent of quick lime added to soil in the form of lime water, destroyed the virulence in 11 days.

Lime can be employed as a thick whitewash on pens, fences, yards, etc., the precaution being taken to see that it is newly burned, caustic and applied in sufficient amount. Lime that has been kept absorbs carbon dioxide and loses its disinfectant property. If ¼ lb freshly made chloride of lime is added to each gallon of the caustic lime white wash the certainty of success is insured. Lime water has the advantage of being applicable to grassy surfaces, without proving hurtful to the vegetation. For buildings and yards it furnishes a ready means of estimating the thoroughness of the application.

Sulphuric acid (1 : 100, or 1¼ oz. to 1 gallon) makes a good disinfectant for buildings and yards. Like lime this can be used freely without fear of poisoning the animals.

Carbolic Acid, 5 per cent, can be used with great safety. The Bureau of Animal Industry advises the combination of this with sulphuric acid, which adds greatly to its solubility.

Formalin may be employed, diluted one to forty of the solution (1 per cent of the gas) in buildings and on wood work generally. It may also be applied in the form of gas by heating the solution in closed rooms. Like carbolic acid it is especially applicable to cars, boats, and other vehicles.

Corrosive Sublimate (1 : 500) makes a convenient and cheap disinfectant, with the drawback that it is poisonous, and destructive to metals. *Mercuric Iodide* though more potent is also more expensive. Blue stone (2 : 100) and zinc chloride (10 : 100) are also effective but poisonous.

The failure to *stamp out* hog cholera in England and America has been largely chargeable on the appointment of laymen to do the work of the expert, and no less so on the attempt to deal with the disease in hogs in transit or in the market rather than in the farm where they have been raised or kept. Let the fat and stock markets be kept rigidly apart, together with the means of conveyance to and from these, and let no stock swine start for a market or destination without a certificate of the soundness of the locality

from which they came, and the purity of the means of transit, and we shall have taken a long step toward the final extinction of the pest.

State limits and rights stand in the way of successful work, but this can be partly met by a frontier supervision by national officials, and should be further, by a prompt and hearty coöperation of the sanitary officers of the two states involved. When it becomes possible to trace infecting hogs, back to the infected place in another commonwealth, and punish the offender who shipped them, we shall be within sight of a satisfactory control or extinction of hog cholera.

Extinction of Hog Cholera in Herds and Districts. As in all deadly plagues this should be a recognized governmental function to be carried out at public expense. It is a question of political economy and its neglect is subversive of prosperity not in agriculture alone but in all public industries whose workers must subsist on the fruits of the soil. The $10,000,000 lost yearly by the farming community, is a dead loss, not to agriculture alone, but to the prosperity of the nation, the markets of which would be revived and improved by such a yearly sum expended.

The existence of the disease at any point should be reported by the stockowner or guardian, under penalty in case of failure. When the nature of the outbreak has been certified by the expert, the district should be scheduled, and the herd appraised, slaughtered and all products disposed of in such a way as to prevent any escape of infection. The carcasses may be burned, buried deeply, or boiled and rendered. The buildings, yards, utensils, fences, manure, cesspools, and infected fields should be thoroughly disinfected, or secluded from all animals for a year. The owners of the herd should be indemnified according to appraisement, and not to exceed ¾ths of the actual market value, provision being made that no award shall be made if the herd sickened within a fortnight after their arrival from another State, or in case the owner, has concealed the existence of the illness, or has otherwise deliberately or carelessly contributed to its spread.

Many minor rules and restrictions will be required to fit the general measures to individual cases and local conditions ,and these require the direct supervision of an expert and not of a mere business manager or layman.

Therapeutic Treatment. With state, county or municipal measures for the extinction of hog cholera, treatment is to be condemned, as calculated to encrease and spread the infection. But until the states can be educated out of the past wasteful system, into economical measures of extinction, the swine breeders are entitled to whatever salvage they can secure through therapeutics. For acute cases there is no hope. For the *chronic* a clean, dry, comfortable pen, well disinfected, and a moderate diet of varied and laxative food are essential. Wheat, bran or middlings, with corn, oat, barley or linseed meal may be allowed in form of a mash. A little green vegetable food may be added. Medicinal agents may be used to meet special indications, but when a whole herd must be treated at once, antiseptics and febrifuges have apparently proved the most generally helpful. The Bureau of Animal Industry especially recommends the following: Wood charcoal, sulphur, sodium sulphate and antimony sulphide, of each 1 lb.; sodium chloride, bicarbonate and hyposulphite, of each 2 lbs.; mix thoroughly and add to each feed in ratio with the size of the patient. In suitable cases, this is said to improve the appetite and contribute much to convalescence. Modifications will readily suggest themselves to meet individual conditions and different stages of the disease—antithermics, eliminants, calmatives, stimulants, tonics, etc.

Serum Therapy. This has been especially exploited and advocated in America by De Schweinitz of the Bureau of Animal Industry, and Dr. Peters of Lincoln, Neb. In Europe, Perroncito has prepared an antitoxin. The serum is produced in the body of the cow or other animal which is inoculated repeatedly with gradually encreasing doses of living hog cholera cultures and with solutions of the bacilli and their products, for a period of eight months, or until no reaction takes place from large doses, and the blood serum added to cultures of hog cholera bacilli causes agglutination of the latter. The serum is further tested as to its power of preserving Guinea pigs inoculated with a lethal dose of live hog cholera cultures. After separation from the blood the serum is concentrated until it reaches a standard at which 10 cc. proves curative to a pig of 40 to 60 lbs. weight.

It proves most successful in animals in which the subacute or chronic form of the disease has just begun. One injection only

was given to each animal. Of 1,923 cases treated (1897-8) 30 per cent. died, and 70 per cent. recovered. Of 3,197 in abandoned herds (checks), 81.24 per cent. were lost. (De Schweinitz).

One drawback was found in the short period of immunity secured, the susceptibility reappearing as soon as the antitoxin had been eliminated from the body. This was met in part by using sterilized cultures (toxins) along with the serum.

The use of *antitoxins* is essentially *therapeutic* and cannot be expected to have more than a transient action on the microbe and toxins. The introduction of *toxins* is logical because these would naturally solicit and secure an encreased permanent production of antitoxins, etc., by the body cells. But the toxins produced by the *B. Choleræ Sius* in artificial cultures have failed to establish a satisfactory immunity, and this failure is now explained by the evidence that the fundamental cause of hog cholera is not the B. Ch. Sius, but the invisible, infinitesimal and as yet unseen microbe. This last procures immunity, but as I noted 26 years ago, so few survive, and still fewer thrive after survival, that any method based on this has been eminently unsatisfactory. The most obvious line of promise would be to employ minute doses of the filtrate of hog cholera blood on pigs, the systems of which were kept charged with antitoxins and other defensive matters procured from the highly immunized body of the cow, horse or other large animal.

The fundamental objection is that it entails the preservation, encrease and spread of the poison, and like all temporizing measures, stands as a barrier to the complete extinction of the plague. Giving a protection of uncertain duration, its repetition may be demanded in a few months or a year, and proceeding on the ground that the pest must continue for all time, the apparent economy of the process will prove, in the long run, but a permanent and grievous tax.

The soundest and only truly economical course in dealing with this and other deadly infections of swine is the radical extinction of the germ. When the people can be educated up to this we shall see the dawn of a brilliant future for our animal industries. Until then we must be satisfied to fall back upon, and make the best use of the temporizing measures now in vogue or that may hereafter be devised. Even if it should be shown that hog cholera is at long intervals developed from a 'sport' of the usually harmless bacillus coli commune, or the filtrable microbe the fact remains that its great extensions and the resulting fatality are due to the contagion alone, so that extinction remains the true watchword of success and economy.

SWINE PLAGUE: SEPTICÆMIA HÆMORRHAGICA SUIS.

Definition. Synonym. Bacillus pestis suis, 0.8—1.5μ, nonmotile. Pathogenesis. Accessory causes, as in hog cholera: less vitality than in virus of hog cholera, bacillus in apparently healthy, deadly to birds and rodents. Lesions: like as in hæmorrhagic septicæmia, lungs suffer more than bowels, lymph glands swollen, hæmorrhagic, liver and spleen may seem almost normal, bowels slightly congested marked emaciation. Symptoms: Acute cases like hog cholera, shorter incubation—1 day, troubled breathing when driven, cough, congested petechiated skin, hyperthermia, costiveness followed by diarrhœa. Diagnosis: constancy and predominance of lung lesions and symptoms, nonmotile bacillus with polar staining, not gasogenic with glucose, very fatal to birds and rodents Prevention: as in hog cholera. Immunization. Treatment: as in hog cholera dangerous. Serumtherapy.

Definition. A contagious bacteridian disease of swine, acute or subacute, characterized by a short incubation, hyperthermia, marked congestion of the mucosæ, petechiæ and circumscribed blood extravasations in the skin, subcutis, mucosæ, submucosæ, and tissues, swelling, congestion and petechiation of the lymph glands, and a marked tendency to inflammatory localization in the lungs.

Synonyms. Th. Smith identifies this affection with the "Schweineseuche" of Germany.

Bacteriology. The bacillus of Swine Plague (B. Pestis Suis) has already been described in the differential table of allied bacteria given under hog cholera. It is a short rod, with rounded

ends, 0.8 to 1.5μ × 0.6 to 0.8μ, staining readily in aqueous basic aniline colors and bleaching in Gram's iodine solution. The staining is distinctly polar, the colored portions being more or less crescentic with the concave or straight border turned inward toward the central clear space. It is destitute of flagella and distinctly nonmotile unlike the very active bacillus of hog cholera. It further differs in its growth on potato which is slight, gray and waxy. On gelatine, too, it gives a feeble growth (or none) unlike the brownish colonies of the bacillus of hog cholera. It fails to liquefy gelatin. On agar the growth is more active, being grayish, translucent or brown. Those forming under the surface are like flat horizontal discs with a small microscopic elevation or knob in the center. It grows in milk producing little or no acid and no clot. It produces gas with none of the sugars—glucose, lactose, saccharose—in contrast with the gas production in glucose by the hog cholera bacillus. It shows much less vitality and hardihood than the hog cholera bacillus, growing but feebly between 65° and 70°F., and most actively at 97° to 100° F.; dying in 7 minutes at a temperture of 58° (moist); dying in 3 days when dried, in 4 to 6 days in the soil, in 10 to 15 days in water, and instantly in 0.04 per cent solution of lime water.

The same infinitesimal microbe referred to under hog cholera is believed to be the most essential factor in swine plague as well. This makes the B. Pestis Suis (B. Suisepticus) an accessory but still an important factor.

Pathogenesis. It is pathogenic to swine, hens, pigeons, pheasants, sparrows, mice, guinea pigs, rabbits, cattle, deer, etc., showing not only a wider range than the hog cholera bacillus, but a more deadly action outside the genus suis. Inoculated birds die in 2 days, rabbits in 16 to 20 hours and guinea pigs in 1 to 4 days.

Accessory Causes. These agree in the main with those of hog cholera already described so that it is needless to repeat them here. The principal distinctions depend upon the lesser vitality of the swine plague bacillus outside of the animal body and its, wider range of pathogenesis outside the genus suis. Infecting materials that have been thoroughly dry for a week may be considered harmless, also that which has been more than two weeks in water, and that which has been more than a week in the soil. If, therefore, the buildings have been thoroughly disinfected, the simple disuse of yards and pastures for a fortnight, and of ponds of water for three weeks may suffice. In the case of hog

cholera it may be necessary to abandon such places for 5 months or for the season.

Abandonment by swine is, however, insufficient: all susceptible animals, wild and tame, (see pathogenesis) must be excluded as any one of these may maintain the infection. The preservation of recovered swine on the premises, or the early return of the immune may become a means of preserving the bacillus for the next susceptible pigs that may be introduced. The bacillus of swine plague may be found on the air passages of swine and other animals that are not themselves, at the time, susceptible to the disease, and these animals accordingly become the occasions of what have been thought to be spontaneous outbreaks, and of invasions of fresh herds after the introduction of healthy pigs which have been thought to be beyond suspicion. The danger of the communication of the germ by wild birds and rodents would be enormous, but for the fact that it is so much more deadly to these animals than the microbe of hog cholera, that few survive to maintain the infection. Yet the rule ought to be, to exclude from the fields or premises occupied by new or susceptible pigs, all animals, that may by any possibility become the means of introducing the infection so recently prevalent. Though so easily destroyed when outside the living body, the microbe of swine plague can be carried by the apparently healthy living animal and we must rigidly exclude the possibility of this occurring.

Lesions. *Acute* and *rapidly fatal cases* of swine plague furnish lesions indicative of a hæmorrhagic septicæmia. The abundance of petechiæ on the skin, mucosæ, serosæ, and tissues generally, with circumscribed hæmorrhages, congestions, inflammations and exudations, agree in the main with what is observed in the acute examples of hog cholera. If the congestive or inflammatory lesions, concentrate in the lungs rather than the bowels it assists in the diagnosis of swine plague. The swelling and blood-engorgement of the lymph glands are nearly alike in the more acute types of the two diseases. The spleen is less constantly enlarged than in hog cholera or swine erysipelas. In the *subacute* and *chronic forms* the lesions may be almost entirely confined to the enlarged and congested or hæmorrhagic lymph glands. Usually, however, the lungs are the seat of lobular or lobar pneumonia, affecting by preference the lower portions of the anterior and median lobes, and sometimes also the posterior lobe. The pl[e]

uræ are often involved, showing arborescent congestion, thickening, exudate, false membranes and hydrothorax. The exudate may at times fill up the interlobular connective tissue, even before the pulmonary tissue is materially involved, suggesting a local infection starting at the pleural surface. The hepatised lobule has a general red color varying in depth at different points, and showing lighter yellowish or grayish spots representing the purulent air sacs and terminal bronchia, and necrotic foci. On section the bronchia often yield pus, while the pulmonary tissue oozes a bloody liquid rich in small lymphoid cells.

The liver and spleen may be all but normal, though in a number of cases they may be congested and softened. The stomach and bowels may be virtually sound, or they may show extensive congestion, petechiation and thickening of the mucosa at different points, with, in some cases ulcers, but these latter are mostly excavated and rarely assume the projecting, button-like, laminated form which is so characteristic of chronic hog cholera. Emaciation is a marked feature as in hog-cholera.

Symptoms. In the most acute type these may not differ from those of similar cases of hog-cholera. If there has been any opportunity of estimating the incubation it will be found to have been shorter, the skin and mucosæ have a darker red blush, showing first on the ears, breast, belly and inner sides of the thighs and forearms, the lymph glands are enlarged, and there is cough and dyspnœa if the patient is roused to exertion. The presence of petechiæ on the skin and of a very high temperature (107° to 109°) is to be specially noted. There are great prostration and dulness, complete anorexia, hiding under the litter, indisposition to rise, often weakness, staggering, paresis or even paraplegia, somnolence, and death in coma or convulsions.

In the *subacute* and *protracted* cases, there is the short incubation (1 day), followed by hyperthermia, drooping tail, hiding under the litter, flushed eyes, nose and mouth, impaired appetite, arched loins, hollow flanks, retracted abdomen, cough easily roused by driving, and signs of consolidated lungs in their lower parts (crepitation, suppressed murmur, abdominal or heart sounds etc). After a day or two the skin becomes flushed and together with the visible mucosæ the seat of petechiæ. The superficial lymph glands are enlarged. The bowels are usually confined but

as the disease advances diarrhoea may set in. There is rapid loss of condition, and the patient may die as the result of exhaustion, toxin poisoning, colliquative diarrhœa, or other condition.

Diagnosis during life is based largely on the shorter incubation, the greater reddening of the visible mucosæ, the comparative absence of abdominal tenderness, and offensive diarrhœa, and the constancy of the cough and other symptoms of broncho-pneumonia.

Post-mortem. It is marked by the constancy and predominance of the lung lesions, and the comparative absence of ulceration of the ileum, cæcum and colon, and especially of the projecting, laminated, button-like, necrotic sloughs.

The morphology of the bacillus, its habit of polar staining, its lack of automatic movements, its comparatively poor growth in alkaline bouillon, on potato, gelatine and agar, its inability to ferment glucose with the production of gas, its rapidly fatal action on hens and pigeons as well as on rabbits and guinea pigs serve to distinguish it from the microbe of hog cholera. (See table p. 38).

Prevention. In this connection the reader is referred to the precautions, given under hog cholera, against conditions, hereditary, hygienic, climatic, dietetic, parasitic, etc., which lay the system more open to microbian invasion. In the matter of exclusion of the bacillus, the swine plague germ is less difficult to deal with, because it is so much more easily destroyed. Disinfect the buildings, and all contaminated objects as advised under hog cholera, clean yards, and shut up these and pastures or runs, and all infected water for one month. In case of public market yards and alleys, and railway loading banks, chutes, and cars a thorough cleansing and disinfecting may warrant that they may be put to use again immediately. No animals that occupied the yards before disinfection should be allowed to mingle with the new stock, as they at times carry the microbe, though themselves apparently healthy and immune. All regulations as to railway and boat transit, recommended under hog cholera, are equally applicable to swine plague. The possibility of protecting private herds, by keeping them in special pens holding two or three each, is also the same for swine plague. Boiling of all food is equally imperative.

Immunization is somewhat more promising than in hog cholera.

Metchnikoff and Th. Smith working independently found a fair measure of success in inoculating rabbits and guinea pigs with three to five small injections of sterilized bouillon cultures, agar cultures of blood, intravenously, intra-abdominally or hypodermically. But as applied to swine it has not proved satisfactory, and the irregularity of the results and the tendency to induce unthriftiness have caused it to be abandoned.

Treatment. The therapeutics of swine plague like that of hog cholera is essentially unsatisfactory. Benefit might be derived in individual cases from a careful and judicious use of drugs to meet the special indications, but with the comparatively low value of the individual animal, the certainty of the multiplication of the deadly poison by the preservation of the diseased, and the extreme danger of its diffusion and extension, treatment is anything but commendable.

Serum-therapy has been advocated for years by De Schweinitz, and under the auspices of the bureau of Animal Industry it has been given a wide trial, but it has not met with the full success that was at first claimed for it. The serum is prepared in a similar way to that of hog cholera and is similarly employed. It is open to the same class of objections, and though when skillfully employed it will reduce the mortality, it does not yet seem to have reached the point at which it can be recommended as a profitable investment. Like all temporizing measures it draws attention from the sounder and more economical measure of extinction and is indirectly a means of the perpetuation and even the diffusion of the infection. So long as extinction cannot be secured, this is a less valuable alternative for the adoption of owners of high priced hogs.

MODIFIED AND COMPLEX FEVERS OF SWINE.

Double infections. Varieties of bacillus choleræ suis, and bacillus suispestis. McFadyean's swine fever bacillus. Marseilles swine plague bacillus.

We accept fully the duality of the Hog Cholera and Swine Plague, though this duality has been hotly contested on both sides of the Atlantic. Many of the best observers in Europe now support this position. These include Selander, Bang and

Jensen who are familiar with the *svinpest* (hog cholera) of Scandinavia; Kitt, Friedberger and Fröhner who are familiar with the *Schweineseuche* (swine plague) of Germany; Raccugla, Canova and others in Southern Europe; and Lignieres and others in France who have studied the hog cholera and *pasturellose porcine* (swine plague). The acceptance of a common filtrable microbe approximates the two diseases at their inception.

But the conceded duality of these two diseases as they occur in typical examples in swine, does not account for all the infectious fevers of swine, in which these microbes or others closely allied to them may figure. Salmon, Smith and their coadjutors describe double infections in the same system, in which both the bacillus choleræ suis and the bacillus pestis suis figure, and in which there result a combination of symptoms and lesions, that together represent both of these germs. It may be that one or other of these germs in a given outbreak, shows a predominance in potency so that the symptoms are more characteristic of it than of its companion; it may be that the more potent germ kills the victim, quickly by an acute septicæmia and gross lesions that would apply almost as well to one germ as to the other; or it may be that both act moderately and the attack is protracted with resulting lesions in both lungs and bowels that respectively suggest the plague and the cholera.

Then as regards varieties in the individual germ. Th. Smith recognizes this as a frequent condition and describes no less than seven different types of bacillus choleræ suis which he had studied and which varied in morphology, cultural qualities and virulence. Lignieres found that the virulence especially of bacillus suis pestis is very easily affected by successive passages through the bodies of small experimental animals. We ought not to be surprised then if we find in different epizootics, in different countries and even in the same, bacilli which for the time at least show characteristics different from those to which we have been accustomed. These give us varying phases of septicæmia which however come together in one great class. Two of these types which have been placed on record may be here named. For others see septicæmia hæmorrhagica of cattle and sheep.

McFadyean's Swine Fever Bacillus. The characters of this microbe found constantly by McFadyean in swine fever,

approximates closely to the hog cholera germ in morphology and motility and in its deadly action when eaten, while it approaches toward the swine plague germ in its cultural habits on potato, gelatine and agar, and in alkaline culture liquids, and finally it differs from both in the absence of pathogenesis to Guinea pigs and in its very moderate action on rabbits. The symptoms and lesions of the *swine fever* of Great Britain are those of the *hog cholera* of America rather than of swine plague.

Marseilles Swine Plague Bacillus. This microbe was found by Rietsch and Jobert in a febrile epizootic of swine at Marseilles, and was studied by Caneva and Bunzl-Federn separately. The latter identified it with the bacillus of ferret septicæmia, as described by Eberth and Schimmelbusch. It was longer and thicker than the hog cholera bacillus, twice as long as broad, actively motile, with flagella, and differed from bacillus choleræ suis, in its polar staining, its free growth in acid media, in acidifying and coagulating milk, and in its forming both indol and phenol in peptonized bouillon.

In this case the source of the disease was in importations from Africa (Fouquet), and it spread widely in Southern France for nine months. It proved almost constantly fatal, in from four days to two or three weeks. The symptoms were weakness especially in the hind limbs, with more or less fever, constipation often followed by diarrhœa, an infrequent cough, and red blotches on the skin. In chronic cases ulcers formed in the mouth and intestines especially the cæcum and colon. Appetite was often retained to the end. The young, under a year old, were the chief sufferers. It made 20,000 victims in several months in the province of Bouches-du-Rhone.

SEPTICÆMIA HÆMORRHAGICA OF BOVINE ANIMALS.

Synonyms. Definition Historic notes. Resemblance to black quarter. Bacteriology; saprophytic cocco-bacillus, non-motile, ærobic, related to microbe of swine plague, chicken cholera, and rabbit septicæmia Pathogenic to deer, buffalo, cattle, horses, swine, rabbits, rats, mice, goats, and sheep. Variability. Vitality: great in soil, dies in 6 to 20 days when dried and quickly in antiseptics, resistant to heat. Accessory causes: rise of soil water in winter or in spring, drying of marshes in summer, wet, rich, swampy, mucky soils; youth, gregariousness, carnivorous habit, insects, vermin, wild animals and birds, epizoa, entozoa, wire fences, wounds of all kinds, hard, woody provender; inoculations in wounds the most fatal. Symptoms: superficial with hyperthermia, functional disorder; muscular tremors; violet mucosæ; segregation; swelling in intermaxillary space, tongue, throat, neck, dewlap, or elsewhere, not pitting on pressure. Petechiæ. Death in six hours to four days; thoracic form kills in four to eight days; abdominal form with colics, and bloody often frothy fœtid fæces. Chronic forms usually pulmonary. Lesions: straw colored exudations subcutem or intra muscular; blood extravasations; in lungs resembles lung plague; on bowels blood effusions, and exudates; softened, blood-stained lymph glands. Spleen usually normal in size. Blood black. Petechiæ extensive. Chronic lesions. Bacillus in exudate, blood and bronchial mucus. Diagnosis: from anthrax, black quarter, lung plague, rinderpest, and maliguant œdema. Mortality 50 to 80 per cent. Prevention: isolate and kill affected; destroy or disinfect carcasses and infected things and places, feeding and drinking troughs and manure. Close and drain infected fields. Immunization, by three inoculations with cultures made at a high temperature (86° to 90°) in free air; or with virus that has been grown in pigeon. In case of deer, drive a few days into a non-infecting enclosure, and then on to a sound range. Treatment.

Synonyms. Wild—und Rinderseuche (Bollinger), Buffalo Disease, Barbone (Oreste and Armanni), Cornstalk Disease (Billings, Moore), Sporadic Pneumonia (Smith), Pneumoenteritis (Galtier), Pasteurellosis Bovina (Lignieres).

Definition. An acute bacteridian disease of domestic and wild herbivora and swine, characterized by sudden onset, rapid and fatal course, marked hyperthermia, accelerated breathing and pulse, and extensive gelatinoid or sanguineous extravasation in the intermaxillary space, tongue, skin, subcutaneous or intermuscular connective tissue, lungs, pleura, pericardium or intestine.

Historic Notes. It is almost certain that in earlier times this affection was often mistaken for gloss-anthrax, blackquarter, or

even lung plague. Metaxa, in 1816 in Italy, manifestly describes it. Oreste and Armanni, in 1882 and 1887, traced Italian cases to the microbe. In 1854 it destroyed many cattle and deer in England (Veterinarian). In 1878 Bollinger records its great fatality among the deer, wild boars, cattle and horses in and near the royal parks at Munich, and for a number of years after in Bavaria. In 1885 Kitt saw a wide-spread epizöotic in Simbach. Friedberger records its presence in Schlüchtern, Prussia, in 1885–6, Condamine in Cochin China in 1868, and Guillbeau and Hess in Switzerland in 1894. In 1898 Pease recorded its prevalence in buffalo and cattle in Hindustan. In America, what appears to be the same affection is noted as corn-fodder disease in Nebraska (Billings), as Wildseuche in Tennessee (Norgaard), and as hæmorrhagica septicæmia in Minnesota (Wilson and Brimhall, Reynolds). I have repeatedly met with the affection in New York in cows arriving from the west, and in the indigenous cattle on wet, mucky, undrained land in spring, about the period of the melting snows.

Bacteriology. The essential cause of the disease is a saprophytic cocco-bacillus, (B. Bovisepticus), ovoid, with rounded ends, about 1 μ long by 0.3 to 0.6 μ broad, but showing involution forms and a variable size. It is non-motile (Kitt claims motility), ærobic (facultative anærobic), takes a polar stain with clear centre in aniline colors, bleaches in Gram's (1) solution, shows neither spores nor flagella, grows readily in bouillon, on gelatine, (a bluish transparent layer without liquefying), serum at 98.6° F., milk (without acidifying or coagulating), and alkaline potato (not on the acid). The cultures have a peculiar odor and yield no indol.

The microbe shows a very close relationship with those of swine plague, chicken cholera and rabbit septicæmia, but it sometimes differs in showing little or no pathogensis for the Guinea-pig.

Animals susceptible. It is pathogenic to deer, buffalo, cattle, horses, swine, rabbits, rats, mice, and to a lesser extent to goats and sheep.

The *pathogensis* varies with the immediate source of the microbe. When obtained from cattle a drop of blood kills rabbits in twelve to twenty hours, with intense hæmorrhagic laryngitis and tracheitis. Guinea-pigs die in forty to eighty hours. When obtained from the buffalo it killed horse, ox, or pig in twenty to

forty-eight hours. That obtained from barbone (buffalo) appears to be more potent than that from septicæmia hæmorrhagica (cattle).

Vitality of the microbe. Simple drying destroys virulence in six to twenty-two days. Virulence is retained for nine days in putrid flesh. It is preserved, and the microbe multiplies in soil or water containing organic matter and nitrates. It is easily destroyed by ordinary antiseptics, 1 : 5000 of mercuric chloride destroying its vitality in one minute (Hueppe). On the contrary it shows a great resistance to changes of temperature. It grows in the soil at 55° to 60° F. (Hueppe), and in old cultures may resist for an hour a temperature of 175° to 195° (Oreste and Armanni).

Accessory Causes. These are such conditions as favor transmission of, or receptivity to the microbe. In Southern France the disease is most common in the winter months, probably because the soil water rises then; on the Roman marshes and in Minn. it was noted from August to December: on the other hand, it prevails especially from May to October, when the water, is lowest and most impure. In New York I have seen it especially at the breaking up of the winter frosts, when the water, pent up in the rich organic soils, is suddenly released. It is pre-eminently the disease of wet soils, rich in the debris of decomposing organic matter, of the rich prairies and bottom lands of the Mississippi Valley, of springy, swampy or mucky soils elsewhere, of the Pontine marshes at Rome, of the Delta of the Nile, of the rich virgin soils in Asia. Youth has the greatest receptivity, the older animals having probably acquired immunity through an earlier attack. The animals that live in herds infect each other by contact, fighting, licking, etc., others are affected by eating the vegetation or drinking the water soiled by the diseased, wild boars by eating the carcasses, and all animals by the attacks of biting or blood-sucking insects which have just come from the diseased. It is claimed that the infection is carried by men and animals, and by the sale in villages of the flesh of infected animals. Dogs, wolves, foxes, and other carnivorous animals and birds will also carry the infection for long distances. Finally, it will travel to a greater or lesser distance with running water.

The entrance of the microbe by wounds must always be counted on, and explains the casual inoculations, by bites of dogs, insects, worms, by barbed wire fences, by wounds with horns,

tusks, or feet, by nails, etc., and in winter by hard, woody aliment scratching the lips, mouth, fauces or pharynx. Shedding of teeth, diseased teeth or gums, and everything that causes abrasion of the alimentary mucosa must be admitted into the list of causes. Infected traumatisms of any kind, like intratracheal and intravenous inoculations usually prove fatal, while infection by ingestion is not necessarily so. The pathogenic potency appears to be impaired in the stomach or intestines.

Symptoms. These vary widely according to the subject, the seat of infection and the violence of the attack. They may be classed under three principal heads: *superficial, thoracic, and intestinal*, and in addition into *acute* and *chronic* cases.

In the *superficial external or cutaneous form* there is usually a sudden onset with high fever (104° to 107° F.), accelerated pulse, (70 to 90), and breathing (24 to 50), anorexia, suspended rumination, muscular tremors or shivering, staring coat, dry, hot muzzle, burning of ears, horns and hoofs, suppression of milk, and more or less stringy salivation. The visible mucosæ are of a deep red or violet tinge, and the patient will often remain apart by himself when the herd has moved elsewhere. Soon there develops a tense, hard, hot, painful swelling of the intermaxillary space, tongue, throat, neck, dewlap or elsewhere, amounting to perhaps six inches in thickness, extremely resistant and not usually indented on pressure with the finger. The breathing becomes stertorous and deglutition difficult or impossible. The mouth is hot and filled with tenacious saliva, and the tongue may hang pendant while on its borders and lower surface are projections of the mucosa swollen by infiltration, yellowish and semi-transparent, or blood-stained. At other points petechiæ are more or less abundant.

Death may take place from pharyngeal obstruction or closure, or as the disease advances, there may be indications of implication of the viscera, of the chest or abdomen: encreasingly difficult breathing, a mucous or suffocative cough, colicy pains, tenesmus, and the passage of moulded glazed fæces, of pseudo-membranous casts, or of profuse liquid stools. The animal may move the hind feet uneasily, lie down and rise alternately, may remain persistently recumbent until death, or he may stand up until he falls to perish of asphyxia. Death may occur in six hours, or may be delayed four days.

In the *thoracic form* the extreme hyperthermia is complicated by early lesions in the lungs, while the muscular or cutaneous ones are omitted or deferred. So long as the lesions are confined to the chest, they are betrayed by hurried and even oppressed breathing or dyspnœa, a frequent, moist, suffocative cough, persistent standing to favor respiration, and there are the percussion and auscultation indications of consolidated lungs or hydrothorax. The mucosæ are usually of a darker red, than in the external form, cyanotic indeed, and the peculiar asphyxial position, with legs apart, head extended, dilated nostrils and open mouth may be very significant. These symptoms are likely to be modified or supplemented before death, by those caused by intestinal or renal disorder. In the thoracic form in young animals death by suffocation may occur in a few hours, but more commonly the disease progresses slowly and a fatal result is not reached until the fourth day or even the eighth. This form is common in the deer, but less so in cattle.

In the *intestinal* or *abdominal form* the usual sudden onset and high fever, are complicated by inappetence, tympany, rumbling of the bowels, uneasy movements of the hind feet, perhaps twisting of the tail, looking at the flanks, and even lying down and rising. There is frequent, violent straining with the passage of fæces at first glazed, later streaked with blood, or mixed with pseudo-membranous casts, and very soon soft, watery, frothy and fœtid. These are usually black or reddish-black from contained blood.

The urine may also be blood-stained.

Before death, complications on the lungs or skin will often come in to assist in diagnosis.

In the *chronic* and *subacute types* the lesions are often concentrated on the lungs, and there are a moderate fever, cough, hurried breathing under exertion, dulness on percussion over limited pulmonary areas, blowing sounds, mucous râles, crepitations and more or less muco-purulent expectoration. These phenomena are all the more significant if complicated by digestive disorders, costiveness, fœtid mucous diarrhœa, tympany, or by the eruption of the superficial swellings.

Lesions. The swellings on or *under the skin* or among the *muscles* show extensive straw-colored exudations, colored at points with blood, with enlargement, infiltration and staining of the

adjacent lymph glands. On the chest walls the sero-sanguineous exudate may extend from the root of the lungs, through the intercostal spaces to the skin in the breast, the axilla and behind. The tongue is often enormously swollen and black, charged with extensive blood extravasations in addition to the yellowish exudate. Along its sides and on its lower surface, the mucosa stands out in projecting masses of yellowish infiltration, which may show equally on the fauces, pharynx, larynx, trachea and bronchi.

In the *lungs* the pleuræ and subpleural and interlobular tissue are extensively infiltrated and thickened by a profuse yellowish serous or sero-sanguineous exudate, so that the appearance may closely resemble that of lung plague. The lung tissue is consolidated, hepatized and dark red, with at some points emphysema. The pleural sac is usually filled by a serous or bloody effusion (2 to 25 quarts) and there is often extensive implication of the pericardium. The tracheo-bronchial mucosa and bronchial glands show extensive infiltration and thickening.

In the *abdomen* are found extensive infiltrations and blood extravasations in the mucosa and submucosa of the stomachs and intestines, softening and shedding of the epithelium, infiltrations of the peritoneum, diaphragm, and sublumbar adipose tissue, and softening and degeneration of the liver and kidneys. The intestinal gastric and mesenteric glands are usually infiltrated, softened and bloodstained. Engorgement of the spleen is exceptional.

The *blood* is very black but not usually materially changed in consistency nor coagulability. Petechiation of the different serosæ and other tissues is a prominent feature.

In *chronic cases* the lesions are mostly shown in the lungs and lymph glands. The lungs show circumscribed lobular islets of congestion, induration or caseation, offering a suggestion of tubercle, which is all the more deceptive when cretifaction has set in. The caseous centres may vary in size from a pea to a walnut, and some may have ruptured to form a vomica discharging into a bronchium. Bronchia leading to affected lobules are blocked with mucopurulent matter, yellowish, thick and tenacious, and their mucosa is thickened and puckered. The enlarged lymph glands are especially those of the bronchia, trachea, mediastinum, bowels, mesentery and sublumbar region.

The bacillus is present in the exudate but is especially abundant in the blood, and in the chronic cases in the bronchial mucous.

Diagnosis. From *anthrax* (gloss anthrax) this affection is easily distinguished by the absence, from the blood and exudates, of the large, square ended anthrax bacillus, by the absence of enlargement and blood-engorgement of the spleen, and of the softness and diffluence of the blood clot which characterize anthrax. Swine which are with difficulty inoculated with anthrax are very susceptible to hæmorrhagic septicæmia. Sheep which are very receptive to anthrax are somewhat refactory to the disease now in hand. Pigeons resist anthrax but readily contract septicæmia hæmorrhagica.

From *black quarter* it is readily distinguished by the absence of emphysema and crepitation and of a secondary cooling in the external swellings, by the presence of the germ in abundance in the blood, by its smaller size, its bipolar staining, and its lack of motility and of spores. Inoculation with black quarter bacillus kills the guinea pig, but spares the pigeon.

From *lung plague* it is distinguished by the suddenness of its attack and rapidity of its progress to a fatal issue; by the usual coincidence of skin and bowel lesions, while the lung plague affects the chest only; by its communicability to pigs, sheep, pigeons, and even horses, which are all immune from lung plague; and by the usual absence of lung lesions of different ages, which are so characteristic of lung plague. The abundance in the blood, of the cocco-bacillus with bipolar staining in hæmorrhagic septicæmia is characteristic. *Lung plague* spreads slowly to exposed cattle, but spares all other domestic animals.

From *Rinderpest* it is differentiated by the history of its advent, by the presence of the surface œdematous swellings, by the absence of the whitish epithelial concretions on the mouth or vulva, and of the deep dark portwine discolorations of the mucosæ of the mouth, rectum and vulva, and by the fact of its inoculability on domestic animals generally. Rinderpest spreads rapidly to all exposed ruminants, but spares pigs, rabbits, Guinea pigs, horses and birds.

From *malignant œdema* it differs in its inoculability on the surface in place of subcutaneously only, in the presence of the cocco-bacillus in the blood during life, whereas in malignant œdema the germ is confined to the local lesion, in the absence of crepitation, which may be present in the swelling of œdema, in

the greater facility with which cultures can be made of the septicæmic cocco-bacillus and in the absence of gas production in such cultures. The malignant œdema comes from a single accidental deep inoculation from almost any rich soil, and is not a malady spreading widely and generally on given limited damp, rich lands which have become infected. Finally the cocco-bacillus of septicæmia hæmorrhagica is found singly in the blood or exudate, whereas the microbes of malignant œdema may be found in form of sporeless filaments intermingled with the bacilli.

Mortality. The hæmorrhagic septicæmia of cattle cuts off from 50 to 80 per cent. of the animals attacked.

Prevention. The first consideration is to isolate and kill all the affected animals, to destroy the carcasses by burning or boiling and to burn or disinfect all objects that may have become contaminated. The buildings, yards, and fences, must be disinfected, and as the bacillus is very resistant a solution of corrosive sublimate and sodium chloride, a drachm of each to the gallon of water may be freely used after thorough cleansing. Or a white wash containing ¼ lb. chloride of lime to each gallon may be substituted. Feeding and drinking troughs may be burned. Manure may be freely treated with sulphuric acid. Infected fields should be closed for years and if possible drained.

Immunization of buffalo and sheep has been secured by making cultures of the microbe in free air at 86° to 90° F. and inoculating the animals with the weakened virus, on three successive occasions with intervals of several days. It induces a transient fever, with no serious phenomena (Oreste and Armanni). A second available method is to pass the virus through the system of the pigeon and inoculate with the pigeon's blood, on three successive occasions, the animals to be protected. It is manifestly impossible to put such immunizing methods in force on wild deer, and for these probably the best course is to drive them from the infected range, to an uninfected one, having retained them for a few days interval in a confined area, to allow of any already infected animals developing the disease. A similar avoidance of waters running from the infected tract is imperative.

Treatment, has been unsuccessful. Friedberger failed with hypodermic injection of carbolic acid, and internal administration of salicylic acid. Gal gave subcutem 5 per cent. solution

of creolin, and doses of 1½ oz. of the same agent by the mouth. Five buffaloes out of seventeen recovered. Friedberger suggests deep incisions of the swellings so as to admit the air, and treatment of the wounds with strong antiseptics.

SUBACUTE AND CHRONIC SEPTICÆMIA HÆMORRHAGICA OF CATTLE.

Synonyms. Entequé, Entejado, Entecado, Pining, Pasteurellosis.

Definition. A subacute or chronic affection of cattle (horse and sheep) attributed to a Pasteurella and manifested by nervous disorder marked hyperthermia and diarrhœa and in more tardy cases by intermittent diarrhœa, advancing emaciation, anæmia, debility and marasmus.

History. Montfallet met with the affection in Chili, in 1896, in cattle imported from Argentina, and Lignieres in 1898, recorded its extensive prevalence in the Argentine Republic. It should be looked for in the tropical and subtropical states of America and elsewhere.

Bacteriology. Lignieres found in the blood and affected tissues a cocco-bacillus resembling that of fowl cholera, but shorter and more slender, like the Pasteurella of influenza in the horse. This stains slowly, best in gentian violet and fuchsin, coloring the poles in the rounded ends, and leaving the median portion without color, the recognized habit of the Pasteurella. It is ærobic, growing best at 37° to 38° C. in neutral or feebly alkaline peptonized or serum bouillon which it renders slightly turbid in 20 hours. Cultures in gelatine give in 3 or 4 hours colonies as translucent bluish points, like millet seed. It does not coagulate milk nor grow on potato. The microbe is found with difficulty or not at all in chronic cases, the theory of Lignieres and Nocard being that the action of the Pasteurella is to enfeeble the tissues by its toxins, so that they can no longer resist the attacks of other microbes, and these accordingly are met with abundantly in the chronic cases. The cocco-bacillus as the essential factor would be more certain, if it persisted in the system throughout, or if the later phenomena, in its absence were more evidently the product of the secondary microbe, still present. Cattle die much

more quickly from artificial pure cultures than from the casual disease.

Susceptible Animals. Cultures have been successfully inoculated on ox, horse, sheep, guinea pig, rabbit and white mouse. It occurs casually in horse and sheep.

Symptoms. Intestinal Form. The young cattle (1 to 2 years) show slight diarrhœa for a time, with soiling of the hips, tail and hocks, before there is any loss of condition, appetite or general health. For some days the stools become increasingly frequent, and fluid, dark green and fœtid, and then the abdomen is tucked up, the back arched, the movements weak and tardy and the spirits depressed. There are now observed advancing emaciation, pallid mucosæ, sunken eyes, and there may even be dropsy between the maxillæ. The victim leaves its fellows and is found apart lying with the head bent on the neck, the hair erect, and the skin scurfy and hidebound. The diarrhœa, which tends to constant encrease, and may show intermissions, becomes frothy but rarely bloody.

A measure of appetite is retained but emaciation, anæmia, debility and exhaustion advance with great rapidity and the animal dies in three or four weeks in a state of marasmus. When intermissions are prolonged death may be deferred for months.

Symptoms. Wasting Form. This may begin like the intestinal form, followed by a marked subsidence of the local digestive troubles. The appetite, though not lost nor even diminished, is depraved, the victim chewing bones and other objects; the skin becomes dry, scurfy and hidebound, the hair stands erect, the back is arched and the belly tucked up, emaciation steadily encreases becoming extreme, the animal is weak and spiritless, and the eyes are sunken and pale. The breathing is short and accelerated, and the pulse rapid small and weak, even imperceptible. When lassoed the animal resists feebly and its bellow is low and weak. Infective arthritis is common, rendering the animal quite helpless. The progress may be a gradually advancing anæmia and debility to an early death, but the majority show a favorable arrest, so that they may even breed and rear calves before the fatal result follows. A gradual wasting extending over years is a main feature.

Lesions. Intestinal Form. These are mainly shown in the

anæmic, pale, and wasted conditions of the body generally, and the numerous minute centers of purulent infection. The stomachs are well filled, the intestines contain pultaceous and gaseous matters, the glands of the duodenum, of Lieberkuhn and of Peyer are gorged and when compressed, the latter throw out drops of white purulent matter. The liver is normal, the spleen atrophied, and the kidneys pale and fibrous. The chest shows pleurisies with extensive exudation, sometimes pneumonia or broncho-pneumonia, or atelectasis or even sclerosis.

Lesions. Wasting Form. The carcase shows extreme emaciation, absence of fat, bloodlessness, with atrophy and even cirrhosis of liver, spleen, kidneys and other internal organs. The mesenteric glands are large, soft and very watery. In the lungs the most characteristic lesions are calcifications surrounding the alveoli and bronchioles, which have a branching arrangement, crepitate when pressed under the finger, and strongly fix aniline dyes. In the large arteries extending outward from the semilunar valves on both sides, the intima is thickened and raised in irregular patches which may become soft like atheroma, or hard and cartilaginous, or calcified. The auriculo-ventricular valves may even be involved and the inner coat of the auricle, especially the right. In some cases the arteries generally are calcified even to the smaller trunks, and this may be felt in the carotids during life.

Prevention. The most promising course is to segregate the affected animals where their products cannot reach places accessible to other cattle, and to exclude sound cattle from already infected fields until years after drainage. The obvious precaution of sterilizing infected carcases and manure is demanded.

Treatment. Medicinal treatment fails notably in checking the diarrhœa. Yet Lignieres claims complete success from intravenous injection of 1 to 1½ qt. of the following: boiled water 1000 grams: common salt 9 grams: soda sulphate 4 grams. A second injection is made if diarrhœa persists. Improvement appears after 48 hours, and in 5 to 10 days the bowels are normal. This will temporarily invigorate, but will the improvement last?

SEPTICÆMIA HÆMORRHAGICA OF THE SHEEP: LOMBRIZ.

Synonyms. Definition. Geographical distribution, Argentina, France, etc. Causes: bacillus; intravenously, etc., youth, verminous affections, low condition. Bacteriology: ovoid bacillus with polar stain, bleached by Gram's solution, ærobic, non-motile. Symptoms: Chronic form in summer, diarrhœa, arched back, stiffness, emaciation, flattened wool, segregation, impaired or depraved appetite, shedding wool, anæmic skin, dependent dropsies, sunken eyes, weak small pulse, temperature variable—elevated, nasal and buccal discharge, weakness, paresis, dullness, torpor, lung symptoms, arthritis. Diagnosis: from distomatosis and strongylosis. Acute form with high fever, constitutional disorder, colics, diarrhœa, death in 24 to 36 hours, subacute form. Lesions: black blood, congestions, and general petechiæ. Lungs, liver, kidneys and spleen, congested, swollen. Subacute cases have lighter blood, and lesions. Chronic cases anæmic, blood diffluent, lymph glands enlarged, congested; connective tissues and serous cavities dropsical. gastric, intestinal and hepatic worms, spleen shrunken. Mortality: great in acute, less in chronic. Prevention: segregation, exclusion of all sheep from unknown or suspected flocks, antiseptic dip and quarantine for new purchases, expose a few as a test; cleanliness. disinfectants, avoid watershed from infected lands, wide range, out-door life, generous diet, remove weak, emaciated, anæmic. Immunization. Treatment.

Synonyms. Pasteurellosis Ovina. Infectious Pneumo-Enteritis.

Definition. An infectious febrile affection of the sheep, chronic or acute, characterized by dulness, stiffness, or paresis, anorexia, thirst, disorder of the breathing and digestive organs, black diffluent blood, petechiæ, reddish effusions in the serosæ or connective tissue, and congestive or inflammatory lesions of the lungs, liver, kidneys and intestines. The presence of a cocco-bacillus (diplo-coccus, strepto-cocco-bacillus, Pasteurellosa) in the lesions is especially characteristic.

Geographical Distribution. Though Lignieres first demonstrated this as a bacteridian disease in the Argentine Republic, he was, after his return, able to identify the same affection in the flocks of almost every department of France, in newly imported English Lincolns, and German Merinos, so that there can be little doubt that the malady exists in all or nearly all countries engaged in sheep husbandry, though it has been usually attributed to parasitisms of the lungs, liver or alimentary canal alone.

Causes. The essential cause is manifestly the bacillus, which Lignieres has isolated, cultivated in vitro, and successfuly inoculated intravenously in the sheep, which he also infected by feeding the pure cultures. Intravenously it proved fatal to Guinea pig, rabbit, pigeon, chicken, rat, mouse, horse, ass and ox. Yet many other accessory causes must be admitted as operating in different cases.

Youth shows the greatest susceptibility whether the victim be mammal or bird. So marked is this influence that the principle sufferers are lambs just weaned, or yearlings. Yet mature animals, that are debilitated from any cause, also fall victims. The measure of immunity usually noticed in mature sheep may well be attributed to a previous mild and non-fatal attack of the bacillus.

Verminous affections are undoubtedly predisposing causes, hence the common practice of attributing the malady to the worms alone. This again in part explains the susceptibility of the young which so often harbor worms to a dangerous extent. It seems to matter less what worms are present than, that they are in sufficient numbers to greatly deteriorate the health. It is noticeable, however, that those worms that make breaches in the mucosæ, have been noted as infesting the victims of this malady. In the stomach worms sent from Argentina, Railliet identified Strongylus Contortus, S. circumcentus, and S. instabilis, and in the duodenum S. filicollis and S. Curticei. These, like the distomata often found in the liver, are blood suckers and not only render the animal anæmic, but make numerous perforations to act as infection-atria. The various lung worms, encysting themselves in the air sacs and determining local congestions may act in the same way, opening channels for the entrance of the microbe.

Low condition or a *low tone* of health from any cause predisposes. Old, worn out animals, ewes in lamb, or those just lambed, sheep that have been shut up and denied proper exercise in winter, those on poor feeding and perhaps nursing twins, those that have suffered from any debilitating disease of any kind are especially obnoxious to a dangerous attack.

Microbiology. The microbe, which Lignieres found in the pulmonary lesions, is one of the colon group of pathogenic bacteria that have been classed together as *pasteurella.* It usually appears as a very minute ovoid bacillus which stains promptly

and deeply at the poles in fuchsin or gentian violet, leaving a clear median part, so that it seems a *diplococcus*. It bleaches readily in Gram's solution. Its form varies in different culture media sometimes showing long *bacilli*, and sometimes *streptococco-bacilli*, but the usual and characteristic appearance is that of a *cocco-bacillus*, and to this it constantly returns. The microbe is ærobic and nonmotile (the slow zig-zag motion sometimes seen does not seem to be automatic). In peptonised bouillon it produces opacity in 18 hours, or in simple bouillon in 24 to 48 hours, the best temperature being 100° F. Gelatine plate cultures are slow because of the compulsory low temperature, yet in 36 to 48 hours it forms pale blue, translucent, round colonies the size of a pin head. It never liquefies. In coagulated blood serum it forms only a thin transparent pellicle hardly visible, and there is no growth on potato.

Symptoms: Chronic Form. In Argentina, Lignieres observed the disease especially during the hot summer months (December to May), and after weaning in the lambs. This may be from the marked change of food, from the greater activity of microbian life at this season, from the exhausting effect of the heat, or from a combination of two or more of these conditions. It appears alike in the sheepfold, and on the open prairie. In considerable flocks the symptoms may be at first overlooked, so that the death of several sheep may be the first thing to draw attention. Then a certain number are found to scour, arch the back, walk stiffly, lose condition, and have the wool flattened and devoid of yolk (*clapped* wool). The sheep may be dull, lagging behind its fellows, or lying apart by itself, ruminating infrequently and for shorter periods than natural, and there may be inappetence, or depraved appetite (eating earth), though some eat well to the end. Irregular and at intervals capricious appetite is a frequent condition. When caught and examined, the wool is easily torn out, the muscles are soft and wasted, (the leg muscles may have practically disappeared), the bones stand out at all points, the skin is pale, thin, bloodless and devoid of its subcutaneous fat (paper skin), there may be œdemas along the ventral aspect of the body, pitting on pressure, and between the branches of the lower jaw (poked), the eyes are sunken, the conjunctiva may be puffy and œdematous, but like the muzzle and mouth

they are pale and anæmic and the pulse is small, though the excitement may have roused cardiac palpitations. The temperature varies from time to time often reaching 105° or 106° F. There is liable to be a mucopurulent discharge from nose and mouth especially noticeable during drinking. As the disease advances the subject becomes weak, paretic, dull and stupid; it remains down without interest enough to seek food, though still eating if it is brought to it. The head is usually rested on the flank, and the animal often lies so for days in a state of semi-stupor without disposition or ability to rise, paretic or paraplegic. Auscultation may sometimes detect a mucous râle or crepitus, and percussion a flatness of sound over some part of the lung. Chronic arthritis is an occasional symptom.

Diagnosis. The symptoms closely resemble those of distomatosis or strongylosis, and the disease is often complicated with one or more of these, so that it may become difficult to judge how much is due to the microbian infection and how much to the helminthiasis. The presence, continuously or intermittently, of the hyperthermia is almost pathognomonic of the operation of the microbe.

Acute Form. This has been particularly observed in the ewe just after lambing, when the system is especially susceptible to microbian invasion, and little able to cope with it. There are hot ears, nose and feet, temperature of 104° to 106° F., accelerated pulse and breathing, anorexia, ardent thirst, deeply congested mucosæ, colicy pains, pawing the ground, frothy or bloody diarrhœa, arched back, pendent head, ears and eyelids, muscular trembling, albuminous urine, plaintive cries, dark red vaginal discharge, mucopurulent or glairy nasal discharge, and death in 24 to 36 hours. Such animals may be in fair condition or even fat, no time having been allowed for emaciation.

In other cases death may be delayed for three or four weeks, with the same general symptoms, only less marked. In such cases, pregnant ewes are likely to abort, and the lambs are born dead, or prove weak and listless, and die when a few days old. Some have too little energy to suck; others suck heartily but perish all the same on the second or third day, after diarrhœa, thirst, hyperthermia, prostration, and stupor.

Lesions. These vary according to the type. In the *rapidly*

fatal cases there is dark colored blood, with congestion of the serous and mucous membranes, which, together with the skin and often the solid tissues, are covered with petechiæ, and even circumscribed hæmorrhages. The lungs, liver, kidneys, spleen, and many of the lymph glands are congested and swollen, seeming at times of a black hue as if blood-saturated. The lesions, indeed, indicate an acute septicæmia.

In *cases that have survived three or four weeks*, the morbid changes are slighter, the blood is brighter in tint, and the congestions less deep in color, ecchymoses may be especially confined to the heart, abomasum and small intestines, which may also show hæmorrhages. Enlargement and congestion of the lymph glands are the rule, while pulmonary consolidation and gastro-intestinal mucous inflammations are frequently found. As in the more acute types the urine is albuminous.

In *chronic cases* the anæmia is prominent. The clot is soft, relatively small, elastic and black, the serum is relatively very abundant and pale. The red globules are greatly reduced in numbers, and there are a number of giant cells which stain deeply as in chlorosis. The lymph glands are usually enlarged, softened and slightly congested but rarely the seat of blood extravasation. The tissues generally are pallid, soft and shrunken. There is a marked absence of subcutaneous and intermuscular fat, while the connective tissue is more or less infiltrated with a transparent, watery lymph. The serous cavities usually contain more than the normal amount of fluid, transparent or straw-colored, and with few globules or granules. Congestions and even shreds of false membrane are sometimes present on the serosa. In some cases the lungs and bronchia are the seat of inflammatory exudates, causing nodular consolidations of from one-half to one inch in diameter. Not unfrequently the lungs show strongylosis as the fourth stomach shows strongylus contortus, the small intestines strongylus filicollis, tæniae (expansa, fimbriata, etc.), the large intestines œsophagostoma Columbiana, and tricocephalus affinis, and the gall ducts distoma hepaticum and distoma lanceolatum. In these chronic cases the spleen is usually shrunken, and the liver firm, sometimes even cirrhotic.

Mortality. The acute cases are usually fatal. Those that assume a chronic form, if free from local lesions in important organs,

well-fed, and, above all, kept in the open air, and changed to a different pasture, tend largely to recovery.

Prevention. The propagation of the infection from animal to animal is slow and somewhat uncertain, and when introduced by the purchase of a new ram or other animal, it may take a considerable time to affect the stock extensively, but for this reason, and because an apparently sound sheep may harbor the germ, it is difficult to oppose it successfully by segregation. All the same, it is desirable to take all possible precautions against its advent, and among these, the exclusion of strange sheep from non-infected pastures and flocks. When the time comes to make an outcross from the home strain, the ram must be selected not only for his pedigree and individual qualities, but no less for the soundness of the flock from which he is taken. If the lambs of that flock have been decimated by disease, the best blue blood, and most faultless form should not tempt the flockmaster. The ram should be rejected in favor of one taken from a flock that is above suspicion. It matters little if it can be plausibly argued that the mortality came from worms of the lungs, liver, or digestive organs; these in themselves may soon ruin any flock, but they, too, often coexist with the microbe of hæmorrhagic septicæmia, and, when this is the case, they prepare the system and open a way for its invasion.

New purchases should not only be selected from apparently sound and guaranteed stock, but they should be passed through an antiseptic dip on arrival, and then if possible quarantined in a special enclosure until they shall have proved their freedom from infection. A valuable ram may be placed with some lambs or yearlings in close quarters to ascertain whether he has brought the infecting matter with him. If all escape after some months the presumption is that he is sound. Perfect cleanliness of the fold should be maintained, and disinfectants may be freely used in it.

The water supplies should be watched, rejecting streams that have drained sheep-pastures where there have been marked losses of lambs and ewes. Water from deep wells without any surface leakage is to be preferred.

When new stock (ram, ewes, lambs) are of necessity mixed with the sound herd, a wide range, an open air life, and abundant

dietary must be secured. The system that is full of strength and vigor can better resist the microbe and even throw it off entirely, whereas the weak, confined subject succumbs. For the same reason, the weak, emaciated and debilitated subjects should be at once separated from the sound flock, and kept in a special enclosure, in the open air, on a rich diet. Should they harbor worms, this seclusion is even more imperative. (See parasites of lungs, liver, stomach and bowels).

Lignieres advocates immunization by serum prepared on the Pasteur method, but, as he has not divulged the exact technique of its preparation, it is impossible as yet, to give this an unqualified endorsement. It has this in its favor that the mature sheep, in full vigor of middle life, though in an infected area, usually resist the infection, while the young, old, debilitated and verminous suffer. Opposed to it are these considerations that are recognized by Lignieres himself;—1st. The acquired immunity is not perfect, as shown by occasional relapses in sheep that have survived a first attack; 2d. The serum inoculation is not only useless, but dangerous in animals that already harbor the germ; I may add 3d. Any acquired parasitism or debilitating disease may tend to break down the immunity and prostrate the system under the infection. Lignieres advises that the serum treatment should be restricted to the new born lambs in infected herds, or herds in infected areas. The first three or four weeks after birth are to be preferred for the operation, though failing this, it may still be ventured on, up to a few days before weaning. The longer it is delayed the greater the danger of a preëxisting infection, and of untoward results from the new access of infecting material, on the back of an infection which varies so extremely in its pathogenic potency. Even among the new born lambs, Lignieres would restrict the serum therapy to the strong, robust and healthy, and, if they survive the resulting fever, would repeat the treatment after the hyperthermia has ceased. No satisfactory *treatment* of the disease has been made. An open air life, a generous diet, and a course of iron, and bitters will, however, be of use in serving to improve health, digestion and vigor, to solicit a better production of red globules, and to enable the patient to survive the period of anæmia, prostration and debility. Antiseptics like quinia, the sulphites and the iodides might be used as adjuncts.

PNEUMO-ENTERITIS IN SHEEP: HÆMORRHAGIC SEPTICÆMIA: SWINE PLAGUE.

Historic note. Microbiology: ovoid bacterium, motile, with polar stain, non-liquefying, chains, grows freely on culture media Pathogenic to sheep, goats, dogs, hens, rodents, calf, ass. Views of Lignieres, Lienaux, and Conte. Symptoms: Acute form in young; hyperthermia, rapid pulse, troubled breathing, dulness, prostration, sopor, anorexia, congested petechiated mucosæ, offensive diarrhœa, emaciation, wheezing, cough, râles, crepitus, percussion flatness, abortion. Death in 6 hours to 3 days. Sub-acute form in mature: symptoms moderate, recoveries the rule. Lesions: fœtid carcass, blood staining of skin and organs, exudates, petechiæ, swollen congested lymph glands, peritoneal exudate, congested liver and spleen, gastro-enteritis, pleural effusion, lobular and peribronchial exudates, caseation, congested womb, placenta and brain, bacterium in lesions. Prevention: isolation, disinfection, secretions, manure, drainage, exclude tame and wild animals. Disinfectants.

Among the different forms of hæmorrhagic septicæmia in sheep, that observed by Galtier in 1889 in Basses Alpes, and later elsewhere in southern and western France and in Algiers, must be specially noted. It seems to be the same affection studied later by Lienaux, Conte, Besuoit and Cuillé and which prevailed from Tarn in the south of France, to Vendée in the west, and Somme in the north.

Microbiology. The pathogenic factor found in the lesions was an ovoid bacterium, a little larger than that of fowl cholera, motile, non-liquefying, with polar staining, and often showing in short chains of two or three joined end to end. It grows easily and abundantly in all common culture media, even on potato which fails to propagate the cocco-bacillus of Lignieres. This, with its ready transmission from swine to sheep and *vice versa*, apparently serves to differentiate it from the cocco-bacillus, and the disease from the hæmorrhagic septicæmia of Lignieres.

Pathogenesis, In Galtier's first observations in Basses Alpes four separate flocks were infected by pigs brought all from one market, and placed in or by the pens of the sheep where they sickened and one in seven died in a few days. Then the mortality began among the sheep and ranged as follows: 1st flock lost 10 in 37: 2d flock 16 in 25: 3d flock 8 in 20: 4th flock 12 in 22. On one farm 10 sheep were sent to a neighbor's just before the

arrival of the sick pigs and escaped, and on another a second flock kept in outlying pens well apart from the home flock kept perfectly sound.

With cultures of the microbe in vitro, he successfully inoculated sheep, goats, dogs, chickens, Guinea pigs, rabbits, and, finally, a calf and an ass. The cultures were inoculated in different cases; intravenously, into the trachea, pleura, lung, and subcutaneous connective tissue, and one goat was infected by ingestion.

Lignieres claims that Galtier must have worked with a complex infection in which his (Lignieres') cocco-bacillus was an essential constituent. The evidence of this is, however, lacking, and we must recognize that Galtier made cultures which showed the close relationship of his organisms to swine plague, and their lack of complete identity with those of the Lombriz. The filtrable microbe of hog cholera was probably present.

Lienaux and Conte had pathogenic results in a limited number of animals only (rabbit, mouse, guinea pig) illustrating the familiar truth of the variability in the pathogenesis of different specimens of septicæmic bacteria of the colon group.

Symptoms. 1. Acute Form. There is a sudden marked rise of temperature, often to 107° F., with acceleration of pulse and respiration. Sometimes death follows so early so as to prevent the observation of other symptoms. If otherwise, there supervene marked dulness, prostration, somnolence, anorexia, suspension of rumination, and more or less tympany of the rumen. The patient is found lying down, apart from the flock, indisposed to rise, with deep red, congested, arborescent conjunctiva and other mucous membranes, and petechial spots on these and on the white portions of the skin. The fæces, at first moderately firm and moulded in pellets, marked here and there with lines of mucus, or even blood, become on the second or third day soft, pultaceous, or watery and highly offensive. Parallel with this, emaciation advances with rapid strides. Breathing becomes more hurried, wheezing or snuffling, with a muco-serous, often bloody discharge from the nose, and an infrequent cough. Careful auscultation and percussion will often reveal the blowing or mucous râles, the crepitation or flatness on percussion of broncho-pneumonia. As the disease advances the petechiæ on skin and mucosæ extend, and extensive deep violet areas show especially on the inner sides

of the arms and thighs, on the under surface of the tail, on the perineum and under the belly. Abortion is a common result the lambs coming dead.

Death may occur in six hours, more commonly in two or three days, or even later, the temperature going down to below normal in the last stages. In case of recovery, convalescence is slow, the animal remaining emaciated and weak, with lung lesions and persistent cough for a length of time. These poor imperfectly recovered animals continue to harbor the microbe and transmit it. Lesions of the liver and other organs, the result of this disease, are found in sheep that have survived the affection and are afterward killed for mutton.

Symptoms: 2. Subacute and Chronic Form. This is seen in mature sheep of two or three years, that have been infected when in the full vigor of life and health, during an open air life and by a limited dose of the virus. The temperature is less elevated, the circulatory and respiratory functions less disturbed, the dulness and prostration only moderate, the interruption of feeding and rumination transient and tympany slight. Cough and nasal discharge are however present and rather persistent, diarrhœa may be manifested, and marked emaciation occurs. There may be congestion of the mucosæ, but petechiæ and extensive bloody discoloration of the mucous membranes or skin are rarely seen. Abortion is not uncommon.

Recovery is the rule in this type of the disease, though in some cases it will be delayed for weeks and may even then be imperfect.

Lesions. In the acute types of the disease decomposition advances rapidly after death, and the carcass from the first exhales a peculiarly heavy odor. The skin is discolored, the nasal mucosa is the seat of a muco-purulent or sanguinolent discharge, the anus is soiled with liquid fæces, the subcutaneous connective tissue marked by red arborescence and blood-stained areas, or by a gelatinoid exudation, the muscles at times deeply colored and petechiated, the lymph glands, especially the bronchial and mesenteric, are enlarged and congested of a deep red—the color being highest in the cortical zone. In some old standing cases these may have softened and acquired a grayish hue. They are rich in the specific bacterium.

The peritoneum is congested, petechiated, thickened, and more or less covered with thin false membranes, and it contains from a pint to three quarts of a sero-sanguinolent liquid which coagulates loosely on exposure. The liver is congested, with points of blood extravasation, and zones of degeneration (fibroid, fatty or necrotic), and sometimes abscess. The congestion extends to the spleen and pancreas. The abomasum and intestines are the seat of mucous gastro-enteritis. The mucous folds of the pyloric sac of the stomach are congested, reddened and petechiated, the small intestine has tracts and patches of congestion, thickening and softening, Peyers patches and the solitary glands are enlarged, and ulcers may be present on large or small intestines.

Effusion is usually found in the pleura, clear, grayish or bloody, with flocculi and false membranes, and further branching redness of the serosa. The mediastinum may be thickened by exudate especially around the glands, œsophagus and blood vessels. The lung shows lobular, interlobular and peribronchial exudates approaching at times to the appearances shown in lung plague, along with the atelectasis, emphysema and, in prolonged cases, caseation.

Less constant are congestions and petechiæ of the pericardium, heart, kidneys, vesical mucosa, testicles, ovaries or womb. The blood is dark and forms a loose coagulum. In case of abortion the umbilical cord is infiltrated and the placental membranes ecchymotic. The brain and spinal cord and their membranes are sometimes congested and infiltrated and a serous effusion exists in the arachnoid.

The bacterium is found more or less abundantly in each of the morbid lesions.

Prevention. Absolute seclusion of the sick and of all their products is the prime essential. The general distribution of the lesions throughout the body and the uniform presence of the bacterium in the lesions indicate that no part of the body and no secretion can be considered as free from infection. All parts of the body, all expectoration, saliva, fæces, urine, milk even must be withheld from all other animals, at least until they have been thoroughly cooked or disinfected. Manure must be burned, or buried deeply in quicklime. Contaminated pens, yards, wallows, streams and fields must be abandoned or thoroughly disinfected.

Galtier found that the virus remained active for six days in putrefying organic matter at 39° to 75° F.; for 25 days in water, at room temperature; the refuse litter, fodder, manure, and drainage matter from the infected place must be carefully guarded against. The virus steadily loses in force in such media, but remains infecting to animals injected with it. The most active disinfectants may be used on the pens and yards (copperas 5%, sulphuric acid 2% solution, or mercuric chloride .2%) while the contaminated fields should be abandoned for the season. All droppings may be treated with sulphuric acid (2%), creolin, lysol, phenic acid or copper sulphate. The susceptibility of practically all the animals of the farm, demands the exclusion of these, while that of rodents, renders necessary the further exclusion of wild mammals; and we may add birds, wild and tame, and if possible flies. The plowing of the contaminated soil will do much to obviate danger, yet the sheep folds and pastures should be separated by a considerable distance from any place where infected animals and objects have been. If drinking troughs for sound animals have been used by the sick or suspected they should be emptied and washed with disinfectant (sulphuric acid 2:100).

SEPTIC (BACTERIDIAN) DISEASES OF THE LUNGS ETC., IN ANGORA GOATS.

Contagious lung diseases in Angora goats have prevailed in Saanen, Simmenthal, in the Bernese Alps (Pusch), in Schmalkalden, Thuringer Wald (Schütz) in Stembach—Hallenburg, at Ismid on the Coast of the Sea of Marmora and inland in Angora (Nicole and Refik Bey), in Khandesh in the Bombay Presidency (Steel), and in Cape Colony (Hutcheon). In 1903, Mohler and Washburn described under the names of *Takosis* (*teko*, to waste) an infectious destructive disease of Angoras in Penn., and Md., and quoted reports of what appeared to be similar troubles in Oregon, Michigan, Illinois, Missouri, Massachussetts, Georgia, and Texas.

Causes. The disease was especially fatal in Penn., and Md., in autumn and winter, having started with goats imported from Texas. It was first attributed to laurel poisoning, but proved

no less destructive in fenced, cultivated fields and pens where laurel was inaccessible. A dry, nutritious diet of corn, oats, and hay made no improvement. Separation in small lots in separate pens proved more helpful. In the Old World its introduction in infected goats has been constantly noted and its prevalence in the native home of the goat (Angora) bespeaks its primary source. The first cases of Pusch and Schütz were brought from Saanen, its appearance in Cape Colony was traced to goats direct from Angora, and the outbreaks in Penn., were in goats just arrived from Texas. Out of their native habitat Angoras are found in comparatively limited numbers, but their introduction is too often followed by an appearance of this infection, which like any other pestilence spreads in an infected locality in ratio with the numbers of susceptible animals and their aggregation in a small area.

In the Old World and the New the microbian cause is held to be a globular or ovoid coccus (*Micrococcus Caprinus Sp. nov.*—— Mohler) found in the heart's blood, pericardial fluid, spleen, kidneys, etc. Mohler failed to find it in the cerebro-spinal fluid, and Nocard and Leclainche elsewhere than in the lung and expectoration. The microbe is 0.8μ to 1μ in diameter and appears in cultures singly or more commonly in chains of 2, 3 or 4. After three days culture at 37° C. they are globular and may form chains of six, or even clumps. The individual cocci vary in size and shape. They stain slowly in methylene blue, but quickly in carbol fuchsin. The coccus is ærobic (facultative anærobic), grows freely in slightly alkaline media (liquid or solid), and much less actively in acid, 1.5 phenolphthalein. Growth is most abundant at 37° C, and tardy at room temperature. In neutral beef bouillon at 37° it causes a slight opacity in ten hours, and in three days it clears by throwing down a pearl gray, ropy precipitate. The culture becomes acid in five days. The coccus grows freely on blood serum, milk or agar: more slowly on gelatine and potato. It survived freezing for four days, but was killed in six minutes at 62° C.

Pathogenesis. The cultures, subcutem, infected the goat (Angora and common), calf, rabbit, Guineapig and white mouse. Rats, swine, dogs and sheep proved immune. Steel claimed the infection of two sheep, but, as the example stands alone, it may have been a coincidence merely.

Identity. The identity of the disease in different epizootics has been questioned. Thus Nocard and Leclainche doubt the identity of the pleuro-pneumonia that the latter investigated in France, as also that observed by Pusch, Schütz and Storch in Germany, together with the disease of Algerian goats known as *bou frida*. In these cases and in those seen in Cape Colony and India the pulmonary hepatization appears to have been more marked than in the American cases, Hutcheon, indeed, believed that he was dealing with the lung plague of cattle. But the variety and grade of lesion may be largely climatic. Even the lung plague of the ox differs greatly in our drier American climate in winter, from the same disease in Western Europe. Then the goat disease is so constantly traceable and even confined to the Angora, shows so uniformly the same microbiology, presents such similarity in its lesions, symptoms, duration and mortality, and agrees so remarkably in the genera susceptible, that the burden of proof should be upon the person who denies the identity of the malady in any two outbreaks.

A variation even in virulence may depend on the environment of a particular herd or the state of the system invaded. Thus the addition of lactic acid to the culture injected, killed in a few hours, so that habitual exertion with the accumulation of sarcolactic acid may determine a specially violent type. So with other cases of auto or fodder poisoning and of complex infection.

Symptoms. In the tardiness of its course, the diarrhœa, the cough, the great emaciation and weakness, this disease strongly suggests *entequé*, or a destructive parasitism. But worms are usually altogether absent and mature cattle do not suffer from exposure to the contagion. In the early stages there is no visible disorder. Then the goat becomes listless, languid, droops its head, ears and eyelids, and lags behind the flock. He mopes around with arched back and head bent toward the sternum, but still eats and ruminates fairly well. The temperature is highest at the start (104° to 107° F.) and may later become subnormal (99.7° F.). Snuffling and nasal discharge are common. Cough is easily roused by percussion of the chest. Blowing and crepitant râles may be detected on auscultation. Sometimes there is distinct flatness of sound over given areas when percussed. In the advanced stages there may be marked dyspnœa, the goat

standing or lying with head extended, mouth open, tongue protruding and mucosæ violet. Sometimes early in the disease, but almost constantly in advanced stages, there is an encreasingly fluid and offensive discharge from the bowels. The encreasing pallor of the skin may escape the observer unless the hair is drawn aside to show the surface. The same is true of the emaciation, but the gradually encreasing pallor and sinking of the eyes must be noted. The growing debility shows first in tardy, listless movement, then stilty gait, swaying or staggering, then the patient is trodden down under the feet of its fellows, later it lies almost constantly with its head on one side or bent on the sternum, and finally there is complete paralysis. The stricken animal bleats or groans at intervals. Signs of enteritis may be entirely absent. Abortion is a frequent complication. Four-fifths and upward of the diseased goats perish. The duration of the disease may be from one to eight weeks.

Lesions. On opening the carcass one is struck by the extreme emaciation and anæmia. The skin and subcutaneous structures are pale and bloodless. The muscles are pale, anæmic, and wasted to the last degree. The rumen may contain a considerable amount of food recently taken. There may be slight areas of congestion on the bowels especially the small ones. The intestines contain a moderate amount of normal contents, or foul liquid, and the mucosa may show enlarged follicles, a covering of slimy mucus, and even necrotic and sloughing centres. The liver is usually normal, the spleen pale and shrunken, and the kidneys soft, pasty, and anæmic. The lungs float in water, but are mottled, pale pink patches alternating with others of a dark red or iron gray, yet extensive consolidation is rare. The lower lobes are usually most affected and in these Hutcheon, Nicole and Refik Bey and others have noted hepatization with distinct marbling as in lung plague. In the congested patches the bronchioles, on section, exude a frothy mucus, and their walls show considerable fibrinous thickening. The pericardium contains more or less of a pink fluid, and the epicardium shows small congested areas and blood extravasation, mostly punctiform. Similar petechial spots may be found on the pleuræ and peritoneum. The blood count shows an increase of red globules (11.000.000 to 12.000.000) and leucocytes (18.000 to 20.000.) This is perhaps accounted for by the great loss of liquid through diarrhœa. It disproves any material destruction of the globules.

Prevention. Angora goats should not be transported long distances, and especially to a higher altitude or more northern latitude except during late spring and in summer when the increasing warmth will compensate for the change. Comfortable sheds or well drained soil should be accessible at all times to protect against severe rain or snow storms and wet beds. The absence of yolk in the fleece allows the water to penetrate much more easily than in the sheep. A generous diet should be allowed, to maintain the bodily vigor and enhance the resistance to bacterial invasion. Attempts at immunization by the subcutaneous injection of the sterilized blood or cultures, gave contradictory results.

The most important precaution is to prevent any sale or movement of goats from any infected flock until the disease has been completely extirpated. No infected flock, nor goat, should be allowed on a watershed which drains into pastures or drinking places of goats lower down. The young should be placed on new sound pastures, and if the dams of any are infected, the latter should be furnished with healthy nurses, (common goats or sheep) or brought up by hand. Imports from Asia Minor or any other infected country should be received only on official certificate that they came from an uninfected district, and on arrival should be quarantined and disinfected. Such measures may appear harsh, but this is a very deadly infection, and the susceptible animals in the United States are as yet in small numbers, so that extinction and exclusion of this disease would far more than compensate for the outlay. In the single epizootic in Cape Colony, introduced in 1880 by an importation from Angora, the goats were sold to different farmers, more than 12,000 goats succumbed, and 6,640 more were sacrificed to stamp out the plague.

Treatment. As in other infections, propagated through the animals, and not resident in the soil, treatment should never replace prevention, and complete extinction. Yet in properly isolated places the sick may be treated. Mohler and Washburn got the best results from 1½ grain doses of calomel twice daily for two days, followed on the third day by: Arsenious acid 1 grain: Iron reduced (or sulphate) 12 grains: sulphate of quinine 2 grains: Repeat this night and morning for ten days: then, after two days interval, repeat the course.

ULCERATIVE (ERYSIPELATOID) INFECTION OF THE LIMBS IN CATTLE AND SHEEP.

New York outbreaks. Causes: wounds by sharp pebbles, streptococci, pure cultures, inoculations. Symptoms: swelling on lower limbs, abscesses implicating tendons, bones, joints, and under hoof. Prevention: avoid septic or frosted mud, irritants, etc., disinfect surface, keep dry and clean, open abscesses, use disinfectants, separate infected.

This affection was seen in several herds in Oneida Co., N. Y., in the Spring of 1897. The geological formation of the region was a calcareous rock with a surface soil thickly impregnated with pebbles and small flat shaly masses. Sheep suffered in Western New York.

Causes. In Oneida, the subjects were dairy cows, which in the early spring, when the frost went out of the ground, had to wade through chilly soft mud reaching to the knees or above, and mixed with small stones with sharp edges, and with semi-liquid manurial products. The abrasions made by these stones furnished convenient infection atria for septic microbes in the manure. In the pus obtained from the ulcers were found in abundance the bacillus coli commune, and a long streptococcus representing on an average from 20 to 40 cocci in a chain. Dr. Moore made pure cultures of these and produced the same symptoms in a cow by injecting the streptococcus subcutaneously in the back of the pastern, and in another animal by simply abrading the surface and rubbing on the streptococcus culture. As the inflammation, suppuration, and resulting ulcers implicated not only the skin but also the subcutaneous connective tissue, the affection had many of the characters of erysipelas.

Symptoms. The affection commenced by swelling in the region of the fetlock, pastern, metacarpus or metatarsus, and exceptionally, the forearm or tibial region, and advanced to a tense swelling, which pitted on pressure, and the formation of centres of suppuration, which burst and discharged. If the exposure to the septic mud were continued, the sores ulcerated and extended both in the skin and subcutaneous connective tissue, but when the dry weather came and the mud disappeared, the tendency was to spontaneous recovery. In some instances ulceration extended

beneath the hoof threatening its evulsion, and in other cases it extended deeply to the tendons and ligaments.

Treatment. The main object should be prevention, to be secured by protecting the stock against contact with septic mud, and especially such as is near the freezing point and intermixed with such sharp stones and pebbles as would wound the surface and open channels for infection. When infection has taken place we should seek to limit it by lotions of an antiseptic character. Bandages soaked in a solution of hyposulphite of sodium, 1 dr. to the ounce will often succeed. A more potent application is iodized phenol, prepared as follows: tincture of iodine 2 drs., carbolic acid crystals 4 drs., glycerine 1 oz., alcohol 1 oz., water 8 ozs. When applied on a bandage this may be diluted with water to make 1 pint. For circumscribed application to forming sores, the undiluted iodized phenol, made of one part each of iodine and carbolic acid crystals, may be applied twice a day with a glass rod. Other favorite applications are a lotion of lead and laudanum; a saturated solution of boric acid; ninety-five per cent. alcohol; a mixture of creolin 1 part, iodoform 4 parts, and lanolin 10 parts; ichthyol and collodion; ichthyol and vaselin; or iodol, iodoform, salicylic acid or resorcin as dusting powders.

Internally, tincture of muriate of iron 3 drms. every three hours helps to keep the affection in check.

CONTAGIOUS FOOT ROT IN SHEEP AND CATTLE.

Definition. A chronic infectious ulcerative disease of the interdigital structures and subcorneal tissues of the foot, determined by the presence of the *Bacillus necrophorus* (Pflügge, Bang, Loeffler).

History. Footrot is described by Chabert as prevailing, in 1791, on the low damp lands north of the Pyrenees and later by Pictet, Gohier and others along the rich alluvial river-bottoms in France and other parts of Europe. It was first noted in Merinos, and extending in the line of their diffusion, was thought to be peculiar to this breed, but wider experience showed that the long wools possessed no complete immunity, though there seemed to be a somewhat lessened receptivity for the infection. The

finer wool, epidermis and horn of the Merino, explain a somewhat lessened resistance to the entrance of the microbe, but there is perhaps a still stronger invitation in the much more elaborate development of the sebaceous glands, in the fine wooled sheep, which affords a free passage for the bacillus not through the skin alone, but through the soft horny pads of the heels as well. Even in America the Merino secured a bad preëminence through the spread of foot-rot in the new flocks, developed in Ohio, Michigan, Indiana, Illinois and especially Iowa. The flocks developed on the prairies were largely Merinos, because being far from markets, and without railroad communication, the fleece which would bring the highest price per lb. was naturally sought after. Moreover it was found that the fine wools could be better cared for in large flocks, than the long and middle wools. And thus in every way the former seemed preferable, the cost being less and the returns greater. But wherever the flocks encreased, scab and foot-rot encreased still more, and as the flocks followed each other on the unfenced prairie and everything favored infection, the golden dreams of the flock masters gave place to visions of ruin. It soon came to be looked on as a public duty to stop and kill any lame or scabby sheep that might be met with.

Causes. The contagion was experimentally demonstrated as early as 1823 by Favre, who took the matter from diseased feet and moistened the skin between the hoofs of healthy sheep causing the disease in 21 out of 32 sheep treated. The uniform experience of the spread of the disease slowly in a flock in which a sheep affected with foot-rot had been placed, had long before established the fact. The complete demonstration came with the isolation, by Lœffler, Bang and others of the microbe from the many which make up the complex infection on the ulcerating sores, and the successful inoculation of the pure cultures. Mohler and Washburn washed thoroughly the foot of a healthy sheep, then sterilized it by a 5% solution of carbolic acid, then washed this off with sterilized water, inoculated it from the foot of a diseased sheep, and bound it up in rolls of sterile cotton covered with a close linen wrapper. The liquid drawn from the starting inflammation in the inoculated foot is injected subcutem into a rabbit, which it kills in four to seven days. The inoculation of

a second rabbit from the first, and of a third from the second is usually necessary to secure a pure culture. In the seat of the inoculation, in the subcutaneous tissues, there is found an area of 1½ to 2 inches of soft, putty-like, necrotic tissue with a strong odor of putrid flesh. Around this is a zone of inflammation about ½ an inch thick, involving all the adjacent tissues, muscles, connective tissue, etc., and beneath this a boggy swelling caused by the inflammatory exudate which has gravitated downward.

Scrapings from the necrotic mass show that it is penetrated in all directions by long thread-like bacilli, *Bacillus Necrophorus*, 0.75μ to 1μ in thickness and of varied lengths up to 100μ. The organism may even be found in the circulating blood and in the lungs, liver and kidneys, where it causes small yellowish white necrotic spots. It grows well in blood serum 1 part, 10 per cent gelatine 1 part, 1.75 per cent agar 2 parts: or again in ordinary bouillon with enough agar to make it a soft jelly: or again in rabbit or beef bouillon. The best growths take place beneath the surface, from stab cultures, and in any case the culture tube should be immersed in a flask of hydrogen, to satisfy the anærobic propensity of the microbe. The microbe is gas producing in artificial cultures and in the body of the rabbit kept on ice. The colonies in 48 to 72 hours show as small yellowish white specks, which later show a fuzzy, cotton-like envelope. The organism stains well in aniline dyes, but not by Gram's method.

Pathogenesis. *Bacillus necrophorus* is pathogenic to sheep, goats including Angora, cattle, rabbits, and mice, and shows not only in the extensive necrosis of the inoculated tissues but also, in rodents especially, as necrotic spots in lungs, liver and other internal organs. The contagious foot-rot in cattle has been shown to be due to *Bacillus necrophorus* (Pflugge, Bang, Von Imminger, Hess, Francke, Cadiot and Almy, Mohler and Washburn, Ernst, etc). Horne describes a contagious foot-rot in reindeer from the sores of which he recovered the Bacillus necrophorus, Francke, Ernst and others have found the same bacillus in the necrotic tissues of horses, deer, pigs and kangaroo.

Accessory Causes. Though not essential these are often operative in making an easier entrance for the necrotic microbe. Traumatic injuries are especially to be recognized. The accumulation of clay or mud in the interdigital space and the

abrasion or laceration of the surface by the dried hardened mass, the wounds inflicted by the sharp edges of broken stones (slaty, shaly) mixed in the mud, coarse sand even in the mud, broken glass sharp metal, or nails or the strong stubble of grain or weeds. Caustics act in a similar way. Inflammation of the heel pads by traveling on hard roads, or standing on wooden, stone or cement floors. Overgrowth of the hoof—above all in sheep, throwing undue strain on the laminæ, imprisoning the hard plates of the sole to act as foreign bodies, and causing incurving of the hoof-wall till it presses painfully on the sole. Section or detachment of the horn by violence or bruises under heavy weights (wheels). Constitutional disorders which concentrate or localize in the feet. Of the latter an overfeed of grain, ground feed, maize, malt, brewers and distillers grains, linseed cake, colza-cake, sesam-cake, peanut-cake, wheat, rye, rice, and cottonseed meal have been specially incriminated. Again inflammation of the feet will follow internal disorders and infections, especially difficult parturition, abortion, retained afterbirth and diarrhœa. Pools, and mudholes are ready channels of infection. Among pastures those with long grasses, which drag through the interdigital spaces of the infected and sound in succession, favor transmission. Dense aggregations of sheep or cattle, as in fairs, shows, stock-yards, steamboats, ferryboats, chutes, runways, loading banks, cars, sheep wagons, sheephouses, folds, etc., tend to distribute and concentrate the affection. Outbreaks on Atlantic steamers show that the germ may be carried on the sound feet, while its pathogenesis is favored by the softening of the hoofs among the heating manure, the strain thrown upon the feet by the movements of the vessel, the warmth developed by lying in a huddle with the feet under them, and perhaps the dry, heating food to which they are subjected. It has been noted that the ventral aspect of the body, resting on infected feet during recumbency, has become infected, and the resulting necrotic patch has been promptly attacked by the gad fly so that the sheep perished from the larvæ. The blocking or inflammation of the interdigital bi-flex canal in the sheep has long been charged with causing foot-rot, and the very presence of the delicate lining of this duct is an invitation to the microbe, and like other parts when inflamed, it is specially favorable to the microbian activity. The entrance of

mud, sand, or other irritant tends to inflammation. The occurrence of a local phlegmon, or boil in the region of the foot, an attack of aphthous fever, or the inflammation or gangrene caused by ergot or smut may prove the starting point for the colonization by the Bacillus necrophorus. Indeed any breach of surface or enfeeblement of the tissues may become the efficient accessory etiological factor.

Symptoms and Morbid Anatomy. Since the disease is a slow and chronic one an infected flock usually presents, side by side, cases which show the earliest and the most advanced stages, together with many intermediate grades. At first there is a slight halting or lameness, and examination of the inflamed point, usually the arch of the interdigital space, shows a circumscribed redness and swelling and a moist condition of the affected surface. Commonly the case is further advanced, the greater part of the interdigital space has become involved, the epidermis has fallen off or is being shed in white or yellowish white layers, and a raw ulcerous surface is exposed. As the ulceration advances, being hemmed in by the unyielding horn, it extends beneath it in different directions loosening and detaching it from the vascular parts beneath, and also extending inward into the subcorneous tissues as far it may be as into the tendons, bones and joint structures. The inflammatory exudation may lead to excessive swelling, but mingled with this is the necrotic process which hollows out deep excavations in the tumefied mass. The raw surface exudes abundance of a whitish, opaque fluid of a strong offensive odor, which will penetrate to all parts of the building or pen. This fluid contains abundance of pus cells, fragments of the ulcerating and necrotic tissue, and bacteria of many kinds but including the specific microbe.

The morbid process may begin at the posterior part of the interdigital space, at the front, or in the median part, in the pad, in the sole, or on the coronet, and it has been named accordingly. As it becomes chronic the hoof may be shed, and in bad cases even the pedal bone, or short of this the joints may be anchylosed, but not infrequently the plastic process acquires the predominance, and large masses of new inflammatory tissue form at the coronet in front, at the heel behind, in the interdigital space pushing the hoofs wide apart, or elsewhere where the morbid process has been

concentrated. These new growths are centres of microbian growth and permeated by fistulæ which discharge the infecting morbid product.

The sheep, no longer simply lame, now moves on three legs, or if the fore feet are affected walks on its knees seeking its food, for appetite is preserved, or it lies down most of the time.

The feet are not worn and, having an extra blood supply at the coronet they grow with increased rapidity, curling into a spiral, so that the wear would come on the outer wall which presses inward and upward on the sole.

A case may progress for months, the infection being in the main local, and the digestive and other functions being maintained for a length of time. The animal however becomes gradually emaciated, and with the increasing debility there is a greater liability to a general infection either by this or by some other pathogenic microbe. The existing sores during the summer and autumn strongly attract flies, and life may be quickly cut short by the attacks of the larvæ.

Apart from such conditions recoveries may take place spontaneously, but usually with permanent distortions of the hoofs, fibroid growths between the claws, thickening and stiffening of the tendons, or anchylosis of the digital joints. The result depends mainly on the care of the affected sheep. If allowed to wade through mud, or liquid manure, or to stand on hot fermenting manure the infecting and ammoniacal and other products determine steady aggravation, whereas if the feet are kept clean and dry, and properly dressed and medicated from the first a prompt and satisfactory recovery may be counted on.

Prevention. As in all injurious infections, eradication and exclusion are the important considerations. To secure exclusion newly acquired sheep should be certified by an official guarantee that they come from a locality free from foot-rot, and where this affection has not existed for years. The purchaser must himself see to other precautions. See that they have not been travelled over ground or roads that have been used by infected or unknown sheep, that they have not passed through public stockyards, nor premises, nor over nor through undisinfected loading banks or chutes, nor have been conveyed in any undisinfected car, ship, boat, or vehicle that has been used for conveying other

sheep or cattle. Thorough cleaning followed by sprinkling with dilute sulphuric acid (10 : 100), carbolic acid (5 : 100) mercuric chloride solution (1 : 500), chloride of lime (4 ozs. to gall.) or even freshly burned quicklime will serve a good purpose. To the mercuric chloride or lime-chloride add enough quicklime to whiten the surface and show if any part has been missed. When the new sheep arrive at home keep them for a fortnight in pastures and premises separate from the rest of the flock, and where there can be no drainage to a place occupied by other sheep, examining them closely and frequently for any indication of lameness or diseased feet. Then subject them to an antiseptic full bath or foot bath, according to the season, before adding them to the home flock.

For home precautions avoidance of all the conditions named as accessory causes is important. Mud puddles, pools, marshes, and soft, wet, stoneless pasturages are to be avoided and, when available, the tracks may be laid with gravel, broken brick, or other material which will wear down the hoofs. In the absence of such wear the toes must be pared at frequent intervals, a stout knife or toe clippers being used. A dewy morning or wet day greatly facilitates this work. Three legs may be tied together, the fourth being left to be operated on. The operator kneels with his leg across the neck of the sheep, to keep head and body still. This must be especially attended to in winter when the sheep are confined. Standing on manure heaps in pens and yards is most injurious, and thus a clean, smooth, stone, brick or cement floor should be furnished, when possible. Keep the range clear of foreign bodies, splinters of stone, nails, glass, broken crockery, and metallic objects. Next comes the question of overdriving as a cause of pedal inflammation, and overstimulating food as a constitutional cause.

Treatment. The first consideration is the separation of the sound from the infected animals, pasturages and premises. It may even be desirable to divide the diseased into two lots, one including those slightly affected and which will get well on one or two dressings, and the other containing the bad, advanced cases, which will demand more continuous treatment and from which there is a greater risk of infection spreading.

If the feet are overgrown or distorted these must first be pared.

The toe is shortened as far as possible, without cutting it too thin or tender, and the foot levelled alike from side to side and from behind forward. Any horn that has been underrun, and separated from the quick, must be carefully pared off and the edges of the sound horny wall bevelled to a thin elastic margin, which will not press in and irritate the tender parts. All moderate cases will now be ready for the antiseptic caustic dressing.

For slight and recent cases with no underrun horn, a foot-bath may be used. A watertight wooden trough is made four inches deep, filled with a solution of copper sulphate (3:100), or carbolic acid (5:100), or freshly prepared chloride of lime (4 ozs. to the gallon), and the sheep are driven into this and left to stand there for two minutes. On leaving the bath they should be run into an uncontaminated field or disinfected yard or pen. The bath may be repeated the second day or oftener if it seems requisite. If a bath cannot be secured, individual dressing with a hair brush or cotton swab dipped in the disinfectant liquid may be substituted. One man throws and holds the sheep on its rump, and separates the hoofs one after another, while the second cleans the interdigital space with a cloth or with water and soaks it and the coronet with the disinfectant. In cattle with the hind feet attacked, it is often convenient to have the leg lifted and carried backward by two men holding a smooth, round piece of wood, like a stout fork-handle, in front of the hock while a third person dresses the foot. The same agents may be employed. Often a dilute sulphuric acid (1:4), or antimony chloride, will operate well.

In the more advanced cases with extensive sloughing and the formation of fistulæ, the treatment must be more thorough and persistent. After removal of all horn that can in any way prove irritant, a thorough soaking with cupric sulphate (3:100); or with cupric sulphate (3:100), lead acetate (3:100) and carbolic acid (3:100), may be followed by packing of wood tar between the hoofs and on all raw surfaces, maintained in place by a bandage tied upward and around the pastern. In particularly bad cases an initial treatment with peroxide of hydrogen is indicated, as this agent is specially destructive to the anærobic microbe. When healthy granulations have started it may be replaced by one of the other antiseptic dressings. Or again, aniline

blue (pyoktanin) (1:1000) may be employed to good purpose. A large number of other antiseptics may be substituted, care being taken that they are not dangerous either as poisons or as deeply penetrating caustics. Creolin, lysol, creosote, cresyl, tincture of iodine, solution of silver nitrate, or zinc chloride, may be used ; or, when a more soothing agent is demanded, tar, crude turpentine, iodoform or tannoform may be substituted. In all cases it is essential to protect the feet against dust and above all mud. Keep on a wooden or other dry clean surface.

When a hyperplasia (fungus growth) has formed at any point it must be removed by knife or curette or by the thermo cautery, any bleeding being checked by the latter, by iron chloride, or adrenalin, or by a compression bandage, and the antiseptic treatment must be maintained as above directed.

Open sheaths or joints may demand a careful use of antiseptic lotions (cupric sulphate, etc.) and diseased bones may render amputation of a digit imperative.

GANGRENOUS INFECTION OF THE CORONET—HORSE. POTCHETCHOUI.

Seen on rich, damp lands. Symptoms: lameness, small areas of congestion, erection of hairs, vesiculation, pustulation, gangrene as deep as tendons, depilation, desquamation, fistulæ, slough of hoof, early fever, impaired appetite, recovery in 14 to 21 days in most cases. Staphylococcus and streptococcus. Prevention: segregation, different attendants, avoidance of infecting land while damp, smear limbs with antiseptics. Treatment: antiseptic bandages.

Under this name Sotsevich describes an infectious disease of the horse, which prevails in the Don province, and especially on rich, low, damp lands. Before the appearance of any local lesion, the horse goes very lame on one limb. Afterward there appears, usually on the coronet or pastern, an area on which the hairs stand erect, with elevation of the epidermis and the formation of vesicles as large as barley corns, filled with a yellowish white liquid and later with pus. Gangrene follows, extending from the skin to the subcutaneous connective tissue, aponeurosis and tendons, and forming large sores, one to two inches in diameter and discharging an abundant yellow, fœtid pus. The adjacent skin

takes on a yellow tint and sheds its hair and epidermis. The disease may extend into the interphalangean joints or under the hoof, leading to offensive fistulæ or evulsion of the hoof. If it extends to the higher parts of the limb it becomes less destructive.

At the onset there is some fever (101° F.), dullness and inappetence, but these gradually subside, and in favorable cases complete recovery has taken place in two or three weeks.

Bacteriological investigation has detected in the contents of the vesicles, staphylococcus pyogenes aureus, and streptococcus pyogenes.

Prevention is largely secured by a rigid separation of the diseased from the healthy. Other obvious precautions would be to exclude those dressing the sores from handling other horses until after thorough disinfection of the hands, and to keep the sound animals, during wet seasons, from the damp infecting soils, or when they must work on these, to smear the lower parts of the limbs with antiseptic ointments (tar, carbolic, iodine, mercuric, etc., etc.)

Treatment with sublimate bandages proves very satisfactory, yet any comparatively non-irritating antiseptic lotion may be used.

STRANGLES, INFECTIOUS RHINO-ADENITIS.

Synonyms. Definition. Historic notes. Bacteriology: streptococcus coryzæ contagiosæ equi in pus, chains of 3 and upward, free cocci, arthrospores, clumps, ærobic, growing freely in serum, or glycerined bouillon; pathogenic to horse and white mouse; relation to other streptococci; clinical evidence; accessory causes, youth, primary susceptibility, dentition, training, impure stable air, grain ration, excitement, sudation, fatigue, chill, change of climate, trading, crowding, sea voyage, catarrh of air passages. Infecting products, pus, ingesta, blood, manure, fodder, litter, water, secretion of mucosa of healthy. Wounds, castration, mangers, racks, troughs, buckets, poles, shafts, harness, halters, twitches, blankets, rubbers, combs, brushes, men, etc. Pathology: infection of lymphatics, through inhalation, sore, ingestion, congenital, milk; congestion of nasal mucosa, epithelial degeneration and desquamation, discharge little viscid, corded lymphatics rare, submaxillary swelling rarely small or nodular, pus creamy, indolent cases, pharyngeal, thoracic, buccal, gastro intestinal, hepatic, pancreatic, splenic, muscular, arthritic, cutaneous, nervous. Forms: mild, malignant, regular, irregular. Incubation 3 to 5 days. General symptoms: hyperthermia, dulness, apathy, costiveness. Specific symptoms: nasal, congestion, sneezing, purulent discharge; epiphora, submaxillary phlegmon; pharyngeal

and laryngeal; parotidian; pulmonary; abdominal, hepatic, pancreatic, splenic, perirenal, cutaneous, genital, nervous, septicæmic. Diagnosis, from catarrh, glanders. Prognosis, favorable, apart from malignancy. Prevention: exclude strange equine animals; avoid public stables, yards, drinking troughs and buckets; also manure, stable utensils, hay, fodder, litter, or watershed from infected places; disinfect cars, wagons, etc. Seclude inmates of infected stable, yard, park, etc., temporarily close public drinking and feeding places, make sale or exposure of infected animal penal; temporarily close dealers' stables; sale with general guarantee only. Disinfection. Immunization; inoculation from mild case. Treatment: hygienic, antipyretic, eliminating, antiseptic, surgical, tonic, antisuppurant.

Synonyms. Distemper; Coryza Contagiosa Equorum; Gourme (Fr.); Druse (Ger.); Cimorro (Ital.).

Definition. An infective, streptococcic, febrile disease of solipeds, usually manifested by a catarrhal inflammation of the upper air passages, and phlegmon of the adjacent lymph glands, or less frequently by phlegmonous inflammation of lymph glands elsewhere or of the skin.

Historic Notes. Strangles was fairly indicated in the writings of the ancient Greek veterinarians, and was clearly described and attributed to contagion by Solleysel in 1664. It was so evidently infectious that it was experimentally inoculated by Lafosse in 1790, by Viborg, in 1802, and later, by Erdelyi (1813) and Toggia (1823) and others. Rivolta, in 1873, found a streptococcus in the pus of its absesses, and to this the contagion was definitely assigned by Baruchello (1887), Schütz, Sand and Jensen (1888). Priority in this demonstration is accorded to Schütz.

Bacteriology. The *streptococcus coryzæ contagiosæ Equi* (Streptococcus rhino-adenitis or S. equi) is easily found in the pus of gland abscesses sometimes in pure cultures (impure in the nasal discharge), stains readily in aniline colors and in Gram's solution so that it stands out clearly among the pus cells. The decolorizing agent must be weak (not muriatic acid) and applied only for a very short time. Beside the chain forms, there are isolated, oval cocci, some of which, larger than others and more elongated, have been held to be arthospores or mother cells. The number of elements articulated in a chain varies from two to four and upward. The chains are straight or sinuous, and may be grouped in bundles, radiating masses, or clumps like staphylococci.

They are ærobic (facultative anærobic), grow freely as transparent droplets on blood serum at 99° F., and in glycerine bouillon,

and less vigorously on agar and gelatine. On agar the colonies reach the size of a pin head in two days with projecting alæ, and on gelatine in three to five days, and then dry and shrink. Multiplication takes place by transverse division, and at such a time the organism may seem to be a chain of diplococci. Lignieres claims that the disease starts in a cocco-bacillus (Pasteurella), only discoverable in the early stages, and which paves the way for the streptococcus.

Pathogenesis. Inoculation of streptococcus cultures on a susceptible horse produces the unquestionable phenomena of strangles, and solipeds alone take the disease casually. In white mice it produces abscess in the seat of puncture and in the adjacent lymph glands. If the action is delayed the abscess may be in lung, spleen, kidneys, liver, or other distant organ. Rabbits, Guinea pigs, pigeons, pigs and cattle are immune unless large doses are employed. Intravenously large doses kill the lamb.

The *identity* of the microbe with other streptococci of animals and man has been claimed. Arloing alleges that, by culture of the microbe in the blood or peritoneum of the live rabbit, he exalted the virulence, and obtained in succession a streptococcus capable of producing *erysipelas; gangrenous erysipelas; suppurating, sloughing erysipelas; pseudo-membranous peritonitis; metastatic absesses;* and *fulminant septic peritonitis.* Hill, Jensen and Sand, and Lignieres, as the result of cultures and inoculations claim that strangles streptococcus is identical with that of contagious pneumonia. Courmont, on the other hand, as the result of his cultures and inoculations, concludes that the microbe of strangles and that of erysipelas are independent organisms.

The clinical evidence is decidedly against the theory of identity. In epizoötics of strangles we meet with a constant succession of cases of strangles and in districts into which contagious pneumonia has never been introduced, no single case of that disease ever comes in to break the monotony of the sequence and to start a series of cases of the latter affection. Conversely, in an outbreak of contagious pneumonia in a locality heretofore free from strangles, strangles do not develop. Again, no matter how prevalent nor how constant strangles may be in a locality, and how habitually men have their wounded hands covered with the pus of the abscesses, no epidemic of erysipelas is entailed in

man. Strangles spreads with remarkable rapidity through a stable, but not to the often more than equally exposed human attendants, nor to any animal apart from the genus equus. The absence of strangles from Iceland (Jonsson) endorses that view.

As a practical question of sanitary science, we occupy a sound position in differentiating the germs of strangles and contagious pneumonia, and further that of erysipelas of man, as a wise health officer would differentiate the microbes of cowpox and smallpox. Whatever may be true or false as to their primary identity, or as to the transition of one to the other in successive inoculations of animals of other genera, they are essentially diverse pathogenically as we meet with them in practice, and our measures may be safely based on this practical diversity.

Accessory Causes. Youth strongly predisposes, most cases occurring between two and five years, and seventy per cent. before five years. It may, however, appear at any age, being congenital in some cases (Nocard, etc.), in others appearing a few weeks after birth, and in still others at over twenty years, if the subjects have not contracted it earlier.

Dentition which is active in these early years, induces congestion about the head and general constitutional disturbance, which make the system more receptive.

Training or *breaking* is another reason for the predisposition in the young. The first experience of the *hot, impure, infected air* of the stable, the unwonted *grain feeding*, the *excitements* and *perspirations* attendant on the first handling, all contribute to temporary loss of resistance.

Fatigue like other weakening conditions lays the system open to attack.

Chill is a most efficient cause hence the disease often prevails most extensively in spring and autumn, at the time of changing the coat, and of passing from stable to field and the converse. Joly relates that in Russia where large numbers die of strangles through imperfect stabling in winter, immunity is sought through a milder first attack, brought on in the milder autumn weather by turning the young animals into a deep pool for half an hour and then exposing them freely to cold winds and giving cold water to drink. The omnipresent germ takes occasion to attack the cold debilitated system.

Any *change of latitude* or of locality acts in the same way. Riquet even alleges that this will bring about a second and even a third attack. It is common, he says, for newly bought young horses to have the disease at Hamburg, and after recovery to have a second attack at Hanover and finally a third one after they join the regiments in France. A similar exhaustion of immunity has been repeatedly noticed in the case of canine distemper.

Horse *trading* and the *stabling of large numbers* together is naturally the most fruitful of infection and hence strangles is a virtual plague in dealers' studs. The buildings in such cases are reinfected at short intervals with virulent types of the streptococcus, and fresh susceptible animals are being constantly introduced to keep it up. Riquet says that in Northern Germany dealers avoid this largely by traveling their purchases in bands of 100 or 150 head, from ten to twelve miles a day, feeding sparingly, and turning them like sheep into an open park at night regardless of the weather. Much of the advantage is doubtless from the avoidance of stable infection and the warm relaxing air of indoors.

A sea voyage especially favors infection and a single victim placed on board will speedily contaminate all susceptible animals present.

Finally the predisposing influence of *catarrh of the air-passages* must not be overlooked. The inflamed mucosa furnishes a most inviting infection-entrance.

Infecting products. The streptococcus abounds in the local phlegmons and abscesses, in the exudate of the submaxillary, pharyngeal or other glandular swellings, in the pustular eruption on the skin, and in the catarrhal discharge from the air passages. It further exists in the alimentary canal, in the ingesta and in the blood to a limited extent. In the bowels of an immunized animal it may remain virulent for months. Thus it comes that the manure is a source of infection, and that soiled fodder, litter and water may prove dangerous. The infected soil can not only harbor but can multiply the microbe, keeping it in readiness to attack any receptive horse. On his part the horse that is immune and in vigorous health may carry the infection for months and transmit it to his less resistant fellow.

While the streptococcus is usually found in the blood, in limited numbers only, its presence there implies its general diffusion

and especially in the lymph plexuses and glands. Hence, the danger of operations on the subjects of strangles, the weakened tissues of the wound forming a most inviting field of growth. Castrations occurring as they do mostly in the growing animal, are especially to be guarded against, and I may cite the case, familiar to many, in which seven cryptorchids died with phlegmon in the seat of the wound, the first one operated on having had strangles.

The nasal and buccal discharges are especially liable to convey the infection through mangers, racks, fodders, drinking troughs and pails, harness, poles and shafts, halters, twitches and the like. Infection through blankets, brushes, rubbers, and the clothes and hands of attendants, dealers, veterinarians and others, is not to be overlooked.

Pathology. The streptococcus shows a special disposition to enter and advance along the lines of the lymphatic circulation. The paucity of the germ in the blood and its abundance in the lymph plexuses, vessels and glands show that its election is preëminently for the lymphatic system. Then the ordinary primary lesions in and around the upper part of the air passage (nose, pharynx, submaxillary, parotidean and phryngeal lymph glands) bespeak infection by inhalation, rather than with the ingesta. Primary solitary lesions on or near stomach or intestine are almost unknown, nearly all such being secondary. Next to inhalation, the most prominent channel of entrance is through castration and other wounds. Abrasions and sores of skin diseases must rank after wounds as entrance channels. Transmission by copulation, the microbe being lodged on the genital mucosa, is well established, also transmission from mother to fœtus through the placenta, and from dam to offspring through the milk.

In the most familiar type of the disease the nasal mucosa is red, congested and somewhat thickened with exudate, and the epithelium is softened and desquamating. As the result of this desquamation there may be slight abrasions or raw sores but these do not show indications of the irregular outline, excavations, or progressive extensions that characterize the ulcers of glanders. The surface is usually plentifully covered with a muco-purulent material with less disposition to adhesiveness than in glanders. It is rare to see any exudate into, and thickening of the walls of

the lymphatics running from the nostrils toward the submaxillary glands. The predominance of the streptococcus in, and the entire absence of the glanders bacillus from the discharge and inflamed mucosa are conclusive. In the regular cases in which the submaxillary lymph glands are implicated, both right and left are usually involved, though not to the same degree, the exudate fills not only the gland tissue, but a large amount of the surrounding connective tissue as well, there is a great accumulation of lymphoid cells, and more or less extensive pus cavities, containing usually a white, creamy product. In the early stages the glands may be hard and nodular, as in glanders, but this condition is very transient, so that the rule is to find an extensive surrounding exudation filling up the whole intermaxillary space, and having a great abundance of small round cells with double or triple nuclei. In the older cases there is usually the open abscess, and if the case is an indolent one there may be extensive organization of the exudate with formation of dense, fibrous tissue. In some instances the nasal sinuses are filled with muco-pus.

When lesions extend farther implicating the pharynx and larynx, the mucosa of these parts shows the same redness, congestion, cloudy swelling and desquamation with, in some instances, small, submucous abscesses, and in others extensive infiltration of the submucosa with lymph so as to narrow or even close the lumen of the larynx. The guttural pouches may be filled with pus though this is far from constant. The pharyngeal lymph glands, are nearly always involved and often the lymph-gland in the parotid so that a general infiltration of the surrounding parts is met with.

If the chest is implicated there is congestion of the bronchial mucosa, engorgement of the smaller bronchia, air sacs and cells with pus, collapse, carnifaction or congestion of lobules, in some cases pulmonary abscess, and, finally, swelling and not infrequently abscess of the bronchial glands. Pleurisy is a not uncommon accompaniment, appearing it may be as a simple extension, from the lung, or, in the worst forms, from rupture of mediastinal or glandular abscesses into the cavity and severe infection of the entire pleural walls. The pericardium is exceptionally involved and coagula on the tricuspid valves have been met with (Zschokke).

Circumscribed phlegmonous exudates and small abscesses are sometimes found in the mouth (tongue, soft palate, cheeks) and less frequently in the œsophagus.

The stomach may show congestions, petechiæ, circumscribed hæmorrhages, ulcers, and abscesses of the gland tissue or submucosa. Rupture of the walls may follow abscess (Cadeac).

The intestines may show congestions, colorless or hæmorrhagic exudate, and suppurations in the agminated or solitary glands, or submucosa. The intestinal, mesenteric and sublumbar lymph glands may be the centres of abscesses of varying sizes.

Abscesses may also be found in the liver, pancreas, or spleen, of variable size and usually as secondary formations.

The muscles and intermuscular tissue may be the seat of more or less extensive exudation, or abscess, and the bones may be congested and swollen especially in their epiphyses. This may extend to suppuration or necrosis.

The synovial membranes of joints and the tendinous sheaths are not infrequently inflamed, causing distension and even suppuration.

Congestions of the skin are sometimes met with, developing as multiple papules or hard nodules which advance to the formation of pustules or small abscesses. These often appear especially where the skin is thin and delicate as around the lips, nose and eyes, close to the anus or vulva, in the perineum, sheath or mammæ, inside the thighs or elbow. They may be of all dimensions from a millet seed upward, and may merge into or become complicated by the extensive engorgements of petechial fever.

Finally, lesions of the nerve centres are to be looked for in the protracted or irregular types of the disease. There may be simple congestion, or serous effusion, intraventricular or subarachnoid, or finally abscess in the brain, spinal cord or meninges.

Forms of Strangles. The types of strangles vary, special forms characterizing given epizootics or seasons, or at other times as individual deviations from the current type. Division has been made into two groups—*mild* and *malignant*, or again into *regular* and *irregular*, under each of which come several varieties. Many of these varieties consist simply in a difference in the seat of the principle lesions, which start in lymph glands or tissues at a distance from the nasal mucosa, or they depend on secondary foci of infection supervening on the primary disease in the head.

Incubation. In inoculated cases this lasts from three to five days, in those due to simple exposure it may appear to extend over eight days.

General Symptoms. In nearly all cases alike there is a marked constitutional disturbance, the temperature often rising at once to 104° to 106° F., and oscillating on successive days between this and 102° ; the pulse is usually normal at first and the breathing is either slightly accelerated or may be made so under slight exertion. There is more or less dulness, or at least a lack of the vivacity of youth, the head is somewhat pendent, the eyes may be semiclosed, the patient may be tardy or even stiff in his movements and the appetite may be diminished or capricious, oats being rejected by some and hay or grass by others. The bowels are somewhat confined, the stools consisting of a few small, hard balls covered with mucus.

Specific Symptoms in the Mild Form. In the vast majority of cases the local symptoms are concentrated on the mucosæ of nose and mouth, and the submaxillary lymph glands. Along with the general febrile phenomena, there appear redness and often mottled congestion of the nasal mucosa, which not infrequently extends to the mouth and eyes, as well. Heat of the mouth, the collection of a more or less tenacious mucus, and even uneasy movements of the jaw may be seen. The nasal mucosa, at first dry, is soon the seat of a watery exudation, passing into a cloudy sticky material, and finally a thick, opaque, mucopurulent flow. This may become colored in a variety of tints, dirty white from inhaled dust, brownish or yellowish from exuded blood, or greenish from food materials. The discharge is usually profuse in the young and may be scanty in the old. Most commonly it flows from both nostrils alike, though exceptionally it is unilateral. In this it differs from glanders which is more often unilateral though at times bilateral. Small abrasions and sores may appear in connection with the softening and shedding of the epithelium, but these are not ragged, irregular and spreading as in glanders. Sneezing or snorting is an inevitable symptom. Epiphora is usually present. Exudations into the nasal sinuses are to be recognized by heat of the forehead and flat sound on percussion.

Very early in the attack a swelling is noticed in the intermaxillary space, which may be at first confined to the nodules of the lymph glands, thus forming distinct, rounded, hard swellings,

but they are early covered by a diffuse exudation into the surrounding connective tissue, that completely envelopes and obscures the form of the swollen glands, and forms a more or less uniformly rounded, pasty swelling, extending to the median line of the intermaxillary space, or filling the whole space from one maxilla to the other and projecting downward below their level. This early, diffuse, pasty, evenly rounded swelling, hot and tender, is distinctive of strangles, and usually exclusive of glanders.

Another characteristic of the strangles swelling is its steady, and usually speedy, advance to suppuration and abscess. It becomes hard, tense, and resistant, then, in the centre, or at various points of the surface, small areas of circumscribed softening can be detected, and soon show distinct fluctuation. Two or more of these may coalesce or they may form several distinct abscesses, which may early point, burst and discharge, when the remainder of the exudate softens and degenerates into pus, and the cavity closes by granulation. In some cases after the formation of the swelling it disappears by resolution, the exudate becoming liquefied and absorbed. In glanders the nodular, insensible swelling tends to persist without extensive pasty exudation or suppuration.

Cases of *strangles catarrh* in which the sub-maxillary lesions are omitted, are quite common. These occur during the regular strangles epizoötic, and protect against a second attack.

Symptoms of Pharyngeal and Laryngeal Strangles. Extension of the morbid process from nose to pharynx is exceedingly common. When concentrated on the pharynx there are extension of the head forward with elevation of the nose, swelling of the throat laterally or downward, uneasy movements of the jaws, salivation, difficulty of swallowing, return of ingested liquids through the nose, gulping, and a loose suffocative cough. The swelling of the throat tends to attain to large dimensions, and may threaten suffocation by interfering with the breathing. This is still further aggravated if the laryngeal mucosa is the seat of exudate. The breathing may become loud and stertorous, the mucosæ of a dark leaden hue and the animal dull and stupid from the venous condition of the circulating blood.

Abscesses forming on the lateral parts of the throat usually make their way to the surface, though this may be below the level of the parotid. If from the parotidean lymph gland, one of the

ducts may be opened, thus forming a salivary fistula. If from the retro-(supra-) pharyngeal glands, the rupture into the pharynx is more likely to take place, but in some cases the investing sac, meeting with equal resistance in all directions, fails to undergo degeneration and softening at any one particular point, and the contents remain pent up indefinitely. If the liquid is absorbed a cheesy or putty like mass may be the final outcome, with chronic cough, some stertor in breathing and it may be difficulty in swallowing.

If the guttural pouches should be involved, there is deafness, parotidean swelling, which may eventuate in a fluctuating swelling at the lower border of the parotid, and a free discharge when the head is lowered, which is likely to last after general recovery. (See Guttural Pouches, pus in).

Laryngeal paralysis and roaring often follow laryngitis in strangles.

Pulmonary Symptoms in Strangles. *Tracheitis* and *bronchitis* are forms of extension of strangles from the upper air passages and *pneumonia* follows of virtual necessity. In many cases these are primarily dependent on the descent into the lungs of the infecting discharges, complicated in many cases by the inhalation of food materials. There are the usual symptoms of bronchopneumonia complicating those of strangles and the percussion and auscultation signs usually imply circumscribed areas of congestion and consolidation with intervening areas of pervious lung. There may be at such points the blowing or mucous râles of bronchitis, the sibilant sounds of emphysema, the crepitation of congestion and the abnormal clearness of sounds carried from distant organs through the consolidated lung. On percussion there may be the non-resonance of the consolidated areas, and the excess of resonance over emphysematous portions or open gas-filled vomicæ. In these last cases there may be an amphoric sound on auscultation and a crack-pot sound on percussion. These pulmonary lesions are often fatal, or the recovery is slow on account of a succession of lobular congestions and absesses.

Abdominal Symptoms in Strangles. The abdominal lesions in strangles are usually secondary, the infection reaching the part through the blood, or by the lymphatics from a castration or other wound, or from infection by coitus. The phlegmon and

abscess may be in the mucosa, especially in the agminated or solitary glands, in the adjacent lymph glands at the connection with the mesentery and in those of the mesentery itself. The animal is dull, listless, with dry, staring coat, tympany and slight colicy pains after eating, costiveness, retracted, tender abdomen, insensible loins, and groaning when rising, when walking down a steep incline, or turning in a very narrow circle. These symptoms following an apparent or partial recovery from strangles are significant, and rectal examination may detect a hard, tender mass connected with the bowel or mesentery.

If rupture takes place into the peritoneum there is general infected inflammation of that structure with sudden access of fever, marked prostration and an early death. In more favorable cases its adhesion to the bowel or to the abdominal wall opens the way for rupture into the gut or externally and there may be a slow healing of the cavity by granulation. It may be a month or two before such an abscess opens and for a length of time thereafter the health is poor, and the animal lacking in condition and endurance.

When the abscess is formed in the *liver* there is high fever with shivering fits, irregularity of the bowels (bound up or loose), dusky or yellowish hue of the visible mucosæ, anorexia, followed by peritoneal infection or pyæmia (secondary abcesses).

Abscess of the *pancreas* or *spleen* is even less definite in symptoms. These may terminate in rupture and peritonitis, or the splenic abscess may become chronic and indolent and in a measure harmless.

Perirenal Abscess is betrayed by specially sensitive loins, stiffness and groaning in rising or in turning sharply on himself, drooping of the back under a load, and by albuminous urine. In a small animal the part may be reached and the tenderness elicited by handling.

Cutaneous Symptoms in Strangles. Though by no means a common form, strangles sometimes attacks the skin, more particularly that of the face, head and neck, appearing in the form of pustules or small abscesses, or it may be of a rounded nodular elevation, which may disappear without forming either vesicle or pustule. The points of election are around the lips, nose and eyes, upon the mucosa inside the lips, along the line of the facial

lymphatics running toward the submaxillary gland, and at points where there is special friction, as under the halter, collar, saddle, crupper, in the hollow of the heel, under the tail, on the perineum, in the groin and axilla. They may extend more or less up the limbs, or around the point of primary attack attended by more or less engorgement. When this engorgement has reached extensive dimensions and is mixed with sanguineous extravasation it is considered as having merged into petechial fever.

Symptoms of Coital Infection. From four to seven days after copulation there appear fever, dullness, stiffness, anorexia, swelling and heat of the lips of the vulva, a yellowish opaque discharge from its lower commissure, a deep dark red blush of the mucosa, with points of distinct infiltration and thickening, developing into vesicles and pustules. The perineum, the groin and mammæ often show an extension of the congestion and eruption. In exceptional cases deep abscesses form and Letard records a fatal case with extensive suppuration among the muscles of the hind limbs and the haunch, inside the pelvis and along the line of the aorta.

Symptoms of Nervous Lesions. The lesions of the brain and spinal cord are usually secondary and often appear when the less dangerous superficial manifestations, are tardy and indolent, when the exudates are indurated and indisposed to soften. There may be violent delirium, pushing of the head against the wall, movements of the limbs as if walking or trotting, rearing, plunging, striking with fore or hind feet, trismus or other muscular spasms. More frequently there is great dullness, prostration, debility, vertigo, drowsiness, amaurosis, paraplegia, general paralysis, coma. If the lesion is in the spinal cord the spastic or paralytic symptoms are likely to be confined to the hind parts.

Fulminant or Septicæmic Form. Bigoteau describes a rapidly fatal, septicæmic form, with sudden onset, anorexia, extreme prostration, uncertain stumbling gait, a deep blue color and ecchymosis of the visible mucosæ, violent heart action, pulse weak and small, hurried breathing (45 per minute) temperature 102° to 106° F., often inability to rise and death from asphyxia in from two to five days.

Diagnosis. In mild and regular cases this is easy. The attack in rapid succession of all the young and still susceptible horses

in a stable or locality, and the uniform coincidence of a profuse nasal catarrh, and the formation between the branches of the lower jaw, of a diffuse, hot, painful swelling rapidly advancing to suppuration and discharge are virtually conclusive. Simple catarrhs even if infective and attacking all young horses do not cause phlegmon of the sub-maxillary lymph glands as occurs in the great majority of cases of strangles. Glanders which is attended by both nasal discharge and sub-maxillary swelling, is slower in its onset, usually with little or no fever, has usually a more adhesive discharge, ragged, unhealthy ulcers on the nasal mucosa with a disposition to extend, often it shows cord-like thickening of the lymphatics on the side of the face, and the sub-maxillary swelling is smaller, made up of a number of small, hard, insensible rounded nodules which show virtually no tendency to suppurate. (See diagnosis of glanders). In cases of doubt it may be advisable to inoculate a Guineapig and a white mouse. The Guineapig resists a small dose, while the mouse forms abscess in the seat of inoculation and the dependent lymph glands. The Guineapig is very susceptible to glanders, and the white mouse immune. Or mallein may be used.

Prognosis. The mild type of strangles almost invariably terminates favorably. In the irregular types with internal abscess the prospect is grave in ratio with the size and multiplicity of the foci and the vital importance of the organ invaded. In 15,421 cases collected by Friedberger and Fröhner, and representing the total in a series of outbreaks, but 3 per cent. proved fatal. Much, however, depends on the special potency of the germ. Horses contracting this in a particular year or from a given stable in the same year convey the disease to others in a malignant form, while others that contracted it in another year or a different stable infect with the mild form only.

Prevention. In the older countries horsemen too often accept strangles as inevitable. They expect that all horses will have it sooner or later, and it is not worth while to guard against it. The too absolute doctrine of the identity of the germ with the microbe of erysipelas, puerperal fever, contagious pneumonia, influenza, of the suppurations of the limbs and feet in sheep and cattle, and other streptococcic infections seems to corroborate this view. But on the other hand the absence of strangles from given

countries like Iceland where erysipelas and its coadjutors are common, its absence from secluded breeding farms and districts in America, though prevailing all around them, the rapidity of its spread when introduced in a sick colt, and the entire failure to extend in the same way from an erysipelatous man to susceptible young horses with which he comes in contact show that preventive measures may be successfully applied for its restriction and extinction. The conceded family resemblance of the microbes and the experimental production, by their inoculation, of lesions showing many points of similitude leave them still sufficiently distinct in their pathogenesis to warrant measures for the suppression of the variety which produces strangles.

Precautions for the private owner. During the existence of strangles in the district, *exclude strange horses, asses or mules* from the farm or stable. Keep young susceptible horses from *public stables or yards* (livery, feeding, training, fair, market, and above all, dealers' and sale stables) and even from *public drinking troughs* and *buckets* used in common. Provide against their contact with *manure* from strange or infected stables, or with *pastures, fields* and *wagons* on which this has been put, also against the use around the stable or on fodder of *forks* or *other implements* that have been used for such manure. Avoid *hay* or other *fodder* or *litter* from a strange barn or one that is open to any suspicion of infection. Avoid *running water* that has drained land, stables, or yards where strange horses have been, or those open to suspicion. In shipping by car or other *public conveyance* disinfect the latter before the animal is loaded. If a *second hand wagon, shafts, pole, harness, blanket* or other object is brought on the place or used, disinfect the same before using.

Measures for Sanitary Police. Make it *compulsory to report,* under penalty for failure, all cases of strangles, or of horses with nasal discharge, or submaxillary swelling. Forbid removal from the stable, or secluded enclosure, of all horses, etc., suffering in this way or which have been pronounced by the official veterinarian to have strangles. Provide for *exclusion of all other solipeds* from such *stables,* or from *contact with* or *dangerous proximity* to animals held in them, also from *infected yards, parks, cars, boats, etc. Close public drinking troughs* during an epizoötic; let each owner use his own bucket. Circumstances may demand closure of public feed-

ing stables as well. *Forbid*, under penalty, *sale*, *exposure* or *movement* on any *public highway* or *unfenced place* of any infected (diseased or exposed) soliped. Enjoin *certificate of sanitary condition of stable and stud* with each animal sold. *Close dealers' stables*, or forbid any sale from them until all infection has ceased and the buildings have been thoroughly disinfected. Compel thorough *disinfection of stables*, *yards*, *cars*, *boats and other public conveyances* that may be open to reasonable suspicion of infection.

Immunization. An attack renders the subject immune, but this may be early overcome by marked change of location, and exposure to a virus of greater intensity or modified quality. Besides the crude and reckless Russian method already referred to, direct inoculation of the virulent products has been often resorted to from the time of Gohier onward, in the different countries of Europe. By selecting the matter from the abscesses or nasal discharge of a mild epizoötic, and preserving the inoculated subjects in clean, dry, pure aired stables, on nourishing diet, and under the best conditions of hygiene, a fair measure of immunity was conferred with absolutely no loss. The same as after casual cases, second attacks will sometimes be shown, but even in severe outbreaks, of a less violent type. The pus from the abscess, or the preceding exudate in the swelling may be simply rubbed on the nasal mucosa, or injected subcutem in another part of the body. In many cases there follows merely a local cellulitis, while in others the general infection leads to the nasal discharge, with or without the submaxillary abscess.

This method is open to the individual owner where no concerted effort is made to *stamp out* the infection. When, however, police measures for suppression are in force, it must be strictly prohibited, or adopted only under official control, and with absolute seclusion and thorough disinfection.

Treatment. In mild regular cases hygienic measures only are demanded. Cleanliness, dry stalls, pure air, warmth (a sunny exposure if available), nourishing, easily digested food, (grass, green corn stalks, bran mashes, roots, carrots, turnips, apples, potatoes, ensilage, scalded oats or hay) and pure water, or linseed tea, grooming and, in cold weather, blanketing may suffice. Rest is indispensable, though exercise may be allowed in a sheltered or sunny field or yard in fine weather.

Castrations and all other surgical operations are forbidden.

For costiveness or tardy action of the bowels 1, 2 or 3 ozs. of sulphate of soda may be allowed daily in the drinking water.

To soothe the inflamed air-passages it is well to steam them with the vapor of hot water to which has been added an antiseptic such as oil of tar, tar, phenic acid, or creolin. This may be placed in a bucket, and a bag with its bottom cut so as to form a tube drawn over the bucket and nose of the horse. It may be continued an hour or more at a time, or a nasal douche of creolin (1 : 100) may be employed. In case of tardy softening of the submaxillary swelling it may be assiduously fomented, or covered with a linseed meal or other poultice to which a little antiseptic (carbolic acid, creolin) has been added. It may be applied on a cotton hood having holes for eyes and ears and furnished with ends to tie back of the ears and down the middle of the face. If still indolent the swelling may be rubbed with soap liniment or smeared with soft soap, or finally a cantharides blister may be applied.

As soon as any indication of softening or fluctuation is detected a free incision should be made to allow the exit of the pus. This further tends to hasten the liquefaction and removal of the adjacent exudate. If the pus lies near to the surface, with little more than skin to penetrate, it may be freely incised with one thrust of the knife, but if there is intervening glandular or other tissue, the skin only should be first incised, and the connective tissue bored through with the finger nail, or the point of a sterilized director, or of closed scissors. In this way the important vessels, nerves and salivary ducts are pushed aside and troublesome bleeding and salivary fistula alike avoided.

Fever usually subsides on the opening of the abscess, but if it fails to do so, or if it reappears from slight absorption of septic matters it may be desirable to favor elimination by small doses of sodium bicarbonate, ammonium chloride, or potassium nitrate. In extreme cases a few doses of acetanilid may be given, or full doses of quinia.

When the discharge from nose or abscess threatens to persist, such agents as sulphur, yellow or black sulphide of antimony, are given with bitters, but a more prompt effect can usually be had from injections of weak solutions of creolin, cresyl, lysol,

etc. When the cough is troublesome it may be quieted by belladonna, or, in case of weakness of the heart, by digitalis.

The various complications require treatment appropriate to their nature. Collections of pus in the nasal sinuses may demand trephining and antiseptic injections. Swellings about the throat threatening asphyxia and which cannot be relieved by evacuation of pus may necessitate tracheotomy until suppuration occurs. The cutaneous pustules and abscesses are dealt with by pricking the collections and washing daily or oftener with astringent antiseptics (phenic acid and alum). Bronchitis and bronchopneumonia may be benefited by sinapisms applied to the sides of the chest, the internal administration of potassium iodide, ammonium chloride, sodium hyposulphite, terpene or terpinol; or by inhalation of weak sulphur fumes, or tar vapor.

For abscesses in the bronchial or mesenteric glands, the brain, spinal cord, or other internal organ, little can usually be done but to sustain the patient and await the course of events. If the abscess can be accurately located it may be aspirated and then injected with an antiseptic; if in the encephalon, trephining may be resorted to; death is certain in such a case in the absence of treatment.

During convalescence it is very important to avoid over-exertion and chill, which are very liable to bring on petechial fever. Also, to feed nourishing food, give pure air and water, and to see that no suspension of action of bowels, or kidneys threatens to shut up toxins and waste products in the system. *Apropos* of impure water Williams quotes the case of a foul stream near Bradford, England, on the banks of which every case of strangles did badly.

CONTAGIOUS PNEUMONIA IN THE HORSE.

Synonyms. Definition. Historic notes. Accessory causes: youth, native susceptibility, inclement weather, exposed stables, nasal and bronchial catarrh, sores as infection atria, lack of stable hygiene, crowding, underfeeding, overwork, excitement, exhaustion, infection from stables, etc., convalescents; doctrine of recrudescence. Bacteriology: streptococcus pneumoniæ contagiosæ equi: pathogenic to mice, rabbits, and Guinea pigs. Coccobacillus of Lignieres in early lesions. Lesions: bilateral, multiple foci, congestion, consolidation, purulent, necrotic, infarction, sequestra in purulent sacs, pleuritic effusion; enlarged, congested liver with centres of degeneration and necrosis. Congested spleen, kidneys, lymph glands and gastro-intestinal mucosa. Yellow mucosæ, mahogany colored muscles. Incubation, 3 to 10 days. Symptoms: staring coat, early extreme hyperthermia, accelerated pulse and breathing, cough, icteric mucosæ, anorexia, dulness, defervescence in 3 to 5 days; convalesence in 10 days; or prostration, swollen eyelids trembling or interrupted labored breathing, cough, nasal flow yellow, multiple centres of percussion flatness, crepitation, râles; urine scanty, yellow or reddish, albuminous, acid, alkalinity as a symptom; throat symptoms, inhalation bronchitis, cardiac phenomena, swelling of legs, stupor, trembling, staggering, vertigo, paresis. Course: duration 2 to 3 weeks, defervesence. Fatal cases, toxin poisoning. Diagnosis: by age, history, tardy infection, prostration usually less than in influenza. Prognosis: gravity depends on violence of attack, susceptibility, hygiene, treatment. Mortality 1 to 20 per cent. Permanent lesions from sequestra, adhesions, cardiac, hepatic, nervous or arthritic disease. Treatment: Pure air, sunshine, comfort, hygiene, pure water, rest, cold rectal injections, damp compresses, hot bath, diaphoretics, expectorants, alkaline diuretics, antipyretics, heart stimulants, derivatives, antiseptics, nerve sedatives, tonics. Prevention: early removal, disinfection, quarantine new horses, disinfection of public and sale yards and stables, certificates. Immunization: by a mild attack; serum-therapy.

Synonyms. Ataxic or Adynamic Pneumonia; Stable or Hospital Pneumonia; Pleuro-Pneumonia Contagiosa Equorum; Contagious Pleuro-Pneumonia; Bilious Pneumonia; Edematous Pneumonia; Brustseuche.

Definition. An infectious adynamic type of pneumonia occurring in horses, asses and mules, characterized by marked hyperthermia; by infiltration of lung tissue, often bloody, infarcted or caseated and usually circumscribed; by a deep yellow discoloration of the visible mucosa and other white tissues; and by

complicating lesions of the pleura, heart, pericardium, liver, bowels, or kidneys.

Historic Notes. This affection was formerly confounded with equine influenza, and it was only in the second third of the nineteenth century that the differentiation was attempted. S. Prangé describes this as a special epizöotic disease in the French hussars in 1841, Leconturier in Belgium in 1845, Seidamgrotzky in Germany in 1832 (strongly emphasizing the contagion), and Dieckerhoff in Berlin in 1883. The latter showed that horses, recovered and immune from influenza, still contracted brustseuche and perished. This distinction was fully corroborated by Lustig, Cagnat (1884), Brun, Delamotte (1886), Jolly, Benjamin, Leclainche, Trasbot, and others. The presence of a particulate, living, self-multiplying cause (microbe) was recognized as the essential condition of the disease (as we still recognize the necessity for such an organism to explain rabies) though the micro-organism itself was as yet undiscovered.

At the same time many concurrent factors had to be considered as accessory in different cases.

Accessory Causes. *Young horses* often show a greater susceptibility than older animals, mainly because they retain all the unimpaired susceptibility of the colt, while old horses have already passed through the disease and become immune. On the other hand, in the absence of acquired immunity, the older, *worn out and debilitated animals* are the most susceptible and tend to have the disease in its worst form. *Susceptibility* and *immunity* are therefore more important factors than mere age. Immunity usually lasts for several years, or throughout life, yet in some animals, or under given conditions, it is overcome much earlier. *Inclemency of the weather*, or special exposure of any kind, as in severe rainstorms, or working with the feet and legs in water, may become the occasion of an attack. Exposure to cold *north-west storms* (America, Atlantic Slope), or north-east (Europe), *standing without blanket* in a temperature at zero, confinement in *draughts of cold air* between doors or windows, without clothing and after severe exercise, weaken the whole system and increase susceptibility. The presence of a *nasal or bronchial catarrh*, or of another debilitating disease may act in the same way. The weakened tissues seem to invite the entrance of the germ. Palat, Boi-

teux and Trasbot found that horses with *local sores* or *suppurations* fell readier victims than others;—perhaps the germs entered by the traumas; perhaps the tone of the whole system was lowered, so that the resistance was lessened.

Close, foul air, bad ventilation, imperfect sewerage, and *overcrowded stables* not only contribute strongly to infection but tend to aggravate the cases. *Underfeeding* and *overwork* act in the same way and in this connection may be named the *excitement* and *exhaustion* attendant on a *long journey by rail.* This, like the foul, crowded stable, furnishes many more opportunities for infection and re-infection, so that the invasion of the exposed animal system is all but certain. Infection clings to the *loading-banks, yards, feeding stables, mangers, troughs, buckets, cars, litter,* and *manure,* so that young horses shipped from the west to the Atlantic Coast States, very frequently come down with contagious pneumonia, and contaminate the stables in which they are placed. Peters suggests that the germ is preserved in the *soil water,* so that after apparent subsidence it may be again brought to the surface in time of rains or freshets, to start a new epizoötic. *Convalescent horses* may carry the germ for weeks, on the mucosa or in sequestra in the lungs, and contaminate horses with which they come in contact.

It is remarkable that the contagious pneumonia is far less diffusible on the air than influenzá, so that it is much more constantly the result of direct contact of a sound, with an infected animal, or with a place or thing that the sick animal has contaminated. It therefore spreads much less rapidly, remains confined to individual stables for a length of time, and in the absence of active interchange of horses tends to die out of its own accord. As the infection is not generally and speedily acquired, so immunity fails to become general, and the infection tends to fix itself permanently in places where many strange horses congregate, (market stables, sale stables, livery stables, etc.), and the constant influx of fresh animals keeps the flame burning by accessions of fresh fuel. In such cases it is manifest that the germ outside the animal body either rests in a dry condition, or lives as a saprophyte in earth or organic matter, and often loses much of its virulence, Under such circumstances animals that would prove readily susceptible to a virulent germ, prove non-

receptive to this resting germ, until under some special devitalizing influence, like exposure, exhaustion or local disease, it finds its opportunity and the weakened system succumbs. Then, acquiring new force through its life in the debilitated system, it starts on a recrudescence, and an epizoötic is mistakenly supposed to have started without a preëxisting microbian cause.

Cadeac even advocates the theory that the same germ possessed of greater or lesser virulence, is always present in ordinary stables and horses, and habitually causes in exposed or debilitated animals an ordinary fibrinous pneumonia with no perceptible tendency to transmission by contagion; that, in other cases when a considerable number of horses have their defensive powers impaired, it gains a wide extension; and, that in some such cases, the germ that has been living as a comparatively harmless saprophyte, suddenly acquires an unwonted potency, and breaking down the barrier of partial immunity, attacks exposed animals on a large scale and irrespective of weather perturbations, or debilitated conditions. He quotes from Trasbot instances that seem to support this hypothesis, which is not at all in disaccord with the habits of bacterial life, yet we require a solid basis in bacteriological experiment to make it unassailable.

Bacteriology. Siedamgrotzky (1882) found in the hæmorrhagic centres in the affected lung and in the pleural exudate *micrococci.* Dieckerhoff (1882) and Mendelsohn (1883) found in the pleural exudate streptococci. Chain cocci were also found by Peterlein (1884), Perroncito (1885), Delamotte and Chantemesse (1888), and Mosselman and Lienaux (1893).

Schütz (1887) found a diplococcus which he studied very fully and this is corroborated by Lustig's ovoid bacterium, by Cadeac's micrococcus and diplococcus. In Dr. V. A. Moore's cultures, at the N. Y. S. Veterinary College, cocci were found constantly in pure culture, sometimes as a diplococcus, but under slightly altered conditions the streptococcus form predominated. As the difference between two, and three or more cocci in chain form is merely a question of early or late separation of cocci which multiply in line, the apparent discrepancies in the above observations do not imply any real difference in the microbe.

Inoculated in pure cultures the Schütz diplococcus killed mice in 24 to 48 hours with enhanced virulence of the germ. In the

rabbits, *subcutem*, it usually killed in 24 to 48 hours, but some survived ; *intravenously* or *intratracheally* it killed more certainly and speedily and in either case with pleural, pericardial and even peritoneal lesions. In the *Guineapig*, *subcutem*, it caused extensive effusion, and death in two to six days with chest lesions. In the *dog* it caused hyperthermia, but no marked lesion and no mortality. In the *horse* there were no infectious resultant lesions. A pure culture thrown into the lung tissue of an old horse at the N. Y. S. V. College, determined an extended pleuritic adhesion and lung hepatization. The age of this subject was opposed to any marked susceptibility. The apparent immunity of the horse in Shütz's cases might depend on the insusceptibility of the animals selected during or after an epizoötic, or on the absence of the predisposing causes so strongly insisted on by Cadeac.

Rats, chickens and pigs proved immune.

On peptonized gelatin at 98° F., and less rapidly at ordinary temperature, it grew as white, opaque colonies which gradually extended and united in many cases. The gelatin was not liquefied. In peptonized bouillon it produces turbidity for one or two days, after which the microbe precipitates leaving the liquid clear. The reaction is unchanged.

It lost virulence rapidly when kept in artificial culture or at a temperature of 122° F., and was killed by a temperature of 150° F. Yet it survived drying at moderate temperatures. Cadeac found that the dried expectoration or blood diffused in the inspired air produced pneumonia with certainty in solipeds. Schütz and Fiedaler injected pure cultures into the lung, and in other cases into the trachea, thereby inducing pneumonia. Twenty grammes of the culture injected into the trachea raised the temperature 2° or 3°, but this lessened on repetition and after four or five treatments the subject proved immune.

Lignières (1897) discovered his *cocco-bacillus* in the exudation in the tissues in the early stages of contagious pneumonia, from which it disappears, giving place to other bacteria, and usually streptococcus, as the disease reaches its maximum. (See Equine Influenza for description). His theory is that the cocco-bacillus, which is slightly smaller than the bacillus of chicken cholera, and appears like a diplococcus when stained, and which may not be found after the first eight days of infectious pneumonia, is the

starting point of disease, in this and influenza, making the system very receptive of the streptococcus of strangles and of other bacteria, the identity of which determines the nature of the malady. The diplococcus or streptococcus of Schütz in his opinion is none other than the strangles streptococcus which, finding a congenial home in the animal invaded by his cocco-bacillus, pervades the system and determines the pathological phenomena of contagious pneumonia.

There are certain obstacles to the unreserved acceptance of Lignières' conclusions, among the chief of which is the absence of evidence that horses, successfully inoculated with his cocco-bacillus in pure cultures, can infect others standing near them with the same rapidity and certainty, as does the casual case of influenza, or even of contagious pneumonia. The same holds true of the supposed identity of the streptococcus and that of strangles. Abscesses containing streptococci, were formed in the seats of inoculation with Schütz's organism, but there is no evidence that the horses suffering from such abscess affected susceptible horses standing beside them, as do ordinary cases of strangles. The cocco-bacillus may be a concurrent cause of contagious pneumonia, but we need more proof to show that it is the essential cause, even as we need proof of the absolute identity of the streptococcus of strangles and brustseuche.

Lesions. The pneumonia is far more likely to be double than in the fibrinous form, and the area invaded, in its ratio with the high intensity of the fever, is usually less. The consolidations are especially common near the lower borders of the anterior parts of the lungs. There may, however, be a number of centres in each lung to be accounted for by the inhalation of the germ and the starting of the morbid process at the various points on which it falls. This, like the double character of pneumonia is therefor in keeping with the contagious origin. Each centre of condensation shows a small area, hepatized, purulent or necrotic, with a surrounding zone of dark bluish red congestion. The consolidated areas are less dry and granular than in fibrinous pneumonia, seeming to be largely infiltrated with a still liquid exudate and dark blood, and thus tend to a greater tenacity, and less friability. Black areas of infarction form in the lung, the thrombosis of the arteries, cutting off the free normal circulation and

the isolated portion fills up with dark blood globules and forms a sequestrum. In the inflammatory and still living parts the color is lighter with, it may be, some straw colored exudate, and always an active leucocytosis, as in other inflamed parts. When infiltration is located near the root of the lung, it is usually attached to the primary bronchi, or larger bronchia and may extend into the upper portion or almost the entire substance of the lung.

Simple abscess is rare, yet purulent sacs containing the gangrenous masses or sequestra are common.

Pleuritic areas are common over the congested and hepatized foci, yet as these are usually circumscribed in extent, an excessive hydrothorax is exceptional. Yet the pleuritic effusion may at times become abundant. Friedberger and Fröhner say seven gallons or more. It may become purulent or even septic, exhaling an offensive odor. Adhesions and fringes on the pleura are frequent.

The *heart and pericardium* may be affected, the first showing the pallid, soft, or parboiled appearance of high fever, with at times fatty degeneration or petechiæ, and the latter congestion, exudation, thickening, false membranes and liquid effusion.

The enlargement of the *congested liver* is a marked feature. It frequently attains the weight of 30 pounds. It may ooze dark blood freely from the cut surface, has usually a yellowish tinge, and shows points of fatty degeneration or even of commencing necrosis. The spleen is like the liver, charged with blood, and shows an increase of pulp and even petechiæ or circumscribed hæmorrhages.

The *kidneys* are congested, friable and petechiated.

The *bronchial lymph glands* and less constantly the mediastinal and abdominal ones, are congested, pink to dark red and somewhat enlarged.

The gastric and intestinal mucosa may be congested, thickened, hæmorrhagic or ulcerated.

The white tissues generally tend to an icteric hue, and the muscles assume a mahogany aspect.

Incubation appears to be longer than in equine influenza, varying in different cases from 3 to 10 days.

Symptoms. These vary greatly in different cases, *mild* and *severe*. Some, in the same stable with the severe cases, simply

refuse food, are a little sluggish in work, cough, have hyperthermia (104° to 106° F.), respirations 20—25, pulse 60, with conjunctiva only moderately yellow, a slightly yellowish discharge from the nose, and no observable lung consolidation. The temperature descends to normal in three to five days, the symptoms generally abate, and the animal may be convalescent in eight or ten days.

In the more *severe cases* there may be seen a shivering fit, or it may pass unobserved. Then the first morbid phenomenon is usually a rapid and extreme elevation of temperature which may reach 104° or 106° F. in a few hours. With this there is great impairment or complete loss of appetite, and a loss of life and energy. In some cases the depression, stupor and muscular weakness suggest influenza but this is not the rule. Still more rare is infiltration of the eyelids and free watering of the eyes, yet in the absence of this, drooping of the upper eyelids is not uncommon. The respiration may be accelerated and short, from 20 to 30 per minute, and the pulse, which is usually small and weak in spite of the fever, may rise to 50 or 70 per minute. The breathing may be trembling or distinctly interrupted in the course of inhalation or exhalation, short and with no interval between inspiration and expiration. Cough may or may not be a marked feature, heard at long intervals only in some cases and frequent and painful in others. It is liable to be dry and husky rather than hard, loose or gurgling. The eye and to a less extent the nasal and buccal mucosa tend to show a yellowish shade, and this may even at an early stage show a distinct brownish orange, or even a dark mahogany hue. Yet dropsy of the lids or even epiphora are uncommon. A yellowish discharge from the nose is an almost constant feature and this may dry up into a yellow crust on the floor of the anterior nares and adjacent skin. The percussion and auscultatory indications of lung consolidation are rarely obtainable before the end of the second or third day and when at all extensive can usually be detected on both sides. Trasbot considers the double pneumonia as almost pathognomonic of contagious pneumonia. When confined mainly to the lower parts of the lungs and occurring in isolated areas, with lung tissue still pervious to air in the intervals, it comes more nearly to being so. Crepitation round the border of consolidated areas, is a more

marked feature than in equal consolidations in influenza. It often becomes inaudible again as the disease advances. Blowing murmurs, coarse mucous râles, heart and intestinal sounds can often be heard with unusual clearness, in unusual situations, when an area of consolidated lung is immediately beneath. A transient dry friction sound of commencing pleurisy is sometimes detected over a tender intercostal area, but soon giving place to the uniform quiet of effusion rising to a given horizontal level. Later still there may be the creaking sound of organizing false membranes in process of being stretched, and which is so often confounded with crepitation. The indications of pneumothorax (tympanitic resonance, and metallic tinkling), are rare. In advanced stages there may be tympanitic sound from the cavities of abscesses or the sacs containing sequestra.

The urine is always scanty and high colored and may at times prove red and hæmorrhagic. Albuminuria is usually present when the disease is at its height, The same is true of uric acid, which replaces the hippuric acid, in cases of high fever and complete abstinence so that the products are drawn from the disintegrating tissues alone. The returning appetite, and the restoration of a neutral or alkaline condition of the urine, therefore tend to occur simultaneously, and to mark improvement.

Great tenderness of the throat, protrusion of the nose, and difficulty of swallowing mark the localization of the lesions on the pharynx and larynx. It is liable to be accompanied by the introduction of exudation and food materials into the larynx and trachea with the occurrence of inhalation bronchitis and pulmonary gangrene.

Symptoms of pericarditis, endocarditis and myocarditis, are especially common in the more severe types of the disease. With soft, weak or imperceptible pulse and tumultuous heart beats they may be suspected, and further indications are a transient friction sound, synchronous with the beat of the heart, intermissions, murmurs with first or second heart sound, and an increasingly low, distant, or muffled heart beat, as pericardial fluid accumulates. As in the case of troubles with the kidneys or liver, stocking of the legs, or dropsical swellings elsewhere may appear.

Exceptionally, acute nervous symptoms may appear, due to functional derangements caused by circulation of the toxins and

metabolic products, or even to congestion or inflammation of the brain or its membranes. This may occur at the outset of the disease indicating the election of the nerve centres for the colonization of the microbe, and advancing to a rapidly fatal issue (Friedberger and Fröhner). It may set in with hepatization (Rey), or it may coincide with pulmonary gangrene (Cadeac). There may be merely dulness, prostration, or stupor; or trembling, unsteady gait, or falling; there may be rolling of the eyes, or amaurosis, or vertigo occurring intermittently; or there may be epileptic attacks or paraplegia.

Course. In moderate cases the disease may last from two to three weeks, and in well conditioned horses, with strong constitutions, tends to recovery. On the third to the eighth day all the symptoms appear better, appetite, expression, alertness, breathing, pulsation and temperature. The temperature which has been a degree, or more, higher in the afternoon than in the morning, remains about the same from morning to night, or is even slightly lowered; it is lower still next morning and in two or three days may have reached 101°, still rising a little in the afternoon. The pulmonary exudate is usually quickly absorbed though less so than in favorable cases of influenza. Convalescence may be completed by the end of the third or fourth week.

In *violent* and *fatal cases* the general symptoms tend to encrease in violence, though the temperature may descend to 103°, and in the final collapse to 100° or lower. In a mare presented at the college clinic after three weeks illness and treatment elsewhere, prostration was extreme, the head rested in the manger, the nose discharged a fetid, glairy, frothy liquid, with grumous, bloody debris; breath offensive; pulse 92, almost imperceptible; respirations 30, very labored; nostrils widely dilated, flapping; temperature in vagina 103.6°, anus was open with constant ingress and egress of air, and a watery glairy, frothv discharge; extensive dropsy under the sternum; percussion and auscultation indicated consolidation of lungs from the lower border up, crepitation, creaking, and loud clucking bronchial sound. The mare survived for forty-eight hours, the temperature descending to 100.5° in the morning and rising to 102° and upward in the afternoon. At the necropsy the right lung was consolidated throughout, the left had pervious areas anteriorly and posteriorly; there

were large areas of infarction, necrosis, with encystment, caseation, and lobular and perilobular exudation and hepatization.

Liver, enlarged, tense in its capsule, but soft and friable on its cut surface, with areas of softening and necrosis. Both kidneys congested, with pale zones of necrosis; right enlarged. Beside the substernal exudate, there was extensive hæmorrhagic exudate between the serratus magnus and ribs. The blood, very dark, brightened on exposure to air.

Diagnosis: This may be based mainly on the prevalence of the disease in the district, or the fact that the victim has come from a long railway journey with risks of exposure, or has stood by a horse just arrived; on the prompt loss of appetite and sudden and extreme rise of temperature, without notable lung lesions; on the deep brownish yellow discoloration of the visible mucosæ, especially that of the eye, and on the yellow discharge from the nose, the tardiness with which successive cases follow each other comparatively to influenza, and the absence in large measure of the early extreme prostration of that affection. (See table under *influenza*).

Prognosis varies with the progress of an epizootic, and the youth and susceptibility of the animal together with the favorable or unfavorable conditions of life. The death rate is usually high at the outset when the more susceptible animals are attacked, and for the same reason, in the young that have not been previously exposed, Old and debilitated animals with broken down constitutions suffer severely, and bad hygiene contributes much to the mortality. The deaths vary from one to twenty per cent. But short of death, permanent injury follows in a number of cases. Encysted sequestra remain in the lungs for months and when liquefied and absorbed, leave fibroid masses in place of healthy lung tissue. The fibrous organization of peribronchial exudates, impairs respiration, and the same is true of the fibrous development of false membrane. Thus the horse is left permanently *broken-winded* or *short-winded*, or from interference with the recurrent laryngeal nerve, laryngeal hemiplegia (roaring) ensues. In still other cases permanent adhesions of the pericardium, or insufficiency of the cardiac valves, or disease and distortion of joints or tendons, or nervous, hepatic or renal degenerations destroy or seriously impair the value.

Treatment. Hygienic measures are of prime importance in treatment. A dry, clean box stall with pure air, and genial warmth (60°—70° F.)—in warm summer weather outdoor air—must be secured. In cold weather a sunny outlook, and clothing—blanket, bandages, and it may be a hood—to counteract any sensation of chill. Tepid drinks, pure water changed often, linseed tea, barley water, are of importance in allaying thirst, lowering temperature and favoring elimination. Absolute rest is all essential. Keeping at work after the onset of the disease is nearly equivalent to signing the patient's death warrant.

The high temperature at the outset of the disease seems to demand antipyretics, and in times past, in Southern Europe especially, bleeding was a constant resort. But even under favorable conditions this does not lower the temperature more than 1.5° F., and the resulting debility is such that it has been long discarded in Germany, England and America. In cases of acute extensive pulmonary congestion it is helpful in relieving the vascular tension on the lungs and allowing the tissues to better reassert their natural functions, in antagonism with invading microbes and their poisons, but even this action is transient, and when it saves life it is probably only by tiding over safely a very transient and urgent danger.

The same decline of temperature can usually be secured by injections of cool water into the rectum, and with the added advantage that this relaxes the bowels, and removes dried irritant masses from the rectum and floating colon.

As in other pneumonias the application of cold to the skin is liable to bring on a chill unless the temperature is very high. A safer and hardly less effective method is to apply around the chest a thin blanket or sheet wrung out of tepid water and cover it closely with dry blankets holding these close to the skin by elastic circingles. No part of the damp compress must be allowed to remain exposed to the air under pain of causing chill. Damp cotton wool applied next the skin tends to maintain its contact by its own elasticity, so that it requires less care in the elastic dry covering outside it. This moist warmth draws a free circulation of blood to the skin, so that it is cooled and sent back internally to cool the burning fever, without sensation of chill. The abstraction of this large mass of blood to the skin, acts like

bleeding in diminishing the blood-tension in the chest and allowing the resumption of the normal vaso-motor and nutritive functions without the dangers of venesection. The soothing action on the skin, soothes by sympathy the infected and inflamed tissues.

A steam or hot air bath may serve a similiar purpose. Conjoined with an aloetic laxative and aconite, I have seen this reduce the temperature from 104.5° F. to 102.7° F. at the next taking 18 hours later. It only once again reached 103° F. in the subsequent course of the disease.

To secure diaphoresis, warm mashes or gruels may be freely used. Alcoholic drinks have been freely used (the weaker wines 1 to 2 qts., sherry, brandy, whiskey ½ to 1 pint), camphorated spirit (2—3 drs.) subcutem, ipecacuan (1 oz.), tartar emetic (2 drs.), liquor of acetate of ammonia (4 ozs.), pilocarpin (2 grs. subcutem). If the alcoholic liquors produce a free circulation and glow in the skin, better still if diaphoresis, they are useful antithermics, but if they fail in this, they may do harm by reducing the vital activities of the leucocytes, and their power of resistance. Trasbot has had uniformly unfortunate results with alcohol in large doses. The same objection attaches to tartar emetic and other depressant diaphoretics, though valuable if free diaphoresis is secured.

Constipation may be met by cold water injections, calomel (½ to 1 dr.), pilocarpin (3 grs.), eserine (1½ gr.), sodium sulphate or other agent graduated to requirement.

An expectorant and alkaline diuretic action may be obtained from potassium iodine (1–2 drs.), ammonium chloride (2 drs.), or ammonium acetate. These not only liquify the exudate, and facilitate expectoration, but secure elimination of toxins, ptomaines and waste products from the blood and system. The iodide is besides somewhat antiseptic.

In the early stages especially medicinal antithermic agents may be called for: acetanilid (2–3 drs.), phenacetin (2–4 drs.), sulphate of thallin (2–3 drs.), sodium salicylate (½ oz.) or in the weaker cases, caffein—natrium salicylate (1 dr.) or quinine sulphate (2–3) drs.). Acetanilid will sometimes relieve dulness, and materially improve the general condition.

In weak conditions of the heart we may resort to digitalis (10–15 grs.), strychnia sulphate (2 grs.), strophanthus tincture (3 to

4 drs.), caffein, or alcohol. Care must be taken not to overstimulate and exhaust a weak and intermittent heart.

Derivatives are often of material value from the first, in mild cases or to succeed damp compresses in the more violent ones. One of the best and most convenient is essential oil of mustard and alcohol (1:12 or 20). This may be rubbed on the surface and like mustard itself, covered with stout paper to prevent evaporation. In the absence of this, soap liniment, or even tincture of cantharides may be used.

The use of antiseptics has been tried with variable results. While it is impracticable to saturate the system, safely, with sufficient antiseptic to destroy the microbes in the blood and tissues, yet when the balance of force between the microbian attack and systemic defence shows little variation either way, a slight increase on the side of the patient may serve to give it the preponderance, and to restrict the increase of the microbes and their products. Above all when the center of morbid activity is largely on and near the bronchial mucosa, antiseptic inhalations serve to hold them somewhat in check and to moderate the amount of both microbes and toxins that enter the system at this point. For this purpose camphor, oil of turpentine, or oil of tar volatilized from hot water may be inhaled in a close room. Or we may use carbolic acid, terpene, terpinol, creolin, lysol, thymol, eucalyptol, or oil of cinnamon. The fumes of burning sulphur diffused in the air of the room and just short of that concentration that will cause cough, suffering and headache, is an excellent resort. The sulphites, bisulphites or hyposulphites may be given by the mouth.

When there are indications of encephalitis, cold to the head and the internal exhibition of bromides, iodides, and acetanilid may be resorted to.

During convalescence, nourishing and easily digestible food may be given, and iron, strychnia, quinia and common salt may be employed. In protracted or chronic cases with fœtid breath and indications of sequestra or opened vomica in the lungs these may be continued along with one or more of the disinfectants referred to above.

Prevention. This is much more promising than in equine influenza. The extension of incubation to three days and the

indisposition of the infection to spread beyond the stable into which it has been brought, or the near vicinity of the diseased animal, gives us a great relative advantage. The early, extreme rise of temperature of the infected horses gives the opportunity of removing these horses to a separate stable or shed, where they can have special attendants, and the stable drainage and manure can be kept apart, and disinfected, or spread and plowed under by oxen. The infected stable should be emptied, the soiled hay, litter and manure burned, and the walls, partitions, floors, ceilings, and above all the mangers and racks must be thoroughly disinfected. Lime wash with chloride of lime or mercuric chloride will suffice. The gutters should be cleaned, washed and drenched with mercuric chloride, followed by the whitewash. If there is rotten wood work or filth-saturated soil these must be effectually treated. In many stables it will be impossible to do all this thoroughly, yet closing the empty building tightly, and filling it with chlorine gas, or even sulphur fumes, concentrated until they extinguish the burning sulphur, and keeping shut up for twenty-four hours will usually suffice. Washing with a solution of formalin (1:40 or 1 per cent. of formaldehyd), or even the evaporation of this agent by heat in the closed building is very effective, with the serious drawback that it is very irritating to the lungs. It can, however, be conveniently used for the sterilization of harness, stable implements, halters, and all movable objects in the building.

Strange horses, such as new purchases, should be placed in quarantine for one week in a separate stable, and not hitched up with sound horses. If they show evidence of recent illness this may be extended to six weeks.

For horses that have been shipped long distances, and stopped for rest or feeding in public stables or yards, a similar quarantine is essential. This might be obviated if a system of thorough disinfection of such stables, yards and cars, could be enforced, between any two successive lots of horses, and if the latter were accompanied by certificates of the absence of contagious pneumonia and all other infectious diseases from the localities from which they were shipped and through which they had come. Such certificates should be made by veterinary officials in the employ of the government, which would thus become responsible for their genuineness.

In view of the frequent persistence of this malady in a given stable for a great length of time, successive animals being attacked at long intervals, and where isolation was impracticable, Beckmann rubbed the nasal discharges of the sick on the nasal mucosa of the unaffected, producing the disease almost invariably in a mild form. The infected animals were placed in the best hygienic conditions, the duration of the infection was shortened, and the horses being rendered immune, the stable was then disinfected with a satisfactory result.

Schütz, Hill, Pilz and a number of others have sought artificial immunity, by the injection of blood serum from a horse that has recently recovered from the malady. The results were very contradictory. In some cases the disease came to a sudden end. In other stables no new cases appeared either in those treated with serum or in those left without treatment. In other experiments, new cases occurred among those treated with the serum ;—in Weishaupt's cases after a lapse of one or two months. This is exactly what might be expected. If the horse supplying the blood-serum had really recovered, and if the microbes (streptococci) had disappeared from the blood, the latter would of necessity retain little of the toxins, but much more of the antitoxins, the active production of which would be continued by the stimulated leucocytes. These antitoxins would neutralize the toxins, in case of invasion and prevent that from reaching the maximum of intensity that it would otherwise have reached, but would be powerless to stimulate the leucocytes of the inoculated animal into the habit of themselves producing antitoxins. This would act rather as a curative than a prophylactic agent, and its value would be spent as soon as the injected antitoxins were eliminated from the system.

The true line of inquiry would have been, whether injection of the toxins, which acting on the leucocytes would have stimulated these to the habit of producing antitoxins in large amount, might not be expected to give an immunity as lasting as that which follows on a casual attack of the disease. Lignieres appears to have approximated to this, in his experiments on mice and rabbits. In horses suffering from contagious pneumonia it lowered the temperature, but did not materially affect the result of the attack. If we adhere to Lignieres' own theory of causa-

tion by cocco-bacillus and later by streptococcus or some other complicating infection, we can scarcely hope that the toxins of the streptococcus or other complicating microbe will immunize against the cocco-bacillus or mutually against each other. If complete protection is aimed at, the toxins of his cocco-bacillus, and of Schütz's streptococcus, and of any other possible microbe which may produce a secondary complication, ought to be employed.

At the date of this writing no satisfactory sero-therapy for this disease has been worked out and publicly demonstrated.

INFECTIOUS STABLE BRONCHITIS. SCALMA.

Definition. An infectious inflammation, of the upper air passages and bronchia, attended by high fever, special nervous irritability and a protracted convalescence.

Dieckerhoff gave the name *scalma* (rogue) to outbreaks in given stables of an infection, showing the high temperature of brustseuche, (104° to 107° F.) a similar incubation (6 to 7 days), a correspondingly tardy extension from animal to animal, and duration of the disease. The apparent differences are in the absence of the profound dullness, the yellowness of the mucosæ, and the yellow or rusty nasal discharge, in the ready response to the voice or touch, the disposition to bite or kick, the spasms of the larynx and sudden dyspnœa, in certain cases, and the paroxysmal cough in others. Apart from these transient respiratory troubles the pulse and breathing are unaffected, relatively to the elevation of temperature. Sometimes the jaws are kept in constant motion, from nervousness or pharyngeal trouble.

In the absence of any conclusive bacteriological investigation, it may be surmised that this is a form of brustseuche which has not advanced to the same grade of destruction of red globules and prostration of the nerve centres, the latter showing only an excited and irritable condition.

Treatment and *prevention* do not differ materially from what is required in contagious pneumonia. The irritable cough may be soothed by inhalations of warm water vapor, with alcohol, camphor, eucalyptol, or opium, or electuaries of bromides, belladonna or stramonium, and local derivatives to the throat.

Most cases are mild and recover in a week, the cough lasting for two weeks more.

OTHER INFECTIOUS PNEUMONIAS OF THE HORSE.

Claims have been made for infections by a variety of other germs which it would be difficult or unreasonable to deny. In given conditions of the horse's lung it may succumb to the attacks of pathogenic microörganisms which at other times or under other conditions would be practically harmless. Thus the round-ended bacillus of Friedländer is claimed by Jacquot and others to cause one form of pneumonia in the horse as it does in man (Vol. I, p. 216). Again Galtier and Violet have claimed a form of pneumonia transmitted through musty or spoilt fodders and attacking the bowels as well as the lungs (pneumo-enteritis). Two micro-organisms are accused, a diplococcus and streptococcus, which is strongly suggestive of the now familiar germ of brustseuche.

EQUINE INFLUENZA. ADYNAMIC CATARRHAL FEVER OF SOLIPEDS.

Synonyms. Definition. Historic notes. Equine influenza of 1872-3; its indication of infection. Other evidence: through stables, cars, manure, clothes, coition, inoculation Bacteriology: streptococci; diplococci; coccobacillus, latter pathogenic to rodents, dog, cat, sheep, pig, ox, ass, pigeon, chicken. Inoculations on horse. Present in early stages only. Uncertainty. Accessory causes: chill, electric tension, high barometer, impure air, overwork, poor feeding, season. youth, primary susceptibility, acquired immunity. Incubation 1 to 3 days. Symptoms: Forms: sudden attack, anorexia, profound prostration, weakness, hyperthermia epiphora, brownish red conjunctiva, pulse, heart beats, catarrhal symptoms, thoracic, pulmonary, pleuritic, cardiac, digestive, urinary, diarrhœal, ophthalmic, nervous, rheumatoid; complications, abortion, laminitis, strangles, contagious pneumonia, cerebro-spinal meningitis, etc. Lesions: inflammation of mucosæ of nose and air passages with blood unaffected; in severe attacks, with heart clots, or later with blood black, diffluent, red globules crenated or dissolved, without viscidity or rouleaux, reddish serum, and hæmatoidin in masses, acid reaction, petechiæ; congestion of mouth, stomach, small intestines or large,—ulcers, tumid follicles, peritoneal effusion, enlarged congested mesenteric glands; liver as if parboiled. with petechiæ and necrosis spleen large and gorged; kidneys infiltrated, mottled, petechiated, swollen;

may be meningeal or ophthalmic congestion ; pulmonary lesions, pleural effusions, adhesions, infiltrations, consolidations, infarctions, sequestra. Diagnosis ; sudden attack, great numbers attacked, marked prostration, conjunctivitis, great hyperthermia, digestive disorder, evidence of infection. Table comparing croupous and contagious pneumonia and influenza. Prognosis. Mortality low,—high in some epizootics, in horses kept at work, under bad hygiene. Treatment : good diet and hygiene, rest, shelter, stimulating diuretics and diaphoretics, venesection, antipyretics, alkaline eliminants, inhalants—water medicated, derivatives, collyria, guarded laxatives, antiseptics, cardiac stimulants, nerve sedatives, tonics, transfusion of blood, normal salt solution,—technique. Prevention : quarantine difficult, yet possible ; examples, applicability to countries, to districts, lines of restriction.

Synonyms. Epizoötic Catarrh ; Catarrhal Fever ; Nervous Fever ; Epizoötic ; Rheumatic Catarrh ; Cocotte ; Gastro-enteric Epizoötic ; Gastro-entero nephro-hepatitis ; Gastro-Conjunctivitis ; Gastro-Hepato-meningitis ; Entero-pneumo-carditis ; Pink eye ; Epizoötic Cellulitis ; Typhose ; Typhoid Fever ; Blitz Katarrh ; LaGrippe ; Septicæmia Hæmorrhagica, etc.

Definition. An infectious fever of solipeds, of a specially low or adynamic type, and with a tendency to localization on the respiratory or gastro-intestinal mucosa, on the eyes, lungs, pleura, heart, liver, kidneys, subcutaneous connective tissue, joints, fascia, or nervous system.

This disease was long confounded with the *influenza* of man and while compelled for the identification of the affection, to retain this name in combination with the qualification *equine*, yet we would prefer to discard it entirely as conveying the idea that the illness is caused by Pfeiffer's bacillus, which it is not. The term *typhoid fever* which is in common use in France, has been selected to convey the impression of its two prominent features of *hyperthermia* and *stupor*. But it is open to the same objection for it has been long applied to a specific disease in man having its own bacillus which is not present in the *equine influenza*. *Septicæmia Hæmorrhagica* which has been adopted more lately, however correct it may be as indicating the tendency of the local lesions, has a generic meaning rather than a specific one, and requires much more qualification to correctly designate the disease. The term *adynamic catarrhal fever of solipeds* has this recommendation, that it expresses the prostration and debility which is such a marked feature of the disease, its great tendency

to become localized on mucous surfaces, and the genus of animals that prove its victims. A better designation is still desirable and may perhaps be reached, when the pathogenic microbe shall be demonstrated beyond question.

Historic Notes. Among catarrhal fevers and epizoötics of early times it is impossible to distinguish this from widespread nasal and bronchial catarrhs, and from contagious pneumonia (brustseuche), yet when the epizoötic attained a sudden and wide extension without any direct climatic cause, the presumption is in favor of the disease now before us. Hints are obtained from Titus Livius of a Sicilian equine epizoötic of this kind 412 B. C. This is corroborated by an account by Hippocrates of a similar outbreak in Greece. Later, Virgil (Georgics), Columella, Absyrtus and Vegetius give similar hints. In 1299 in Seville horses suffered with drooping head, watery eyes, beating flanks and anorexia and 1000 died (Laurentius Rusius). The horses of the French Army in Germany suffered severely in 1648 (Solleysel), horses in England in 1688 (Short, Rutty), again in 1693 (Webster, Short, Foster) and again in 1699 (Webster). In 1712 horses suffered extensively in Europe (Lancisi, Kanold) and in 1727-8 in England and Ireland (Rutty), in 1732-3)Arbuthnot, Gibson) and again in 1736-7 (Short). Other such equine epizoötics are recorded for Europe in 1729 (Löw), for Ireland in 1746 and 1750-1 (Rutty, Osmer), for Europe and the British Isles in 1760 (Bieset, Rutty, Webster), and again in 1762 (Rutty, Webster) and 1767 (Forster), also in America (Webster), in Europe in 1776 (Fothergill, etc.), in Europe and Asia in 1780-2 (Gluge), in England in 1798 (Wilkinson, White), in Europe and England in 1814-15 (Heusinger, Wilkinson, Youatt), in England in 1819, 1823 (Field) and 1827 (Brown), in Europe in 1833 (Prinz, Wilkinson, Hayes, Spooner), 1834 (Heusinger), 1835-6 (Prinz, Friedberger), 1840, 1846, 1851, 1852, 1862, 1870, 1873, 1881, 1883, 1890, 1891, 1892 (Friedberger).

In the United States as in Europe the affection has in the main smoldered in the large cities in ordinary years, to break out without obvious cause, in given years into an advancing epizoötic which sweeps the whole continent. Such were the great outbreaks in Europe in 1881 to 1883, and in America in 1872-3, and 1900 to 1901. The great recrudescence of the disease in North

America in 1872-3 was so remarkable in its progress and limitations that it seems desirable to recall its more prominent historic features. The unquestionable demonstration of the microbe of the affection may make such a record superfluous, but until then, and so long as books are published which attribute the disease to the environment, or to the soil, it is not altogether unnecessary.

PROGRESS OF EQUINE INFLUENZA OF 1872-3.

Last week of September, 1872. Toronto, *Ontario:* (30th) and neighborhood.

First 2 Weeks of October. Barrie, Collingwood, Owen Sound, Guelph, *Ont.*; St. John, *N. B.* (13th); Niagara Falls, *N. Y.*, (11th); Montreal (8), Ottawa (12th), Stratford, Brantford, London. *Can.*; Buffalo, *N. Y.*, (14th); Detroit, *Mich.*, (13th).

Third Week in October. Goodrich, Kingston, *Ont.*, (19th); Rochester (18th), Syracuse, Lockport, Canandaigua, Geneva. Albany, (19th), Ogdensburg, (21st); New York, *N. Y.*, (21st); Bangor, *Me.*; Port Huron, *Mich.*

After Third Week in October. Quebec, *Can.*, (28th); Utica, (29th), Watertown, Oswego, Schenectady, Saratoga Springs, Poughkeepsie (28th), Elmira, Binghamton, (28th), Jamestown, Ithaca, (31st), Port Jervis (29th), Nyack, *N. Y.*, (30th); Revere, (22d); Springfield, (23d), Worcester, (27th), Fall River, New Bedford, *Mass.*; Waterbury, (27th), Norwich, (23d), New Haven, Hartford, *Conn.*; Providence, (23d), Newport, *R. I.*; Burlington, (26th), St. Albans, *Vt.*; Concord, Nashua, Portsmouth, *N. H.* (23d); Bath, (28), Portland, *Me.*; Philadelphia, (26th), Harrisburg, Lancaster, York, Erie, Corry, Titusville, (28th), Pittsburg, (29th), *Pa.*; Baltimore, (25th), *Md.*; Washington, *D. C.*, (28th); Norfolk, (31st), Richmond, *Va.*; Cleveland, *O.*; Chicago, (29th) *Ill.*

First Week of November. Kingston, *N. Y.*, (1st); Rutland, *Vt.*, (3d); Meadville, (2d), Pottsville, Williamsport, (6th), Reading, Easton, Bethlehem, *Pa.*; Trenton, *N. J.*, (2d); Wilmington, *Del.*, (6th); Jackson, *Mich.*, (7th); Milwaukee, *Wis.*; Raleigh, *N. C.*; Charleston, *S. C.*, (4th).

Second Week in November. Scranton, *Pa.*, (13th); Lynchburg, *Va.*, (11th); Wheeling, (13th), Parkersburg, *W. Va.*; Dayton, Sandusky, Toledo, *O.*; Adrian, Kalamazoo, (9th),

Grand Haven, *Mich.* (8th) ; Davenport, *Ia.*, (14th) ; Janesville, Green Bay, *Wis.* ; Louisville, *Ky.*, (9th) ; Wilmington, *N. C.*, (12th) ; Columbia, *S. C.* ; Savannah, *Ga.*

Third Week in November. Steubenville, (15th), Columbus, (16th), Zanesville, (16th), Springfield, *O.* ; Indianapolis, (17th), Fort Wayne, Lafayette, Evansville, (21st), *Ind.* ; Bloomington, Galena, *Ill.*, (21st) ; Dubuque, Iowa City, *Ia.* ; Madison, Fond du Lac, *Wis.* ; St. Paul, *Minn.* ; Memphis, Nashville, Chatanooga, *Tenn.* ; Augusta, Atlanta, *Ga.* ; Helena, Fort Smith, *Ark.* ; Havana, *Cuba*, (20th).

In November after third Week. Terre Haute, Madison, *Ind.* ; Peoria, Galesburg, *Ill.* ; Keokuk, Muscatine, Des Moines, *Ia.* ; Paducah, *Ky.* ; Knoxville, *Tenn.* ; Charlotte, New Berne, *N.C.* ; Macon, Rome, Columbus, *Ga.* ; Jacksonville, Lake City, *Fla.* ; Mobile, Montgomery, Selma, *Ala.* ; Natchez, Vicksburg, *Miss.* ; New Orleans, (25th), *La.* ; Galveston, (29th), Houston, (28th), *Tex.* ; Little Rock, *Ark.*

First Week in December. Cairo, *Ill.* ; Lincoln, Omaha, Nebraska City, *Neb.* ; Yankton, (5th), Vermilion, *Dak.* ; Tallahassee, *Fla.* ; Camden, *Ark.* ; St. Louis, Kansas City, *Mo.*

Second Week in December. Quincy, *Ill.* (8th) ; Shreveport, *La.* ; Hanibal, *Mo.* ; Fort Scott, Lawrence, Leavenworth, Topeka, *Kan.*

December after the Second Week. Denver, Central City, *Col.* ; Cienfuegos, *Cuba.*

January 1873. San Antonio (3d), *Tex.* ; Cheyenne, *Wyo.* ; Cimarron, (1st week), Elizabeth City, (2d week), Alberquerque, (4th week), *N. M.* ; Salt Lake City, (2d week), Corrinne, (3d week), *Utah* ; Santiago, *Cuba.*

February. Brownsville, (3d week), *Tex.* ; Winnemucca, *Nev.* ; Monterey, (1st week), *Mex.*

March. Prescott (1st week), Tucson (2d week), Yuma (4th week), *Ariz.* ; Boise City, (3d week), *Ida.* ; Helena, (4th week), *Mont.* ; Virginia City, (1st week), *Nev.* ; Carson City, *Nev.* ; Santa Barbara, Visalia, *Cal.* ; Guaymas, Mazatlan, Manzanillo, *Mex.*

April. San Diego (1st week), Mariposa, Stockton, San José, Oakland, Sacramento, Marysville and Shasta (2d weed), San Francisco, Vallejo, Nevada City and Weaverville (3d week), and

Lava Beds, *Cal.* (4th week); Baker City (2d week), Jacksonville, *Ore.* (4th week); Walla Walla, *Wash.* (4th week); Acapulco (1st week), *Mex.*

May. Eugene City, (3d week), Dallas and Portland, *Ore.* (4th week); Olympia, *Wash.*

June. Seattle, *Wash.* (3d week).

July. Guatemala City, Guatemala (2d week), Victoria, *B. C.*

August. La Union, *San Salvador* (1st).

Among the deductions from this record are:

1. The affection advanced gradually from Toronto over the whole continent of North America, where horses are kept, taking full ten months to accomplish this. Nothing checked its advance, over lowland and highland, swamp and arid land, in summer as in winter, with a temperature at 0° or at 100° F., in country pasture or in city street or stable, idle or overworked, on all kinds of soils and geological formations, under all successive conditions of meteoric and terrestrial electricity, in all conditions of the air—pure, impure, dense, light, moist and dry. No one condition of the environment operating on the animal system, can be conceived of, that could advance as this disease did from place to place in regular sequence for this length of time.

2d. The rapidity of its progress was manifestly subordinate to the activity of the movement of the equine races from points already infected. Its most rapid advance was along the lines of railway while the back districts shut out from railway traffic were much later in being invaded. The larger cities situated on the through railroad routes suffered earlier than the smaller places on the same lines. The outbreak was several days earlier in Montreal than in the nearer and smaller cities of Kingston, Ottawa, Belleville, Port Hope, Peterboro, Stratford, Brantford, Guelph, London and Owen Sound. The important port of St. John, N. B., suffered two weeks earlier than Quebec. Along the N. Y. Central and Erie Railways, etc., Boston and New York suffered nearly a week earlier than Utica, Poughkeepsie, Binghampton, Elmira, and Jamestown, while the smaller places like Kingston, Nyack, Ithaca, etc., were later still. In Baltimore the disease was seen a day earlier than in Philadelphia, and in these cities and Washington over a fortnight before it was seen in Scranton, Pa. So it was almost everywhere and in these large

cities the outbreak could in nearly every case be traced to horses just arrived from a pre-existing centre of infection. In Detroit, Syracuse and Chicago it spread first in stables that had just received Canadian horses, in Ithaca in one which had received horses from an infected centre in Northern New York and in Pittsburg and Washington in stables that had just admitted horses from infected New York.

3d. It advanced with much greater rapidity eastward than westward, being in the line of greatest horse traffic, the animals being mainly raised and fitted in the West and shipped in large numbers to the great cities near the Atlantic seaboard.

4th. In the absence of this active railway traffic in horses, the advance was most rapid through other lines. In Pa., in a number of valleys opening to the south, the disease reversed its general direction, and extended northward up these valleys. In Lehigh Co., Pa., it followed the course of the canal, being carried by horses and mules employed on the towpath. In Davidson and Sumner Cos., Tenn., it followed the track of a circus which came through an infected locality. It reached the Pacific coast at Santa Barbara (not at the railway terminus at San Francisco) having followed a mule stage route in the absence of an active, westward progress of horses by rail.

5th. The affection failed to overstep any serious gap over which there was no movement of equine animals. It prevailed in Victoria, B. C., in July, but, owing to a strict quarantine on horses and mules, it failed to reach Vancouver Island. It ravaged New Brunswick and Nova Scotia in November but failed to reach Prince Edward Island which was then ice bound and shut off from all traffic with the mainland. It ravaged Cuba to which it was brought by American horses landed at Havana, but no other West Indian island was attacked. Its southward course was finally arrested at Central America, where horses are few and horse traffic nearly unknown.

Every fact in connection with its eruption and progress agrees perfectly with the hypothesis of transmission by contagion alone, and taken altogether the history excludes all other causes from being anything more than accessory. Before the days of modern bacteriology we had ample proof that glanders, rabies, sheep-pox, lung plague, and Rinderpest were due to contagion alone as an

essential cause, and so now we have the same evidence concerning equine influenza.

Other testimonies to Contagion. Trasbot says the virulence is "almost equal to that of Rinderpest or aphthous fever," and adds "all practitioners have become assured that the bringing of an affected animal into a stable constantly introduces the malady to the others." Cadeac says "the diseased or infected animals are the main channel of propagation of the malady" and again the disease is "essentially infectious." Friedberger and Fröhner are more definite—"influenza which is as highly infectious as any other disease can be produced only by infection." Cadeac implies nearly as much in saying: "in all the epizoötics that have invaded Paris, the disease has been carried into the four quarters of France by horses bought in this city. In most regiments the malady shows itself after the arrival of horses from remounts where it was prevailing. At Sibourne it is through horses from St. Jean d'Angely. At Lyons it is by a horse from Cæn. At Bourges the source was not traced but it spread from the garrison to the whole surrounding country. The Omnibus stables in the Rue d'Ulm were invaded when a horse was introduced from Clichy where influenza raged."

Stables are fruitful sources of infection hence dealers' horses and horses travelling from place to place have long been objects of just suspicion (Trasbot, etc.).

Cars are often infected, and spread the disease widely. (Poucet, Salle, Trasbot, etc.).

Manure is especially dangerous. Trasbot gives a number of cases of the infection of farms, by the manure taken from the Alfort Veterinary College, and other infected stables in Paris. Friedberger conveyed the disease experimentally in the manure.

The conveyance of the virus on the *clothes of attendants* has been alleged by Friedberger and Fröhner, and considering that it has been noted to pass over intervals of about half a mile without the intervention of any horse, it must have been wafted on the air, or conveyed on the surface of man or non-equine beast.

Jensen and Clark allege that the contagium may be conveyed to mares by *coition, for months* after the stallion has shown all outward signs of recovery. This would be entirely in keeping with the analogous fact in swine plague.

Inoculation. Experimental inoculations have transmitted the disease with difficulty and uncertainty. Those of Hertwig, Nocard, Arloing, Labat, Friedberger, Trasbot, Pasteur and others came to naught. Even the transfusion of the blood of the sick, proved as harmless as the inoculation of the serous exudate. A probable explanation is found in the extreme diffusibility of the germ of equine influenza, which spreads over a city or county in a few days, attacking practically all equine animals. Inoculation is necessarily made at the time of the prevalence of influenza and at such a time all horses in a wide area are likely to be suffering from the affection. Those that are unaffected and therefore apparently available for experiment, are the immune animals. If they were susceptible the probability is that they would speedily show the disease through infection drawn from another source than the inoculation. If the inoculated animal failed to contract the disease, and yet very shortly afterward became infected by simple exposure, there would be some basis for alleging that inoculation was always inoperative. Dieckerhoff, on the other hand, transmitted the disease to healthy horses by subcutaneous and intravenous inoculation of the blood of the sick, and the same seems to be true of inoculations of the cultures of the cocco-bacillus by Lignieres.

Bacteriology. Our knowledge of the bacteria of equine influenza is as yet very imperfect and uncertain. Galtier and Violet found streptococci and diplococci in the blood and tissues of cases showing intestinal lesions, and held that they were derived from musty fodder. Injections of infusions of such fodder into the trachea of the horse produced broncho-pneumonia, double pleuro-pneumonia, and at times intestinal or meningeal congestion. But there is no proof that the malady so caused, passed with the certainty and rapidity of equine influenza from horse to horse in the same stable.

The *cocco-bacillus* found by Lignieres in the blood and exudate of the patients has more plausible claims to being the specific germ. This is an ovoid bacterium, somewhat smaller than that of chicken cholera, and like it pigmented at the poles and clear in the central part, a characteristic feature of the group of Pasteurella of Trevisan. This group includes the non-motile germs of swine plague, the septicæmic pneumo-enteritis of sheep, wilde-

seuche, and septicæmia of rabbits and chickens, as well as that of fowl cholera; all stain easily in gentian violet and fuchsin, and all cause some form of hæmorrhagic septicæmia. The germ is aerobic and grows best in peptonized bouillon to which a little serum has been added. It forms, in peptonized gelatin, round colonies, at first transparent and later opaque or milky, and without liquefaction. The cultures when inoculated subcutaneously proved fatal to Guinea pig, rabbit, rat, mouse, dog, cat, sheep, pig, ox, ass, pigeon and chicken.

Intravenous inoculation on the horse of 1 to 2 cc. of the culture kills in a few hours, the temperature having risen to 104° F., the mucosæ acquire a dull brown tint, the eyes are swollen and weeping, enteritic colics appear, the limbs may swell and there may be painful arthritis and jaundice. At the necropsy the blood is black and incoagulable, the muscles as if parboiled, the liver a deep violet, the intestinal mucosa congested, a yellowish or reddish effusion in the pericardium and numerous petechiæ on the serosæ.

Subcutaneous inoculation causes an enormous inflammatory œdema resulting in a sanguinolent abscess, in case the subject survives. There are also hyperthermia (106° F.), dullness, stupor, weakness, staggering, and congested, swollen, weeping eyes.

Intratracheal injection is harmless to the horse.

Lignieres finds his cocco-bacillus in the expectoration at the outset and in the nasal and guttural forms of the disease later, but not in the blood nor lungs after death, as it is then replaced by streptococci, the great reproduction of which is favored by its presence. In ordinary cases of equine influenza it is often impossible to find the cocco-bacillus in the lung or other organs after an illness of 8, 10 or 15 days. (Lignieres).

Lignieres appears to have omitted the obvious test of the infection of other horses in the same stable, from the cases produced by his experimental cultures, so that we must still call for more confirmatory proof. Cadeac, indeed, assures us that cultures of cocco-bacilli taken from cases of equine influenza, are often innocuous. Deadly as the germ cultures of Lignieres prove, they appear to lack that element of extreme infectiousness shown by equine influenza when the susceptible animals come into proximity with the sick.

Accessory Causes. The recognition of the one essential cause in the microbian invasion, need not exclude as accessory factors the many unwholesome conditions which have long been recognized as contributing to the severity of epizootics. As the seed requires the rich field, the rain and sunshine to bring it to an abundant harvest so the microbe of equine influenza flourishes best where the conditions are most favorable and the antagonisms least.

The *chill* which comes from a sudden extreme fall of temperature, or the standing in a cold draught when wet or perspiring, lays the system open to this as to other microbian invasions.

The *electric tension* preceding a thunderstorm, to which many of the lower animals are excessively susceptible equally prepares the system to succumb to the germs. It may here be noted that September, 1872, the last days of which witnessed the start of the great epizootic, had no less than eleven thunderstorms, while in September of the previous year there were but two in the vicinity of Toronto. It is just possible that the great and frequent electric tension, lowered the animal vitality, allowing a violent invasion by the hitherto slumbering germ, and gave to the latter that encreased potency which sent it forth on that year of almost unparalleled epizoötic record.

The *high barometer* and *low dew point* similarly affect the animal economy and encrease receptivity to disease. Rain fell at Toronto 16 days in September, 1872, and but 8 days in September, 1871.

Impurities in the air whether originating in volcanic eruptions, telluric emanations, close, filthy overcrowded buildings or compartments or large collections of decomposing organic matter, impair the animal vigor and lay the system open to a more violent attack. For this among other reasons epizoötics of equine influenza are nearly always more deadly in the closely packed city stables than in the pure country air.

Overwork and *poor irregular feeding* and *watering* pave the way for debility, prostration and severe invasion.

Sudden vicissitudes of temperature, which are so common in *spring* and *autumn*, associated as they are with the *shedding and growth of the coat*, materially encrease susceptibility and sometimes determine an encreased severity in the attack.

Youth has its influence, even if it means only that the system that has never before been exposed to the poison, retains all its native susceptibility, and has none of that acquired immunity which comes from a previous exposure to the virus and successful resistance.

Acquired immunity must of course be reckoned with. After a non-fatal attack this is usually to be relied on for several years or even for the rest of the lifetime, yet it varies with the individual animals, and, under the baleful combination of a specially potent germ and strongly conducive accessory causes, it may become worn out in a year. Yet the older horses can always be trusted to show a large measure of this immunity, so that in the absence of extraordinary epizoötics it is mainly the young that suffer, and it is only when a country has had no general invasion for a length of time, or when the germ has acquired an unusual pathogenic potency, or when these two conditions conjoin, that the invasion of the equine population becomes universal, as it virtually was in the United States and Canada in 1872–3. Under other circumstances the germ, temporarily shorn of its power, lingers in city and dealers stables, biding its time until circumstances become more favorable for a new general outbreak.

Immunity largely explains the comparative mildness of the last cases in any particular locality. The more susceptible animals are attacked first and most severely, while the partially immune ones, which for a time resist, throw off the disease with greater readiness. The explanation has been sought in a lessening potency of the germ, but though this may hold true of some cases, it manifestly does not apply when slight lingering cases only are left in one locality, and the disease is advancing over the neighboring state with all its original force and vigor.

Incubation. This appears to vary within certain limits. When during an epizoötic a sick horse is brought into a new locality and stable, other cases usually develop in from one to three days. Trasbot gives examples of one day, Salle, Cadeac and others of two, others claim four, seven and even, exceptionally, fifteen days. One reason for an apparently prolonged incubation may be found in the seclusion of the germs in the alimentary canal, so that they escape only when passed with the fæces. The pathogenic potency of individual germs, and the varying susceptibility of the animals exposed must also be taken into account.

Symptoms. Equine influenza is liable to show a special predilection for a given set of organs in different epizootics, so that we find descriptions of the different forms as independent types or even separate diseases: as the *catarrhal form*, *thoracic form*, *abdominal form*, *bilious form*, *nervous form*, *pink eye*, *infectious cellulitis*, and *rheumatic influenza*. These forms may, however, appear in different subjects in the same epizootic, and when they are not due to complications, may be looked on as a concentration of the morbid processes on one class of organs rather than another.

Initial pathognomonic symptoms. Certain prominent and striking symptoms are so constantly present in the earlier part of the disease that they may be held as virtually diagnostic. These are the *suddenness of attack*, the *anorexia*, the *profound early prostration and weakness*, the *high temperature*, the *swelling and watering of the eyes*, and the specially *brownish red coloration of the conjunctiva and other visible mucosæ*. The attack may come on with almost lightning rapidity. The animal which yesterday, or it may be but an hour or two ago, appeared to be in the most vigorous health and spirits, is found with pendant head, resting perhaps on the manger, ears drooping, eyelids swollen and half closed, epiphora, conjunctiva of a brownish red or violet, lips loose and drooping, and one or two legs partially flexed, while the body is balanced on the others. The patient is indisposed to move, and when compelled to walk may sway and stagger from nervous and muscular weakness. The arched back, cracking limbs, and their stiff, rigid movement further indicate the suffering in muscles or joints or both. Appetite is greatly impaired or lost, thirst marked, and hyperthermia 102° to 105° or upward. Sneezing, cough or symptoms of some other special localization may be present, but the above occurring in a number of horses at once, without appreciable climatic cause, when one or two new horses have been very recently acquired, or when influenza has been prevailing in the vicinity or in a neighboring place, will usually stamp the nature of the attack.

Cadeac considers the sudden attack, high fever, and profound nervous prostration and stupor as the manifestations of the uncomplicated disease, while the localizations in the lungs, bronchia, pleura, liver, bowels, etc., are indications of complications by

germs of other diseases, which find the debilitated influenza system especially open to attack. The *fever* which always sets in early may be little above the normal in mild cases, and may reach 107° or 108° F. in the more severe ones. It may last thus for five or six days and then rather suddenly descend to near the normal. In other cases it descends a little daily, the lowest temperature for the day being seen in the morning. Shivering is often non existent or passes unperceived.

The *pulse* does not usually encrease in ratio with the temperature. It may be at first only 40 or 50 per minute, though later, and especially with extensive disease of important organs, it may reach 60, 70, 80 or even 100. It usually lacks in firmness and force, even when the heart beats forcibly, being soft, somewhat compressible, and often irregular in successive beats, the weakest corresponding to the last part of the inspiratory act, or when the lungs are full and the heart compressed. The *heart* impulse behind the left elbow is usually forcible and may show variation in rhythm or even intermissions.

Mild *catarrhal symptoms of the nose and throat* are usually present, the discharge being at first serous and later muco-purulent. As a rule this is complicated with more or less bronchitis, but this does not indicate anything serious. Acceleration of the breathing, sneezing, and cough are present. Cough may be at first nervous, husky and paroxysmal, but later as the discharge is established it assumes a looser, mucous character. It is liable to be roused by excitement, by drinking cold water, by inhalation of dust, or by giving medicine. In connection with these symptoms there are some indications that the digestive organs are involved. The pharyngeal and sub-maxillary glands may be swollen and tender. If the subject has been seized just after a full meal, there may be slight tympany, and in any case, the fæces are passed in small balls, a few at a time, hard and with a baked or glistening surface. These may have an unusually strong or heavy odor, and laxatives are liable to act with dangerous energy. The urine is scanty and high colored, sometimes icteric.

In such mild attacks, which constitute the majority, improvement may be noted as early as the fourth day, and a prompt recovery follows.

With extensive *thoracic lesions*, the symptoms are much more severe and the danger greatly enhanced. These may occur in any patient, but there appears to be a special predisposition in the young and still very susceptible animals, in those crowded together in close, badly aired buildings, in the over-worked, poorly fed or in any way debilitated subject, and in horses that have been especially excited and exposed, as by railway travel.

In exceptional cases *congestion of the lungs* may be so acute as to lead to speedy death, and the objective symptoms do not differ greatly from those of ordinary cases of this condition, if we except the very high temperature in influenza, associated as it is with the fact of the epizoötic prevalence of the disease.

In *pneumonic* cases the lesions are usually double and have a tendency to develop toward the lower borders of the lungs, just behind the elbow or farther back, and less frequently in the centre of the organ. It may be impossible to detect crepitation, but sounds of distant organs (heart-beats, bronchial blowing, intestinal rumbling) are heard with unwonted clearness over the consolidated parts. A mucous râle can usually be detected behind the shoulder blade, along the line of the larger bronchia. Percussion sounds may be indefinite, as the area of consolidated lung is usually small in ratio with the hyperthermia. The area of flatness in ordinary fibrinous pneumonia is usually much greater with a high fever, and if the lesions are on one side only, right or left, it is still more suggestive. The crepitation too in pneumonia is significant. When the pulmonary lesions are extensive by reason of œdema, a marked infiltration may often be noted on the lower surface of the trunk or in the limbs as well.

Pleuritic symptoms may show in the same connection. The breathing becomes more hurried and shorter, friction sound may be heard but it is very transient and soon superseded by an absolute flatness on percussion, rising to a definite horizontal line, representing the boundary of the effusion in the lower third or half of the chest, and usually rising to the same height on both sides. Tenderness of the intercostal spaces may or may not be present. As the disease advances creaking sounds may be heard from the stretching of the consolidated false membranes. The combination of double pleuro-pneumonia constitutes a very fatal type of the disease.

The symptoms of *pericarditis* and of cardiac disorder usually

accompany those of pleurisy. The tumultuous heart-beats, often associated with soft, weak or even rapid pulse, and later, a deadening or muffling of heart sounds, as in hydropericardium are characteristic when present. With *endocarditis* the early tumultuous heart-beats, with small weak pulse, irregular and sometimes intermittent, become complicated by a blowing or hissing murmur with the first heart-sound. In such cases clots of blood are liable to form in connection with the valves, and may cause sudden and early death. When the heart is involved the tendency to extensive infiltration of limbs and lower aspect of the trunk is much enhanced. (See *diagnosis* for table of phenomena in influenza, fibrinous pneumonia and contagious pneumonia respectively).

Symptoms of digestive disorder are usually in evidence. Even in the thoracic forms the mouth is dry, hot, and has an offensive odor; the tongue coated above, has often red margins and tip; it may even be yellowish; the gums may be swollen and dark red or violet especially around the incisors; mastication may be slow and unwilling; the pharynx may be swollen, the pharyngeal and submaxillary lymph glands may be tumid and tender, and swallowing may be difficult.

Congestions of the stomach and intestines are indicated by inappetence, sometimes flatulence, passage of flatus, constipation with small, round, mucous-coated balls passed in small numbers, and by slight transient colics, pawing, looking at the flanks, and retraction of the abdomen. The retention of bile and destruction of blood elements are indicated in a deeper yellow of the conjunctiva and visible mucosæ, and in a yellow, brown or red color of the urine. There may be tenderness of the abdomen, but this, like the colics, is moderate, the senses being blunted by the attendant stupor which is usually even greater than in the thoracic forms. Urination may become frequent with straining, and the urine may become turbid, opaque, with flocculi of cystic epithelium and mucus, and even albumen. In from three to five days diarrhœa supervenes, the fæces becoming soft, pulpy, watery, glairy or bloody, and escaping through a permanently dilated sphincter. The diarrhœa may alternate with periods of torpor or complete inactivity, otherwise tenesmus of the rectum is marked. The exposed rectal mucosa is congested, of a deep red or it may be of a dark violet hue. Eversion is not unknown.

In the worst cases death may ensue by the third or fourth day, but in others the diarrhœa is critical and heralds an improvement which goes on to a speedy recovery. In still other cases the bowel troubles continue, the fever does not give way and the privation of food and rapid metamorphosis of tissue produce steady emaciation and fatal marasmus.

A striking feature of the gastro-intestinal disease is the extraordinary susceptibility to laxatives. So much is this the case that I have known of two drachms of aloes proving fatal by superpurgation in a large, mature Percheron horse. It is never safe to use laxatives in equine influenza until one has ascertained whether in the special form of the epizoötic in question the gastro-intestinal organs are or are not especially involved.

Disorders of the eye are so common or constant as to have procured for certain epizoötics the name of *pink-eye*. They set in suddenly, and equally in both eyes, with infiltration of the lids and particularly of the mucosa which is of a more or less deep red, and may bulge between the margins of the eyelids, (chemosis). The flow of tears is profuse, seropurulent matter accumulates at the canthi and in the lachrymal sacs, vision is impaired and there is intolerance of light. The cornea becomes bluish, cloudy or milky white, with a red zone around its margin and, above all, on the adjacent sclerotic. In some cases the aqueous humor becomes turbid or flocculent, and the iris changes its clear, healthy dark lustre for a dull brown or yellow tint. The tension of the globe may be materially encreased. They are readily distinguished from recurrent ophthalmia by the attendant weakness, stupor and hyperthermia, and by their non-recurrence in case the patient survives.

The *nervous symptoms* are especially manifest in the sudden seizure, great prostration, extreme weakness, profound stupor or lassitude, the staggering gait, in bad cases, insensibility to voice, slap or, it may be, even to the whip, the rigidity of the loins, their insensibility to pinching, the difficulty of turning in a short circle, or of backing. The high fever, disproportionate to the appreciable local lesions, and its sudden improvement at the critical period, the excessive weariness and the disposition to lie down contrary to the habit of other inflammatory chest diseases are further indications.

This may go on to coma, there may be more or less complete anorexia, muscular trembling, paresis, especially of the hind limbs, or delirious manifestations indicating meningitis.

Rheumatoid attacks of the muscles and joints usually appear in the advanced stages of the disease, but may appear earlier. They may occur in any latitude but seem to be especially common in cold, damp, inclement northern regions, and at seasons when climatic vicissitudes are sudden and extreme. These may appear suddenly and disappear with equal rapidity, or they may last for a time during and even after an apparent recovery in other respects. When the joints are involved they usually become engorged with exudates in the synovial membranes.

Lameness in either *fore* or *hind limb* may assume an *intermittent type* developed by exercise and subsiding with rest, evidently bespeaking local arterial embolism, and in such cases it is likely to persist for months.

A tendency to *transudations* and *dropsical effusions* is common in severe cases, showing especially in the filling of the legs, but in certain epizootics, these become strikingly prevalent and have secured for such a special name (*epizoötic cellulitis*). Apart from the limbs these affect particularly the inferior surface of the chest and abdomen. The swellings are not necessarily hot nor painful nor petechiated as in petechial fever, yet they may merge into that affection or they may become phlegmonous and develop abscess.

Complications of many kinds are to be looked for, pregnant mares may *abort; laminitis* may set in; the microbes of *strangles*, *contagious pneumonia*, *cerebro-spinal meningitis*, *septicæmia*, *pyæmia*, etc., may take occasion to attack the debilitated system, and thus complex diseases and manifestations are developed.

Morbid Anatomy. The lesions may predominate in different organs in different subjects and successive epizoötics. It is a protean disease and may expend its main energy on any one of a number of different organs or systems of organs.

In the slighter cases the lesions are often largely confined to the anterior part of the respiratory organs. The *fauces*, *pharynx*, *larynx*, *guttural pouches* and *nasal mucosa* are tumefied, congested, red and covered with mucus, and this condition may extend down to the bronchia. In such cases the *blood* is normal or may

coagulate with undue readiness and firmness. The pharyngeal and intermaxillary lymph glands are red and congested in their outer zone.

In the more severe cases the *alterations in the blood* are perhaps the most constant of the morbid features. The blood is fluid and incoagulable, or the clot is soft, diffluent and black, the red globules are crenated or broken up, and show little tendency to adhere in rouleaux. The escaped hæmatoidin accumulates in masses in the serum in crystalline forms, giving it a high staining power when a line is drawn with it on white paper. Fatty globules also float in the mass. The leucocytes are relatively very much encreased and the red globules diminished Dieckerhoff found 30,000 and Trasbot 40,000 leucocytes in a cubic millimetre.

The diffluence is not constant. Blood drawn in the earlier stages of the disease, coagulates with extraordinary firmness, influenced, doubtless, by the encrease of the leucocytes, the disintegration of the blood globules, and the liberation of globulins. This serves also to partially explain the early and sudden deaths from coagula in the heart and large vessels, which are occasionally met with. Such clots in the heart are often found adherent to the valvular or ventricular endocardium which at such points shows cloudy swelling, thickening, cell proliferation and even encreased vascularity and granular elevations.

In advanced cases, however, the prominent features are usually acidity, blackness, and incoagulability of the blood, its resistance to oxygen, altered and broken down red globules, free coloring matter, relative encrease of white cells, and, if necropsy has been delayed, the abundance of septic microbes (cocci and bacilli). Petechiæ are abundant on the serosæ especially on the pericardium.

Lesions of the alimentary mucosa are very constant. There may be stomatitis, with tumid follicles and even ulcers (Kowalavsky). In the *stomach* the *right sac* has its mucosa thickened, softened, red, congested, petechiated and discolored. The summits of the folds may be ulcerated (Labat). Similar lesions are presented in the *small intestines*. The agminated glands may show many rounded elevations, with or without open discharging follicles. The mucosa is covered with a mucopurulent material. Otherwise, the small intestines, like the stomach, are usually empty.

The *large intestines* present similar lesions, the nodular elevations often representing the solitary glands, and the masses of ingesta are likely to be dry and indurated, in the earlier stages or semi-liquid in old standing cases. The *peritoneum* may be congested, petechiated and at points infiltrated and usually contains a reddish serum in variable quantity. The *mesenteric glands* are more or less enlarged and congested.

The *liver* shows more or less congestion as in other infectious diseases localized in the bowels. It usually has a parboiled appearance, and yellowish gray areas of necrosis may be manifest, or again, fatty degeneration may be present. Petechiæ, and even small blood clots may be found on or beneath the capsule. The *pancreas*, and, still more, the *spleen* may be the seat of congestion or engorgement but this is far from constant.

The capsule of the *kidney* may be petechiated or elevated at points by serous exudate or extravasation. The surface of the organ and of sections show a mottling with darker and lighter areas, and petechiæ and patches of congestion may be found on the bladder and urethra.

The *nervous centres* exceptionally show meningeal congestions, and exudations, and petechiæ as has been noted of other serosæ.

Lesions of the *eye* may be confined to the mucosa, or they may extend to the membrane of the aqueous humor, the iris, or even the deeper structures.

Other lesions such as *laminitis*, *bursitis*, *arthritis*, like those attendant on *abortion* need no special description.

When *pulmonary lesions* are extensive, the bronchial mucosa is not only softened, opaque and covered with a serous, or mucopurulent discharge, but deeply congested and petechiated. When the chest is opened there is usually an effusion, pale straw, red or bloody and more abundant than in contagious pneumonia. False membranes may exist and show a blackish tint from extravasated blood. The lung fails to collapse and shows on the surface and throughout its substance petechiæ and small black infarctions. In some instances the whole lung is blood gorged, black, almost jelly like, as in acute congestion. In others these are limited infiltrations, concentrated especially in the anterior and lower parts, and almost invariably affecting both right and left lungs. The infiltration is circumscribed in area in compari-

son with the attendant fever and constitutional disturbance, resembling in this respect, the lesions of contagious pneumonia. It differs however in having a greater tendency to liquid infiltration of the connective tissue, and but for the lack of such tissues in the horse's lung it would tend to approximate to the lesions of the lung plague in cattle. It shows a distinct thickening of the interlobular septa, a tendency to extension to the pleura, and to issue in pleural and sub-pleural infiltration, and to a more copious effusion into the pleural cavity than in either fibrinous or contagious pneumonia. The lung tissue may be granular and hepatized, but far more frequently it is only splenized, the lung being the seat of a bloody infiltration, yet retaining much of its elasticity and coherence. Portions may be infarcted and black and large areas may have a pale or par-boiled appearance, and gangrene is by no means uncommon.

Diagnosis. This is based largely on the suddenness of the attack, its epizootic character, the numbers attacked in rapid succession, and over a large area as contrasted with contagious pneumonia, the sudden and extreme prostration and weakness, the swelling, watering and discoloration of the eyes, the mildness of the average case, the congestion of the upper air passages, and in the mild cases a comparative immunity of the lungs, the irritability or congestion of the gastro-intestinal mucosa, and the history of the case :—the arrival of the infected horses within a few days from an infected place or coming through infected channels, or the attack of new arrivals in a previously infected stable, or the known advance of the disease towards the place where the patients are, will usually serve to mark the true nature of the affection.

As a help to correct diagnosis we give below some of the prominent conditions and phenomena of the three forms of lung disease known as *fibrinous pneumonia*, *contagious pneumonia*, and the *pneumonia of equine influenza* :

Pneumonia: *Croupous Fibrinous.*	**Pneumonia:** *Contagious, of Equine Animals.*	**Pneumonia:** *of Equine Influenza.*
From climatic vicissitude, exposure, etc. Attacks exposed animals only, and all at once	Slow succession of cases in the same stable, irrespective of climate or exposure	Rapid succession of cases in the same stable or locality, irrespective of climate or exposure
Prevails in inclement seasons, spring, autumn, (Winter).	Any season: worse in inclement season	Any season: worse in inclement season
	Infection from close proximity, contact, stall, manger, rack, bucket, trough, etc.: Spread slow	Infection spreads widely and rapidly through the air. Spread rapid and often general
	Carried in manure, on harness, wagons, clothes, etc.	Carried in manure, on harness, wagons, clothes, etc.
	Incubation 3–10 days	Incubation, 1–2 days
Rigor may be late: after exudation has commenced	Rigor early and well marked, before exudation	Rigor not always well marked
Sets in slowly or with acute congestion; appetite and pulse vary with inflammation	Sets in slowly, cough, dulness, impaired appetite, pulse rapid, prostration slight	Profound nervous prostration like opium poisoning, appearing early and suddenly
Nasal discharge, watery or rusty, later mucopurulent	Nasal discharge, yellow.	Nasal discharge, watery, may become yellow
Eye pink, dark red	Eye yellow, rarely swollen or watery	Eyelids bloodshot (pink eye), violet, madder hue, swollen, watery, closed
Temperature rises with inflammation	Temperature rises early and extremely, before exudation, 104°–107°.	Temperature rises early and extremely, in some hours 104°–107°.
Swelling of limbs rare	Limbs swell rarely	Limbs often swell greatly
	Rheumatoid arthritis may follow	Rheumatoid arthritis may set in; often in advanced stage
Crepitation more constant around the exudation	Crepitation less constant around the exudation	Crepitation may escape recognition
Exudation (Hepatisation) in lung, lower, posterior, anterior, or central, usually undivided area	Exudate affects lower or anterior border, of lung; often in small isolated areas or around bronchia; less blood engorgement than in fibrinous pneumonia	Exudate less granular than in pneumonia; forms in or gravitates to lower part of lung. Congestion passive.
If pleurisy effusion may be copious	Pleural effusion infrequent, or limited, though pleurisy is common	Pleural effusion frequent and abundant

Pneumonia: *Croupous Fibrinous.*	**Pneumonia:** *Contagious, of Equine Animals.*	**Pneumonia:** *of Equine Influenza.*
Abscess not infrequent.	Abscess rare.	Abscess not infrequent.
Pulmonary gangrene infrequent.	Pulmonary gangrene and sequestra frequent.	Infarctions, sequestra, and cavities not uncommon.
Pericarditis infrequent.	Pericarditis frequent.	Pericarditis frequent.
Blood, decrease of red globules; encrease of white, hæmatoblasts, fibrine formers and soda salts.	Less altered than in either of the other forms. Shows coccobacilli in the earlier stages: streptococci later.	Blood at first clots firmly, later becomes thick, black, sizy, hæmatosis tardy. Reaction acid.
Coagulation firm, buffy coat.	.	Coagulum loose, buffy coat slight.
Hepatic congestion, not hepatitis.	Hepatic congestion excessive, hepatitis, necrotic changes.	Hepatic congestion excessive, hæmorrhagic; fatty degeneration.

Prognosis. Mortality. As usually met with and under favorable conditions, equine influenza is a mild disease. In 1872 when the disease, sweeping the continent and hardly sparing an equine animal, might be assumed to have reached its maximum, the actual deaths varied from 2 per cent. in country districts to 7 per cent. in large cities. The same holds for Europe where Friedberger and Fröhner gives 4 to 5 per cent. Früs (Denmark) 1 per cent., Aureggio (Italy) 3 per cent., Siedamgrotzky 10 per cent. Much depends on conditions: In horses infected in transit on a long railroad journey it may be 100 per cent. and in fat dealer's horses, out of condition for active work it usually reaches a high figure. If the patients are kept at work the complications and mortality run very high. The same applies to debilitated animals kept in close, foul, ill-aired stables, or reduced by exhausting or long standing diseases. The very young and the senile suffer more than animals in middle life and vigorous condition. Finally the parts invaded have a controlling effect. The milder cases affecting the upper air passages only, nearly all recover, in those showing abdominal lesions the indications are still favorable; while with double pulmonary and pleural lesions the patient is too often in a hopeless condition. Brain lesions are almost equally redoubtable.

The actual money losses, in an epizoötic of influenza, are more in the way of the loss of work and the complete stagnation of

trade in all departments, than in the number of deaths. Yet even in this sense it may prove more ruinous than would a disease having a less universal sway though far more fatal to the animals attacked.

Treatment. A disease like this, which tends to spontaneous and perfect recovery, needs mainly dietetic and hygienic care in the vast majority of cases. Rest is a prime consideration in a pure genial atmosphere. In the summer season, in the absence of rainstorms, an open air life, at pasture is the best. Shelter must be had in case of storms, and in the cold season a clean, sweet, roomy, well-aired, loose box, with a sunny exposure, is important. Clothing, bandages, blankets and even hoods may be required if there is any tendency to chill. Food should be laxative, cooling and of easy digestion. Bran mashes, scalded oats or barley, ensilage, roots, potatoes, apples, fresh grass or scalded hay may be suggested. Milk has been strongly advocated, from twelve to fifteen quarts a day, and linseed tea as being especially adapted to the irritable stomach and bowels. Food should be given often, in small quantities so as not to destroy the appetite.

Costiveness is best met by injections of water, blood warm, or if there is much hyperthermia, of cold water. This latter stimulates the peristalsis more actively and at the same time lowers the temperature. By unloading the large intestines it removes irritants, without danger of encrease of congestion or diarrhœa.

If anything more is wanted for these mild cases, small doses of stimulating diuretics or diaphoretics may be given. Sweet spirits of nitre ½ oz. or liquor of acetate of ammonia 2 ozs. may be given twice a day, the latter in the drinking water or gruel. In the absence of these, saltpeter ½ oz, or potassium acetate ½ oz. may be given.

In the more *severe cases* more active treatment is resorted to, but in all cases one should avoid measures that tend to greatly depress the vital powers and especially the circulation. Heroic treatment has been all but universally condemned and yet Trasbot claims to have had excellent results from a moderate venesection (2-5 qts.) in strong, muscular, well conditioned animals. The blood became more fibrinous, the general symptoms improved and complications were far less marked. During twenty years, in a large number of cases, it had constantly the

same results, restoring appetite, opening the closed eye, imparting new life, lowering temperature, and checking congestions. The results were best in all cases when it was employed early, but they were almost equally good when later congestions set in, or when the ordinary inflammatory localisations were advancing, and he found the measure hurtful only when, in the advanced stages the animal was worn out, and destitute of all power of recuperation.

Antipyretics have been lauded and decried, and it is doubtless best to use them with due discrimination and caution. Acetanilid, the agent in most common use, is a powerful cardiac depressant, and in this disease the heart is often already dangerously weak. Yet in cases with very high temperature, seriously threatening life, acetanilid in doses of 2 drams, repeated every second or third hour until the temperature falls, and thereafter twice or thrice daily, for a day or two, may save the patient. Similarly, where there are indications of violent headache (drooping head, eyelids and ears, congested and watery conjunctiva, stupor or irresponsiveness) a dose or two of acetanilid with the same amount of sodium bromide will often give material relief. The general suffering and disorder attendant on the cephalalgia, if allowed to continue, prove a direct bar to improvement, and endanger complications that might otherwise be escaped. On the other hand, there is constant danger from too large doses, or a too long continued use of acetanilid in influenza. Phenacetin may be substituted in 2 dram doses, the action being somewhat more prompt and transient. As less dangerous than the coal tar antipyretics, we may fall back on such agents as sodium salicylate, ½ to 1 oz., or sulphate of quinia, 20 grs.

Elimination of toxins and waste products is to be secured and this is more safely conducted by the kidneys or skin than by the irritable alimentary canal. Plenty of pure cold water is one of the best and simplest resorts. Aside from this, where there is much hyperthermia, bicarbonate of potash or soda will serve the various purposes of an antidote to the acid blood, an eliminant and an antithermic. Under the same circumstances saltpeter may be resorted to in ½ oz. doses twice daily. When, however, the heart is weak it is better to employ ammoniacal or etherial diuretics :—spirits of nitrous ether 1 oz., or liquor of acetate of ammonia, 2 to 4 ozs. By combining these with extract of belladonna and camphor a fairly standard prescription may be prepared.

Inhalations of warm water vapor, rising from hot water or a hot mash in a bucket, over which a bottomless bag is drawn, while the other end receives the nose of the horse, will greatly relieve the irritation and the cough. It can be made even more soothing by introducing a little alcohol, eucalyptol, menthol, camphor, poppy-heads, or other anodyne. Or the water vapor may be set free in a close stall from a boiling tea-kettle or a steam pipe, and an admixture of sulphurous acid made by burning a few pinches of sulphur, more or less according to the size and closeness of the stall. If the stall is tight enough a steam bath may be given with much profit in the early stages.

Counter-irritants applied to the throat, or, if need be, to the breast and sides of the chest will often give material relief, acting as derivatives and probably also by modifying the globulins in the exudate and thus influencing the course of the disease. In case of sore throat of a very high type it may be best to apply a compress or poultice, or even a piece of sheepskin for a day or two, until, by antithermics, cooling diuretics and soothing inhalations, the severity of the inflammation has abated. The common mustard pulp made with tepid water, rubbed in, and covered by paper, may be applied for an hour ; or the *soap liniment* (soap 6 ozs., camphor 3 ozs., and proof spirit, liquor ammonia and linseed oil *aa* 1 pint) may be rubbed on repeatedly ; or a blister of cantharides may be used.

The *ophthalmia* may be treated by a moist atmosphere, a few drops twice daily of a solution of atropine beneath the lids, a similar treatment with pyoktanin, (1 : 1000), or a solution of mercuric chloride (1 : 2000).

In case of *gastro-intestinal inflammation* elimination is to be sought by ⅓d the usual laxative dose, supplemented if need be, by injections. Half a pint of olive oil with 20 grs. calomel will usually be well borne. Counter irritants may be applied to the abdomen, and the bowels must be carefully watched and any inactivity or derangement corrected. A slight diarrhœa is not to be too hastily checked as it may serve at once to eliminate offensive matters and subdue mucous inflammation. Solutions of flax seed, gum, or slippery elm in the drinking water may serve a good end.

In case of *cardiac weakness* with intermittent or irregular pulse, hurried breathing, and an undue contrast between the vio-

lence of the heart action and the weakness of the pulse, circulatory stimulants are called for. Digitalis 10 grains twice daily, strophanthus tincture 1½ dr., strophanthin subcutem ¼ grain, caffeine 5 grains, veratrine $\frac{1}{10}$th grain, or strychnia 2 grains.

Nervous symptoms will exceptionally demand the application of cold water or even ice or snow to the head, with counter-irritants to the sides of the neck or chest, and the internal use of bromides, iodides, chloral or other nerve sedative.

Other complications must be treated according to their indications.

As the skin becomes cooler and more moist, and the pulse slower and fuller, a tonic and stimulating treatment may be desirable. Gentian 4 drs., saltpeter 4 drs., sal-ammoniac 2 drs. may be given night and morning, or in case of great debility ammonia carbonate may replace the sal-ammoniac. Or the gentian may be replaced by nux vomica, and the ammoniacal preparations by alcoholic ones.

When prostration becomes extreme and stimulants and bitters appear inadequate, transfusion of blood from a healthy horse may save the patient's life, or a normal salt solution sterilized may be introduced into the vein.

The first method is accomplished through a caouchouc tube with a short tube of silver inserted in each end; the jugular groove of each horse is washed and disinfected, and the vein opened; the tube disinfected with a salicylic acid solution, and cleared out with boiled water is inserted upward into the vein of the sound horse, and when the blood begins to flow the other end is inserted downward into the vein of the sick one. In this way the blood is allowed to flow from the one to the other, the finger being kept on the pulse of the patient to detect any faltering, which like heaving up of the head or rolling of the eyes, may be taken to indicate undue arterial tension, or disturbed brain circulation, and should be the signal for an arrest of the flow. To effect this, pinch the tube in the centre, and wait a minute or so; if the symptoms subside the current may be reopened and a little more carefully admitted, but if not the tube may be withdrawn and the wounds pinned up.

The normal salt solution, .6 per cent., is sterilized by boiling, and placed in a sterilized vessel where it is allowed to cool to the

body temperature ; then a caouchouc tube furnished with a silver tube at one end and sterilized as for transfusion of blood, is filled with the solution and the vessel containing the latter having been placed at a level higher than the patient, the tube is used as a syphon. When the liquid flows in full stream the silver tube is inserted downward into the jugular of the patient and the liquid is allowed to flow in, subject to the same precautions as regards sudden blood tension as in the case of the transfusion of blood.

Prevention. No country appears to have attempted the absolute exclusion of the disease or the extinction of the germ by a compulsory quarantine and disinfection. The nearest approach to this is in Prussia where in the event of an outbreak of equine influenza, the official veterinarians and police authorities must send in reports to be published in the official papers and communicated to the directors of government breeding studs and to the army authorities. We have here the germ of an effective system of extinction, for if those in charge of government horses in an infected country can protect them against infection, much more could such protection be secured by putting an end to the infection which is now allowed to remain generalized. Moreover in our great outbreak of 1872-3, when germ-potency and all but universal susceptibility were so remarkable, effective quarantine showed the most signal successses, in the resulting immunity of Vancouver's Island, Prince Edward Island, the whole of the West Indies except Cuba, Central and South America, and isolated districts in Mexico.

Sanitary police in this disease has been abandoned mainly because the virus is so diffusive on the air that quarantine must be more than usually comprehensive to prevent extension of infection, and because the disease is fatal only in a small percentage of cases, so that the loss is apparently minimized. But a panzoötic like that of 1872-3, prostrating 1,000,000 horses, asses and mules in the United States for one to two weeks, and paralyzing the agriculture and commerce of the continent for that length of time, may well make one hesitate to supinely accept for all time an evil, which, experience has shown, can be circumscribed and stamped out. When horse owners, legislatures and veterinarians can be educated up to the needs of the case, our yearly local losses from equine influenza, and the occasional all

pervading epizoötics of this disease can probably be abolished by the extinction of the germ on the Continent. To achieve this result an immediate large outlay would be well spent. In a new generation, or a new century perhaps, this desirable object may be achieved.

Meanwhile the individual owner can do something to secure a partial protection. On farms and in barracks the animals may be secluded from all other equine animals during the local prevalence of the disease. Men, dogs, and wild animals can be similarly excluded. Litter, fodder, bags, clothing, manure, vehicles, etc., from infected stables and places must be carefully guarded against. Newly purchased animals, carried in any public conveyance, or kept or fed in any public yard or stable must be quarantined at a considerable distance from others, and treated by disinfectant sponging and fumigation before they are allowed to mingle with other equine animals. Stables where the disease has occurred must be thoroughly disinfected, together with all manure made during the epizoötic and for some time thereafter.

EPIZOOTIC CELLULITIS: PINK EYE.

Williams follows a popular fashion in describing under the above names an affection which may be only a form of equine influenza, but which may be named by itself until its true place can be defined by proof of its actual pathogenic microörganism. Beside the general constitutional disturbance, this condition is distinguished by marked hyperthermia (103° to 104°), swollen, congested watering eyelids, cough, strong pulse becoming gradually feeble, firmly coagulating blood, irritable bowels, painful passage of fæces, and, above all, a frequent movement of the feet indicative of discomfort and followed by swelling, often excessive, of the limb or limbs, and by a cutaneous and subcutaneous exudate. These various phenomena may all be but manifestations of the rather protean disease, equine influenza, and the cellulitis but variations of the rheumatoid and arthritic forms which are so common in the regular type of that disease in cold or wet climates, or seasons. Williams claims that a prominent danger is

the formation of clots in the heart and large vessels and advocates the free use of the salts of ammonia and potash with stimulants. The treatment does not essentially differ from that of equine influenza except in the call for special applications to the inflamed eyes and infiltrated limbs.

PETECHIAL FEVER. ACUTE HÆMORRHAGIC—ANASARCOUS—TOXÆMIA.

Synonyms. Definition. Causes, obscure, bacteria variable, pus microbes, no active contagion, toxic products, any toxin causing vaso-dilatation, examples, toxins from fermenting ingesta, debility; impaired innervation, nutrition and function; gravitation; primary and secondary forms: predisposing diseases. Lesions: petechiæ and slight blood extravasations in skin, subcutis, mucosa, submucosa, serosæ, and solid tissues, largest in soft tissues; round cutaneous swellings one to two inches across, patches, cracks, oozing, fissures, sloughs, section shows yellow serous and blood infiltrations, capil laries greatly dilated, infiltrated thickened mucosæ, blocked nasal passages, ulcers, serous and bloody discharges, pharynx, larynx, lungs, stomach, intestines, kidneys, blood firm or diffluent. Symptoms: Hyperthermia, nasal petechiæ, extravasation, pink or yellow oozing, swellings on nose, lips, face, limbs, oozing, cracks, sloughs, turning up of toe from detachment of the flexors, metastasis from skin to lungs, or abdomen, dyspnœa, colics, serous or bloody diarrhœa. Course. Duration: acute two days, average one to three weeks, tardy one to two months. Diagnosis: from glanders, anthrax, urticaria, malignant œdema, horse pox. Mortality. Prognosis: 50 per cent.: hopeless and grave indications. Sudden retrocession. Treatment: excellent sanitary conditions, cleanliness, air, light, green food or mashes, pure water, laxative food, or salts, diuretics, alkaline diuretics, vaso-motor contractors, antiseptics, quinine, strychnine, phenic acid, lysol, ichthyol, sulphites, etc.; locally aluminium acetate, cold water, scarification, nasal injections and tubes, tracheal iodine injections, argentum colloidale intravenously, antistreptococcic serum subcutem, Menveux's solution.

Synonyms. Purpura Hæmorrhagica; Morbus Maculosus; Anasarca (Fr.); Typhus: Anthrax; Dropsy of Connective Tissue; Mal de Tete de Contagion; Coryza Gangrenosa; Malleus Gangrenosa; Carbon Blanc; Diastashemia; Leucophlegmasia.

Definition. An acute (or subacute), toxæmic, generally secondary disease, manifested by capillary dilatation and petechiæ on the mucosæ, skin, serosæ and elsewhere, and attended by extensive effusions of lymph and blood into the skin and connective

tissue, to form hot, tender, nodular, diffused, or general swellings.

Causes. The causes of petechial fever are not clearly made out. Bacteria are found in the early exudate, but these are not constant in kind and it is not unreasonable to suspect that these may be results rather than causes of the lesions. On the other hand there may be an as yet undiscovered organism present, the products of which are capable of producing the disorder. Or the latter may be due to a combination of the toxins of two or more. Among the bacteria found may be named: the pus microbes, cocci, streptococci, staphylococci, and bacilli, have been found and adduced as causes, also diplococci, the bacillus hæmorrhagicus of Kolb, the streptococcus of strangles, the microörganism of contagious pneumonia and that of influenza, with a variety of others too great and too inconstant to be accepted as proof of cause. Frasey injected str. pyogenes in two horses and produced the recognized symptoms of the disease. The fact that rarely more than one animal suffers in the same stable at the same time might be held to oppose the idea of contagion and of a definite organized germ.

Hence the theory of its causation by the presence in the system of the toxic products of bacteria rather than the bacteria themselves. (Dieckerhoff.) Cadeac supposes that any toxin which causes capillary dilatation may determine the disease, and calls attention to the fact that the injection of mallein (a vaso-dilator) aggravates the phenomena of petechial fever and determines enormous local exudations and engorgements. He notes further the potent vaso-dilator action of the products of strangles, contagious pneumonia, and influenza which are among the most frequent antecedents of petechial fever. Dickerhoff also looks on the phenomena as the result of poisoning by the absorbed toxins of the microbes of suppuration, which modify the nutritive changes in the walls of the capillaries and determine exudations and hæmorrhages. Zschokke thinks that there is infection of the intima of the capillaries, with the formation of coagula. Clots are not found, however, apart from hæmorrhages.

The toxin theory receives indirect support from the absence of the same specific lesions in simple mechanical congestion of the capillaries and veins. Ranvier had no such results from tying the veins of a rabbit's ear. Roger tied the auricular veins of the

rabbit, and then cut the sensory nerves without effect: he then destroyed the cervical sympathetic ganglion, when there supervened marked exudation, lasting for three days. The complete blocking of veins by pressure or aseptic ligature, does not produce a spreading œdema, whereas in ordinary suppurative phlebitis, with abundance of toxins in the tissues this is a constant result.

The toxin theory does not fully account for those cases that occur suddenly, without any manifest pre-existing disease, and as the result apparently of cold and chill. On the other hand, it is only a very small proportion of horses exposed to the same degree of cold and chill that contract petechial fever, and it might well be surmised that in these few an unknown focus of suppuration or other lesion existed prior to the chill or that toxins having the requisite devitalizing and vaso-dilating properties had been absorbed from fermentations in the bowels or elsewhere. The mere exposure is harmless to the very great majority of subjects.

In any case it must be accepted that the debility and impaired local innervation, nutrition and function, that attend on the exposure to cold and toxins are to be looked on as potent contributing causes. The predilection of the swellings for dependent parts (limbs, venter, face) shows the influence of gravitation and congestion. Whether there is present any special microbe which has yet eluded discovery, but which is the main pathogenic factor, must be left to the future to decide.

In cases that appear to be due to cold or chill alone, the disease is held to be *primary*; in those following on another affection, *secondary*.

Among the diseases on which petechial fever supervenes as a secondary affection, contagious, inflammatory disorders of the lungs and air passages hold a bad preëminence. Strangles, influenza and contagious pneumonia, about in the order named, are especially causative factors or occasions of petechial fever. Among the other affections on which it supervenes may be named pharyngitis, abscess of the nasal sinuses, hepatic, renal and other internal abscesses, acute coryza, laryngitis, or bronchitis, enteritis, abortion, aggravated grease, suppurating wounds of the skin, abrasions by harness, suppurating sores after firing, infective arthritis with open joint, amputation of the tail, and castration.

Lesions. In certain cases these may be largely confined to petechiæ and slight blood extravasations, which are distributed

very generally throughout the tissues, but show especially in the skin, subcutis, mucosa and submucosa of the nose, eyes, pharynx, guttural pouches, larynx, trachea, bronchia, mouth, stomach, intestine, bladder, vagina and womb; also in and on the lungs, pleura, pericardium, heart, liver, spleen, kidneys, peritoneum, pancreas, ovaries, bones, lymph glands, brain and nerves. The largest extravasations are liable to be in the softest tissues, and in the lungs they may reach the size of the closed fist, though usually they vary from a mere spot up to this. The spleen is sometimes engorged even to rupture. Beside the extravasations, and associated with them in position, and probably largely as an effect of them, there is more or less serous effusion infiltrating the tissues, congestions, suppurations, degenerations, and necrotic changes.

The *skin*, if white, and the dark skins on section, are seen to be marked by petechiæ. The cutaneous swellings may appear on any part, commencing with nodular thickenings varying in size from a pea to a walnut, and merging together into extensive elevated areas terminating abruptly at their margins in the smooth skin. The larger and more persistent engorgements settle on the lower aspect of the body and other dependent parts like the limbs and face. Cracks, oozing, deep fissures, and extensive sloughs are not uncommon. When the skin is incised it shows serous infiltration and thickening, with spots and patches of blood extravasation. The subcutaneous connective tissue is similarly infiltrated and discolored, and often in the limbs, face, and under the breast, sternum and abdomen so as to form a tremulous gelatinoid cushion of several inches in thickness. The capillaries may be distended to more than 20 times their normal calibre. The exudate may extend deeply between the muscles, and sloughs may lay these freely open and invade their substance. The muscular tissue is mottled with petechiæ, and apart from these it is pale, yellow, or grayish, having to some extent undergone granular or fatty degeneration. Detachment of the perforans and perforatus from their insertions is occasionally met with.

The *nasal mucosa* may show only petechiæ and circumscribed blood staining, but in fatal cases it is more likely to present extensive blood extravasations involving it may be the whole

mucosa, and narrowing the lumen almost to complete occlusion. Sloughing is not rare, and the resulting ulcers may extend into the subjacent tissues, so as to penetrate the septum nasi or the thin plate of the turbinated bone.

The *buccal mucosa* and *sub-mucosa* are often involved in common with the skin of the lips, cheeks, and intermaxillary space, the tissues being involved in one common infiltration of blood and serum. In some cases circumscribed necrosis and ulcerations are formed.

In the *pharynx* and *larynx* infiltration of the mucosa and adjacent parts of a deep blood red, with or without ulceration, causes serious narrowing of the passage, that on the vocal cords threatening suffocation. Suppuration of the pharyngeal glands and guttural pouches is not uncommon. Alimentary matters are frequently found in the larynx, and bronchia.

Beside the petechiæ and hæmorrhages in the *lungs*, œdematous infiltration in dependent parts, hepatization abscess, and limited areas of necrosis are met with. The pleural sacs often contain a sanguineous effusion.

The *stomach* and *intestines* are usually more or less mottled with petechiæ involving mucosa, serosa or muscular coat ; they are raised in rounded or irregular elevations by œdemas ; or they are the seats of more or less extensive and even perforating ulcers. The contents of the bowels may be deeply discolored by the escaping blood.

The *kidneys* may be pale except where blood stained, and œdematous infiltration of the surrounding tissue may be marked. Serous effusion into the peritoneum is not rare.

The *eyelids* are often implicated, infiltrated, thickened, and rigid, and the conjunctiva, bulbar and palpebral, the seat of extensive petechiæ.

Barreau mentions extravasations on the divisions of the lumbo-sacral plexus causing sudden paraplegia.

Petechiæ and hæmorrhages mark endocardium, pericardium and cardiac muscle, otherwise the muscle is pale.. The blood is sometimes in firm clot, at others diffluent or nearly incoagulable.

Symptoms. If hyperthermia is not already present as a feature of the pre-existing malady it usually shows itself early, at first slight, it may be (101° F.), and afterward rising in some cases to

104° to 106° F., or even higher. The general symptoms are usually those of the pre-existing disease (strangles, contagious pneumonia, influenza, nasal catarrh, pharyngitis, bronchitis, etc.) pursuing, it may be, a persistent course, or attended by special toxæmic symptoms of prostration and other signs of depression of vital functions. In some cases the hyperthermia is either absent at this stage or overlooked. On the prostration supervene the petechiæ on the visible mucosæ, and often also the swellings of the skin and subcutis. One of these may be seen before the other and it is difficult to decide whether the petechiæ always appear first as has been claimed. Cadeac claims that when œdema is first seen it has been preceded by petechiæ in that tissue (skin).

The *petechiæ* are usually first noticed on the nasal mucosa as fine red points, pin's heads, or up to half an inch in diameter, or a number of these have coalesced to form extensive patches, and by and by to cover the entire wall. At first the mucosa is spotted with purple, without any marked elevation of the surface, but as the lesions extend it becomes swollen and raised at the points of extravasation and immediately around them and oözes a serous, sometimes a pinkish or yellow fluid. Even in the smallest petechiæ the color is persistent and does not disappear on pressure like the blush of the adjacent mucosa.

Usually *cutaneous swellings* coincide with the petechiæ, or appear within two days thereafter. The first manifestation is in the form of rounded abruptly elevated nodules, about 1½ inch to 3 inches in diameter, strongly resembling the eruption of urticaria. These show a certain predilection, for the more dependent parts of the body,—limbs, abdomen, sheath, mammæ, sternal or pectoral region, nose, lips, face, etc.,—but they may develop on any part or on the whole surface. Neither tenderness nor heat is usually excessive. The swellings tend to run together so as to form extended elevations enveloping the entire limb up to a given point, forming a great pad under the chest and abdomen, or distending the whole face or head so that it seems more like that of a hippopotamus than of a horse. In such cases the lips and nostrils become so thick and rigid that prehension is impossible, and breathing if it can be accomplished at all is accompanied with a marked snuffling. The swollen eyelids are closed, and the

general turgid surface of the face is hard and resistant and no longer pits on pressure.

Under the *chest* and *abdomen* the swellings show as a continuous pad or cushion, on one side mainly, or extending across continuously on both sides, and from the breast to between the thighs. It usually pits on pressure, and may shed the hair and become rough and scabby or oöze a serous fluid from the surface.

On the limbs the swelling usually shows first on the fetlock or pastern and gradually extends upward until it reaches the body,

As the disease advances chaps, cracks and fissures tend to form on the swellings, showing about the head, on the lips or on the nose and maxilla where the noseband of the halter crosses ; on the lower part of the body where the circingle crosses, or where the part is pressed upon in lying down, and in the limbs in the flexure of the joints—behind the pastern, or knee or in front of the hock. In many cases the skin and connective tissue slough, and drop off exposing the muscles, the tendons or the ligaments of the joints. In other cases the tendons are involved in the degenerative process or necrosis and become detached from their lower insertions so that the toe may be turned up or the fetlock pad may come to the ground. The matrix of the hoof wall (coronary band) may separate from the horn, leaving a gaping opening which exudes liquid freely, and if the animal survives, the entire hoof may be shed.

In other cases, and often quite early in the disease, the swellings may suddenly subside and disappear, with it may be, a recovery, or, in other cases, with an exudation into the lungs or chest, the digestive organs or abdomen. In case the lungs are attacked, there is hurried oppressed breathing merging into dyspnœa or asphyxia. In case the bowels suffer there are colicy pains more or less acute, with much constitutional disturbance, marked prostration and serous or bloody diarrhœa. These mostly prove speedily fatal. Less redoubtable are those cases in which the swellings alternately subside or moderate, and reappear or increase, without implication of the internal organs. The absence of internal lesions and the moderation and intermissions of the external ones, give good hope of the preservation of the vitality of the tissues and of recovery.

Course and Duration. These vary much with the severity of the case. In subacute and tardy cases with few petechiæ and restricted swelling in the limbs, the symptoms become remittent and recovery finally takes place after one or two months. In other cases the morbid phenomena which developed rapidly may subside as quickly and recovery occurs in a few days. In the more typical case the visible lesions may encrease or remain stationary for one, two or three weeks and then terminate in death or recovery. In the most violent types death may occur within forty-eight hours. The average duration of the affection is found to be about 16 days.

Differential Diagnosis. In typical cases of petechial fever, diagnosis is easy. The supervention on a protracted or debilitating disease of the respiratory passages of an access of hyperthermia, and marked prostration, with the appearance on the nasal or other mucosa of petechiæ and swellings of a dark red color throughout, and of cutaneous swellings in the form of nodular elevations and more extended salient patches, having a tendency to oöze blood or serum, to crack and fissure, is virtually pathognomonic.

Acute glanders may resemble it but lacks the extended sloughs of petechial fever, and the nasal ulcers that form in glanders are on a yellowish base and periphery, whereas the purpura ulcer is on an uniformly dark red base, and without the elevated margin seen in glanders. In cases of doubt the mallein test is not available as the purpuric patient is already fevered, or liable to be so at any moment, and any wound in such a subject will give rise to extensive swelling.

In glanders the nodular submaxillary enlargement is almost pathognomonic and still more so if the facial lymphatic vessels are thickened (corded) and both symptoms fail in purpura. In cutaneous glanders with swollen joints or limbs the attendant pain is much more severe, and the farcy buds, forming on the thickened and indurated lymph vessels, bursting and discharging an albuminoid fluid like oil have no counterpart in petechial fever. In cases of doubt the search for the glander bacillus, and above all the inoculation of a male Guinea pig in the flank and the discovery of the bacillus mallei in the resulting exudate and diseased testicle will decide.

From *anthrax* and *emphysematous anthrax* petechial fever is distinguished by the absence of the large bacilli of these respective diseases from the exudate. It is not communicable, like anthrax, to the sheep, Guinea pig and rabbit, and does not crackle on manipulation, like emphysematous anthrax. The swellings are much more generally diffused than in anthrax and the hyperthermia much less.

Urticaria furnishes a skin eruption which may be indistinguishable from the earlier skin lesions of petechial fever, but these lesions are not associated with the petechiæ in the nasal and other mucosæ, and the swellings do not advance to great sanguineous engorgements, cracks, fissures, necrosis and deep and extensive sores as in purpura. Urticaria is, moreover, usually traceable to some digestive disturbance and fault in feeding.

Malignant œdema is usually confined to the seat of the inoculation wound and an extension around that, the exudate is very watery and may be mixed with fetid gas bubbles, so as to crepitate slightly, and it contains an abundance of its specific, round ended bacillus, often in chain form. The carcass putrefies with great rapidity.

Horse pox affecting the pastern and limb with attendant swelling is distinguished by the absence of petechiæ on the mucosæ, and by the formation on the affected part of little pea-like papules, which early exude an abundant liquid, the concretion of which on the hairs forms a remarkable yellowish encrustation, embedded in the angry red sores beneath.

Mortality. Prognosis. The mortality has generally averaged about 50 per cent. Much, however, depends on the violence of the attack, and the reduced and worn out condition of the patient. The most hopeful cases are those in which the temperature remains near the normal, the strength and appetite are well sustained, the swellings are comparatively slight, and there is no indication of any internal complication. The unpromising symptoms are : persistent high temperature ; complete anorexia ; great dulness and prostration ; excessive swellings not only cutaneous but in the nose and throat as well ; a marked oozing from the swellings with a tendency to form cracks, fissures and sloughs ; the serious obstruction of breathing and prevention of hæmatosis by blocking of the nose, pharynx or larynx by sanguineous and

serous exudate ; the appearance of oppressed breathing and other indications of exudate with the lungs and chest, or of colics, diarrhœa, and other suggestions of effusion on the bowels or in the abdomen. Extreme fetor of the expired air and of the fæces is a bad symptom, though not always a fatal one. Sudden retrocession of the cutaneous swellings, may be the precursor of internal exudations and death, yet in the absence of marked acceleration of the pulse, and of the objective symptoms of disease of the chest or abdomen, it is rather to be taken as a herald of recovery.

Treatment. Whatever may be the precise cause of petechial fever it is largely connected with and maintained by an unhealthy condition of the blood, and especially with the presence of toxins and waste products in that liquid. The first consideration is to secure for the patient the best possible sanitary conditions. A roomy loose box, dry, clean, well lighted and well aired, nourishing, easily digested food—green food, carrots, turnips, or mashes—and pure water are desiderata. A sunny exposure is desirable especially in winter, and everything like chill should be guarded against. Blanketing may be called for in cold weather, but the circingle like the halter should be avoided as being calculated to cause indentation, cracking or sloughing of the swellings.

A moderate action of the bowels should be secured by the nature of the food (linseed meal or tea), or by small doses of saline laxatives (sodium sulphate), or calomel. Suppression of the urine too, must be counteracted by diuretics (saltpeter, oil of turpentine) when necessary.

Further internal medication has been aimed to correct the dilatation of the capillaries, and to prove antidotal to, or to eliminate the poisons present in the blood.

As *vaso-motor* stimulants have been employed ergot, belladonna, tannic, sulphuric and hydrochloric acids, oil of turpentine, iron sulphate, potassium bichromate and chlorate, quinia sulphate, and strychnia sulphate and arsenate. The value of any one of these is dependent on its early employment, the slight character of the lesions and the remissions that so often occur even in severe attacks. None are of much account in a violent attack at its worst.

In the slighter cases *ergotine*, 5 grains daily in two doses, has been apparently useful. An objection is that its continued action on the nerve centres and digestive organs is liable to prove depressing and injurious. Of the acids, *tannic* is liable to engender constipation, locking up the injurious products to which, however, it acts to some extent as an antiseptic. *Sulphuric* and *hydrochloric* acids have a tonic effect, and the latter is a stomachic under ordinary conditions. The same may be said of the *iron salts* and to some extent of *oil of turpentine*, which have both proved useful in favorable cases. Cadeac condemns ol. terebrinth as calculated to abolish kidney secretions. The *potash salts*, *bichromate* and *chlorate*, and *quinia sulphate* are decided antiseptics and though the admissible dose would not ensure the destruction of bacteria, yet, acting in the system, with leucocytes and leucomaines, they may serve by keeping them in check. The *chlorate of potash* is given to the extent of an ounce the first day, and of half an ounce on succeeding days. The *quinine* salt is given in half ounce doses once or twice daily. In combination with bitters they seem to be of material value. *Nux vomica* (1 dram) or *strychnia sulphate* or *arsenate* (2 grains) given twice daily has seemed to be among the most promising agents of this class. As a potent vaso-motor stimulant, a bitter tonic and stomachic, strychnia has seemed at times to rouse the vitality and enable the system to throw off the load of poison that depresses it. The wonderful power of *adrenalin chloride* as a vaso-motor stimulant more than warrants its use both locally and generally. In using any one of these agents, we should not neglect concurrent attention to the bowels and kidneys, to antisepsis internally and externally, to hygienic and tonic measures generally.

Antiseptic agents have been given by the alimentary canal, the skin, the subcutis, and the air passages.

By the *stomach* the following have proved more or less useful in checking gastro-intestinal fermentations, and perhaps in hindering absorption of toxins from the specific lesions on this track: *phenic acid*, *creolin*, *lysol*, *ichthyol*, *boric acid*, *salicylic acid*, *sodium salicylate* and *hyposulphite*, and *calomel*.

To the *superficial swellings*, *fissures* and *sores* the same agents may be freely applied, alone, or combined with astringents, such as *alum*, *lead acetate*, *aluminum acetate*. In the hot season they

may be applied cold, whereas in the cold weather hot applications are often preferable. Aruch claims excellent results from covering the engorged head with a woolen cloth and irrigating it with cold water.

The question of drainage of the worst cutaneous swellings by *scarification*, has been variously dealt with, Dieckerhoff advocating deep incisions, while Friedberger and others forbid them as encreasing the tendency to necrotic infection and sloughing. In the slighter cases it can well be omitted, but in extensive swellings the disintegrating and debilitating action on the tissues is so great as to well warrant its prevention by incisions and drainage It is well to first give the surface a soapy wash, then apply a mercuric chloride or carbolic acid lotion and lance it in the prominent and tense portions. As a further precaution against septic infection it may then be covered by cotton soaked in one of the above lotions and covered with a bandage. For swellings beneath the abdomen or chest this may be held in place by loose circingles. Scarifying becomes imperative in case of paraphymosis arresting the flow of urine. Deep fissures and sores following sloughing may be treated with mercuric chloride solution, (1:1000), creolin or phenic acid (2:100), iodoform, dermatin, naphthalin or salol.

Disinfection of the *nasal passages* has been attempted by pervading the air with carbolic acid, creolin, cresyl, lysol, or naphthalin, by hanging saturated cloths in the stall, or spreading them on the floor. Perhaps a better method is to flush out the nose, by injecting each chamber in turn with a piece of rubber tubing, one end of which is introduced into the nose, and the other raised four feet and furnished with a funnel. The injection may be 1 dram creolin to 2 quarts water, or carbolic acid or alum may be substituted or alternated.

The swelling of the nostrils may be so great that it becomes requisite to hold them open mechanically. The suture of the two together, across above the nose has been practised, or a tube of light wood or aluminum can be inserted in each nostril.

Injection of the trachea and bronchia with a solution of iodine 1 part, potassium iodide 5 parts, and water 100 or 200 parts, was advocated by Dieckerhoff and met with a fair measure of success in mild cases. In some cases, however, granular tracheitis and

bronchitis followed, and in others necrotic tracheitis and pulmonary gangrene, so that the method has not been widely accepted. If resorted to, the weaker solution is to be preferred, and may be injected through a tracheal ring, or the membrane between two rings twice a day in doses of 3 to 5 centigrammes (45 to 75 drops).

Injection into the veins of Argentum Colloidale, 1 per cent solution in doses of 1⅔ oz. (50 to 60 grammes) at intervals of 2 hours has been very strongly advocated by Dieckerhoff who in four successive cases had complete recoveries in from three to six days. After disinfection of the skin a small trochar and cannula, or a hypodermic needle is introduced into the jugular and the injection made. Local treatment for the swellings was also applied.

Injection subcutem of the *antistreptococcic serum* of Marmoreck has been lauded as promptly overcoming the capillary dilatation, and enabling the leucocytes to neutralize the toxins. Extensive experience resulted in many failures, apparently often dependent on the fact that the streptococcus to be antagonized is different from the one used to make the antidotal serum. This introduces an element of great uncertainty in any individual case. The identity of a given streptococcus is not to be determined even by its agglutinins. Besredka thinks it can be by the action of the immune body (fixator, amboceptor), but even if proved, intelligent treatment must be deferred until the laboratory decision can be made, and perhaps even until the immune body can be secured from cultures in an animal body. Until a satisfactory investigation has been made, the anti-streptococcic serum is virtually a step in the dark, which may be a brilliant success, or an equally remarkable failure.

Finally Menveux recommends an artificial compound as possessing all the good qualities of the antistreptococcic serum. This is composed of sodium chloride 5 grammes, sodium phosphate 1 gramme, sodium sulphate 20 centigrammes, sodium carbonate 1 gramme, caffein chlorhydrate 10 grammes, boiled filtered water 100 grammes. Inject daily in three doses 135 grammes. Cheron's serum contains the same agents with ½ grm. carbolic acid added. Mainet advises 200 grms. of salicylic acid solution (1:100) subcutem.

PETECHIAL FEVER IN CATTLE.

Synonyms. Causes: microbes, maturity, hard work, chill, poor hygiene, drying of damp lands in summer and autumn, impermeable soils, corrupt water, bacillus, diplococcus; resemblance to hæmorrhagic septicæmia. Lesions: petechiæ and hæmorrhages on mucosæ, serosæ and skin; gelatinoid exudates gravitating to the lowest parts of face, chest, belly and legs; chaps, oozing, sloughs, depilation, exudate in tongue, bloody diarrhœa, duration 14 to 40 days, mortality light; indurations. Diagnosis: from anthrax, black quarter, malignant œdema and acute hæmorrhagic septicæmia. Prevention: avoid infecting soils in summer and autumn; drainage, cultivation; chills. Treatment: venesection: laxatives, diuretics: intratracheal injections of iodine: colloidal silver. Locally: antiseptic astringents: remove eschars: facilitate breathing by surgical or mechanical means: good hygiene.

Synonyms. Purpura Hæmorrhagica. Anasarca. Inflammatory Œdema. Yellow Water. Morbus Maculosus.

This malady presents phenomena very similar to those found in the horse, and shows itself in mild and severe types. It has been noted by writers on diseases of cattle from Vegetius down, though under quite a variety of names.

Causes. These are uncertain, though much seems to point to microbian invasion or intoxication by microbian poisons. In France, it has been seen mainly in mature and robust working oxen after a hard day's work, when the animal has had time to cool off. Also in cows used in the yoke and milked at the same time. Poor food, starvation and low condition have also been cited as causes. In other cases, high feeding and plethora have been incriminated. It shows itself most commonly in summer and autumn, so that cold or chill must be looked on as a cause secondary to another—probably microbian or toxic. Again, it is by far the most common in the adult animal. What is more suggestive is its relative frequency in localities characterized by swamps, springy fields, hollow basins, damp hill-foots, bottom lands, and damp, impermeable or undrained soils. Again, on heavy clays where the water is preserved through summer in open ponds and often in a very corrupt condition. This at once accounts for its common appearance in summer and autumn when the offensive products of fermentation are most abundant, and corroborates the doctrine of a toxic ferment. But its origin has not been definitely traced to such products as yet. This, not-

withstanding that Kolb attributes it to *bacillus hæmorrhagicus*, and Robert, Fabert, and Dinter found the exudates swarming with *diplococci*. This may assimilate it to *septicæmia hæmorrhagica*

Lesions. In cattle killed during the early stages, circumscribed hæmorrhages and petechiæ are found in the nose, trachea, bronchia, gastric and intestinal mucosæ, cerebral membranes, pleuræ, peritoneum, bladder, and skin. In many cases these are accompanied by congestion and thickening of the walls of the lymphatics, and effusion in and under the skin in different parts of the body, but especially along its lower aspect—(belly, sternum, dewlap, limbs, lower jaw, tongue)—of a gelatinoid exudate, which raises the skin abruptly in the form of a thick cushion. At first this is soft and tremulous, but later it may have coagulated giving a great degree of resistance to the structure. Extensive cracks, fissures, and sloughs, and unhealthy sores form on these swellings. Franck especially notes the enlarged tongue gorged with blood and yellowish exudate as in gloss-anthrax. The blood extravasation may be further evidenced in the black or blood-streaked fæces, the reddish urine, and a rosy tint of the milk.

Symptoms. The animal is dull, sluggish, moves stiffly and with difficulty, and shows hyperthermia, (102° to 106° F.), inappetence, impaired or suspended rumination, heat of the roots of the ears and horns, and of the dry muzzle; the spine, and usually certain points beneath the sternum or abdomen at which swellings are about to appear, are tender to the touch. On the affected parts of the skin the temperature is raised, and there may be detected pea-like elevations which become surrounded and enveloped in extensive swellings that pit on pressure. The swellings show a preference for the thinner and looser parts of the skin, and gravitate rapidly toward dependent parts. Thus, the eyelids, roots of the ears, intermaxillary space, throat, muzzle, dewlap, ventral aspect of the body, axilla, mammæ, scrotum, groin, thigh, knee and hock are favorite spots, the liquid rapidly gravitating downward through the loose connective tissue to the lowest points. The face becomes infiltrated to unsightly dimensions, interrupting prehension and threatening suffocation, the dewlap swells up to a great size, the forearms or thighs become rounded and tense, and a thick pad forms along the ventral aspect of the body. The white skin on such swellings becomes red,

they may ooze a yellowish liquid or blood, the surface becomes depilated, scaly and rough, chaps and cracks appear, going on, it may be, to deep fissures, more particularly at the flexures of the joints, on the throat or muzzle, or on other points that are subjected to pressure. Necrosis of great patches of skin is not uncommon, and these sloughing off leave large wounds with unhealthy, indolent surface and little disposed to rapid healing.

The petechiæ show early on the muzzle, the nasal and buccal mucosæ, and on other mucous membranes. Exudations also appear and a serous, often bloody, discharge escapes from the nose and concretes in colored encrustations around the nostrils. The nose may be obstructed causing the animal to breathe through the open mouth, protruding the tongue which is often also the seat of extensive swelling, discoloration and induration.

As the disease advances there is encreased dullness and prostration, marked emaciation, and anæmia, sunken eyes, encrusted eyelids, extensive areas of depilation including even the long hairs of the tail, and quite often an abundant bloody diarrhœa.

The affection may last for 24 to 40 days and under rational treatment the majority survive. There remains, however, in a certain number of cases, a permanent enlargement and fibrous induration from the organization of the exudate.

Diagnosis. A fully developed case is easily recognized. The fever and constitutional disorder, complicated by petechiæ on the mucosæ and skin ; the extensive swellings suddenly formed, oözing serum or blood, and tending to fissures and necrosis ; and the discharging of blood from the nose, bowels, kidneys and udder, in the absence of the bacilli of anthrax, emphysematous anthrax, malignant œdema, and wildeseuche ; the slower progress ; the low mortality ; the occurrence on a damp, springy, or impermeable soil, or one known to produce this disease ; and especially if in late summer or autumn, become virtually pathognomonic.

Prevention. Seclude cattle in late summer and autumn especially, from soils known to be productive of this affection and above all from damp wet clays, underlaid by hard-pan, from swamps, from drying up ponds and basins, from wet river bottoms and deltas and from springy fields generally. Fields of this kind may be reserved for cultivated crops or for raising hay. The fundamental remedy is thorough drainage, and a subsequent abandonment of the land for a year or two to other crops to allow

of a dissipation of the poison. Sudden chills after being heated or fatigued, and exposure in the pasture in cold nights are to be avoided.

Treatment. The French writers up to the present extol bleeding for the early stages. Its benefit must apparently depend on the diminution of the blood tension allowing the distended capillaries to resume their normal contractility, and in the lessening of the tendency to exosmosis, and the encrease of endosmosis so that much of the poison in the blood is removed and what remains is largely diluted and rendered comparatively harmless. However well this may operate in the strong and plethoric, it cannot be considered as applicable to the weak or anæmic, nor to advanced cases in which the vital powers are already seriously reduced by the abundant exudations and extravasation, and by the narcotic and devitalizing action of the circulating toxins.

With us the demand is usually met by laxatives and diuretics, under the idea that these are less debilitating and that if the morbid process can be arrested, recovery is more prompt and perfect. A purge of sulphate of soda is followed by full doses of nitrate or acetate of potash, in combination with iodide of potassium, or chlorate of potash; or bicarbonate of soda associated with hyposulphite of soda. These or other diuretics should be pushed so as to produce free diuresis. Stimulating antiseptic diuretics like ol. terebinth, may be added. Iodine solutions injected into the trachea have been recommended as for the horse (Huber, Heuberger, Rohr,) and the serum or silver preparations or adrenalin may be tried.

Locally, astringents and antiseptics have given good results. Solutions of alum or lead acetate with carbolic acid and iodine; frictions with soap liniment, with a mixture of aqua ammonia, oil of turpentine and oil, or with mixtures of oils of origanum, cajeput and peppermint, and iodine, or simple painting with tincture of iodine may be cited. Fissures and open sores may be dressed with mercuric chloride solutions (1:1000), Lugol's solution, phenic acid lotion, solution of creolin, lysol, or chloronaphtholeum, camphorated spirit, or other antiseptic agent. The nose should be injected, cleared of eschars, and if necessary propped open.

An abundant, wholesome, easily digested diet, pure air, a dry stall and protection against cold are essential.

CHICKEN CHOLERA. FOWL CHOLERA. CHICKEN TYPHOID. CHICKEN PASTEURELLOSIS.

Definition. Historic notes. Bacteriology: Bacterium choleræ gallinaceæ, nonmotile, with polar stain, bleached by iodine, nonliquefying, causes septicæmic lesions. Lignieres' bacillus, culture features, non gasogenic, acidifies dextrose; vitality in disinfectants; survives drying. Accessory causes: new birds in flock, or eggs for hatching, mingling of flocks, manure, watershed, streams, ponds, dust, wild birds, buzzards, rabbits, insects, infected soil. Susceptible animals: hens, doves, peafowl, pheasants, parrots, ducks, small birds, guinea pigs, rabbits, white and gray mice. Effect on sheep, horse, man, cow, hog. Incubation 18 to 48 hours. Symptoms: in fulminant cases rarely seen, in acute, anorexia, depression, debility, apathy, ruffled feathers, sunken head, neck, wings, tail, tremors, nasal and buccal discharge, hyperthermia, sighing, violet comb and wattles, thirst, pultaceous fæces, later glairy, green and fetid. Temperature becomes subnormal, inability to rise, stupor, convulsions, death in 1 to 3 days. Mild cases last 7 days. Cases caused by one microbe have slough only. Lesions: congested, petechiated, hæmorrhagic intestinal mucosa; contents of bowels watery, frothy, bloody; epithelial degeneration and desquamation; abrasions, croupous exudates, enlarged congested lymph glands, fermenting contents of crop. Petechiæ general, spleen and liver swollen, congested, friable; kidneys congested; lungs hyperæmic or blood gorged. Blood diffluent with microbe. Anæmia. Emaciation. Arthritis, Diagnosis: by rapid spread, infection origin, early excessive mortality, hæmorrhagic lesions, microbe in blood and liquid ingesta. Prognosis: Mortality 90 to 95 per cent. at outset. Prevention: Quarantine new birds, inside screens in summer; burn or acidify manure; exclude buzzards, vermin, wild birds, and visitors; separate sick, kill, burn, disinfect, divide infected flock in small lots; prevent wandering in fowls, destroy insects. Phenic acid subcutem. Immunization: inoculate in breast with one microbe (Salmon): or with weakened virus (Pasteur). Limitations. Treatment: gastric and intestinal disinfection—copperas, mineral acids, carbolic or salicylic acids, aromatics, quinine, naphthol, tar, phenic acid, subcutem.

Definition. A febrile hemorrhagic septicæmia of chickens and other fowls (pigeons, ducks, geese, parrots, etc.) communicable to certain rodents and other animals, and characterized by a short incubation, rapid progress, great prostration, violent diarrhœa usually greenish, and a high mortality (90 to 95 per cent).

History, Geographical distribution. It is quoted as prevalent in Lombardy in 1789, in East India in 1817, in France in 1825, and generally in Europe and America in the last half century.

The *losses* from the ravages of this disease are far greater than the average value of the individual animal would lead one to suppose, but with domestic fowls numbering 300,000,000 and a yearly egg crop bordering on a billion dozens it may well be called enormous.

Bacteriology. Chicken cholera is caused by a very small ovoid bacterium (B. Choleræ gallinaceæ, B. Avicidum) about 0.3 to 1.8μ long, (0.3–1μ, Friedberger; 1–2μ, Ward), as found in the blood and tissues of the fowl. It has the general characters of the groups which cause hemorrhagic septicæmia, thus: 1. It fixes, above all at the poles, the ordinary anilin colors; 2. It is decolorized by the methods of Gram and Wiegert (iodine solutions); 3. It grows on gelatine without liquefying it; 4. It produces acute septicemic lesions; 5. It tends to polymorphism when grown under different conditions. In the peritoneum of the guinea pig it forms cocco-bacilli tending in acute and violent cases to diplococci. Fed to rabbits it appears in the fæces as a minute bacillus. Even the mode of staining causes a difference in appearance. Fixed in alcohol-ether, and then stained in Ziehl's phenated preparation, coloring is polar, and the central area clear. If in place of Ziehl's fluid, hot fuchsin solution is used warm for ½ a minute, a bacillus or cocco-bacillus is shown (Lignieres). It is nonmotile, though some observers have been misled by Brownian movements. There are no flagella. In old bouillon cultures short chain forms are met with. No spores are formed.

From fresh cultures, in flask or in animals, the bacterium grows readily in alkaline culture media. In *bouillon* a turbidity ensues, and after some days pellicles form on the surface and walls, and the liquid slowly clears. The addition of a little blood serum, sugar or glycerine encreases and hastens the growth, while acid retards or prevents. This is common also to other septicæmic germs. In *gelatine* growth is tardy, but in two or three days there are whitish glistening colonies, becoming opaque later, and appearing granular if slightly magnified. In punctures minute colonies form along the line of culture and one at the surface, at first translucent; later opaque. On *agar* the colonies grow faster with similar appearance. On *gelose* at 37° C. the colonies are bluish and iridescent, at first, and later opaque. If the germ has been repeatedly passed through the Guinea pig, they are more translucent. On *potato* with alkaline surface,

there is a delicate grayish yellow growth after 48 hours; if acid, growth ceases. In *milk* there is no coagulation nor acidification for four weeks; then it becomes slowly clear and opalescent. With *sugars* it is not gas producing. With dextrose it forms an acid solution; with saccharose or lactose an alkaline one. With Canadian germ formed acid in lactose solution, (Higgins). Cultures in peptone gave a strong *indol* reaction (V. A. Moore). Lignieres found no indol in cultures in pancreatic bouillon.

The bacterium perishes when heated to 58° C. for fifteen minutes. It dies in carbolic acid solution (1:100) in five minutes; in sulphuric acid solution (0.25:100) in ten minutes; in lime water in ten minutes; and in sulphur fumes in three hours. Easily destroyed by disinfectants, it remains potent for months in flasks or buildings that are not subjected to disinfection. It is not killed by drying (T. Smith and V. A. Moore) nor by zero temperature maintained for seventeen hours.

The cultures, especially those made in agar have a very characteristic odor.

Accessory Causes. Birds sent to poultry shows will often contract the disease and introduce it into the home flock on their return. It may also be imported in newly purchased birds, or on eggs obtained for hatching. It is even alleged that it has been propagated by feeding healthy hens on the eggs of diseased ones. When chickens run at large it passes easily from flock to flock in the immediate vicinity. The infected manure is, however, the most common channel of infection. Carried on the feet or bill this contaminates the food and drinking water, and washed into streams and ponds, it finally in any case reaches the alimentary canal of the susceptible bird. Or drying up and raised as dust it is inhaled into the lungs. Or finally from any such source it infects any open sore. As granivorous birds, wild and tame, suffer from fowl cholera, it is often introduced by the wild, especially coming from infected poultry yards. Predatory birds, like hawks and buzzards, but the latter especially, are common bearers of infection. Rabbits, which contract the affection so readily, transmit it equally with birds, but man or beast, soiled by the manure will convey it. Insects are among the most prolific bearers, hence, as noted by Salmon, the infection may fail to overstep a close fence in winter, but is subject to no such limitation during the fly

season. In this respect chicken cholera agrees with Asiatic cholera, typhoid fever and other affections in which the virus abounds in the alvine discharges, The sale and transportation of the guano from the infected poultry yard is a direct cause of new outbreaks. Feeding on the carcasses or offal of the infected birds is a further cause. It must not be forgotten that the microbe is largely saprophytic, living indefinitely in the organic matter in soils, and determining new outbreaks when brought in contact with susceptible animals. Thus a period of immunity may be followed by infection when new birds are brought in or when young and susceptible ones grow up.

Susceptible Animals. Fowl cholera is preëminently a disease of chickens, but the microbe is successfully transferred to pigeons, peafowl, pheasants, parrots, ducks, canaries, sparrows and other small birds, also to Guinea pigs, rabbits, white and gray mice. The Canadian form is deadly to turkeys (Higgins). Guinea pigs have abscesses in the seats of inoculation (Pasteur); the same is alleged of sheep and horses (Kitt), and man (Marchiafava, Celli). Injection into a cow's teat caused chronic catarrhal mammitis in which the microbe persisted for a long while (Kitt). Like other members of the group of microbes causing septicæmia hemorrhagica, the pathogeny and even the morphology are liable to material modification as grown in different environment (genera). In the Canadian as compared with European epizootics there were the following differential features:—The bacterium was smaller; several were often connected together; they grow more slowly in gelatine cultures (Colonies not before the 3d day); they were more easily destroyed by solutions of carbolic acid (1:100 in 5 minutes); they developed acid with dextrine and lactose; they caused earlier deaths (birds sometimes perishing in 3 hours), (Higgins). Some of the forms of cholera occurring among domesticated birds and held to be distinct diseases may find in this an explanation. Rabieaux claims that under favorable conditions it has been transmitted to the frog.

Incubation. This varies from 18 to 48 hours, the usual being 24 hours.

Symptoms. In some *fulminant cases* the animal is found dead a few hours after apparently blooming health; it may even have died on the nest or fallen dead from the roost. Cadeac speaks of

transient symptoms even in such cases—extreme dulness, prostration, somnolence, seclusion in a cool, dark place, ruffling of feathers, sinking of the head between the wings, drooping, trailing wings and tail, violet comb, gaping, discharge of glairy mucus from the bill, convulsions and death. These symptoms last from two to five hours.

In *acute* but less fulminant forms there is loss of appetite, depression, debility, apathy, erection of the feathers. sinking of the head, swaying when made to walk, drooping wings and tail, sitting on the breast, convulsive tremblings, discharge of filmy or frothy mucus by the nose or mouth, vomiting, hyperthermia (108° to 111° F), sighing breathing, inflation of the crop, violet colored comb, wattles and mucosæ, great thirst and diarrhœa, at first pultaceous and light yellow, later glairy, green and fetid. The feathers round the anus become soaked and matted with the discharge. Temperature becomes subnormal, the patient falls and is unable to rise, and finally dies in a stupor or convulsions, the illness having lasted 1 to 3 days.

Milder cases occurring chiefly towards the end of an outbreak when the less susceptible animals only are left, or when the microbe has become less virulent, show a larger ratio of recoveries. These show a lack of spirit and vigor, impaired appetite, diarrhœa, emaciation, dulness, prostration, moping, ruffling of the plumes, dark discoloration of the comb, and often swelling of one or more important joints (femoro-tibial, etc.). These may burst and discharge a reddish pus, or simply form dark or grayish swellings. These cases may drag along for a week or more and finally die in marasmus. The minute bacillus is not obtainable from these (Lignieres).

Cases inoculated in the pectoral muscles with only one or two microbes usually have only a circumscribed slough, with loss of condition, and after the elimination of the slough and the healing of the sore the bird proves immune.

Lesions. The alimentary canal is the main seat of morbid changes. The intestinal walls, and especially the mucosa, have points and patches of blood extravasation, extensive areas of congestion with ramified redness, exudation and thickening. The intestinal contents are watery, frothy, browned or blackened by effused blood, and swarming with ferments including the

specific bacterium. The mucous surface is brownish or blackish, and epithelial degeneration and desquamation with abrasions are not uncommon especially on the summits of the duodenal folds and villi. Croupous exudates and swelling or ulceration of the follicles are met with. The lymph glands at the base of the cæcum are often enlarged and congested. The crop is full of watery, pulpy, frothy or slimy contents, and its mucosa and that of the pharynx may be deeply congested.

Elsewhere the lesions suggest rather the action of destructive toxins and the profound changes in the blood. Pericardium and endocardium are usually studded with dark petechiæ, and congestion and even slight exudation may be present. The spleen is enlarged, soft, and gorged with blood. The liver is swollen, congested, extremely friable, and mottled, grayish white from degenerations. The kidneys are dark red, and friable. The lungs may show slight hyperæmia only, or a blood engorgement and consolidation, and are then easily reduced to a dark red pulp. Friedberger and Fröhner say that respiratory changes are most frequent in land birds ; and intestinal and cardiac in water fowl.

The blood is diffluent coagulating loosely if at all, of a brownish red color, reddening slowly and imperfectly in contact with air, and like the tissues contains an abundance of the characteristic bacterium staining deeply at the poles and clear in the center.

In birds that survive a few days there are marked anæmia and emaciation, and the muscular system is of a grayish red color, with fatty degeneration. In acute and fulminant cases on the other hand the muscles may be full and of the normal red color.

In arthritic cases the congestion and thickening of the soft tissues, and the excess of synovia, are supplemented by destruction of the articular cartilage and by areas of bone abrasion. In the more tardy cases collections of caseous matter are found.

Diagnosis. This is based on the demonstrably highly contagious character of the disease, its rapid spread in a flock, and from the first to nearby adjoining flocks in summer, the short period of incubation, the constancy and nature of the diarrhœa. the speedy and great mortality, and the hemorrhagic lesions of comb, bowels, heart, lungs, liver, kidneys, spleen, and serous membranes. The demonstration of the bacterium in the blood

and affected tissues is conclusive. Kitt points out that inoculation of a pigeon kills the bird in 12 to 48 hours, with dry yellow exudate in patches of from ½ to ¾ inch in diameter on the surface of the muscles, and yellow discoloration and nodular induration beneath.

Prognosis. The mortality reaches 90 to 95 per cent. The negative chemiotaxis exerted on the leucocytes by the microbe, precludes defensive phagocytosis, and the progress of the deadly microbe is comparatively unhindered. Toward the end of a severe outbreak, and in certain mild epizoötics the recoveries are much greater.

Prevention. All birds bought or otherwise acquired and all birds returning from shows should be quarantined for one week before being allowed to mingle with the flock. In summer this should be conducted inside fly screens. The manure should be burned, carefully secluded, or treated with dilute sulphuric acid. Buzzards and vermin as possible bearers of the infection should be excluded from poultry yards. So with human beings, dogs, etc., coming from infected places. In an infected flock the sick should be at once separated, killed and burned or treated with sulphuric acid. All manure should be treated in the same way. Buildings, yards and runs should be thoroughly cleaned and liberally sprinkled with a dilute sulphuric acid (2:100). If the birds can be divided up in small groups (say of 5) the appearance of the disease will only endanger that group. In small flocks or with very valuable birds it may even be well to take the body temperature morning and night and separate at once any bird showing a rise. Any diseased or suspected flock should be kept where its manure will not be washed into wells, running streams or ponds to which other birds have access. In a locality where the disease exists fowls should not be allowed to run at large. In winter this is very effective ; in summer owing to the danger from insect bearers, it must be supplemented by the most scrupulous cleanliness of poultry houses and yards, and by a liberal sprinkling with dilute sulphuric acid, or other disinfectant, to be made especially abundant and frequent on the manure. Nocard cuts short the disease by injections, subcutem of a 5 per cent. solution of carbolic acid.

Immunization. With valuable birds it may be desirable to

secure immunization by non-fatal inoculations. Salmon secured this by first estimating the number of microbes in a mm. of the blood, then diluting until five drops would contain but one, or at most two of these organisms, and injecting this amount into the pectoral muscles. A sequestrum forms in the muscle and is gradually sloughed out, and the cavity heals, with resulting immunity.

Pasteur produced a weakened virus by exposing the artificial bouillon cultures to air for from three to ten months, the strength decreasing with the length of exposure. The weaker form produces slight illness only, from which recovery is prompt. A second and stronger virus is used ten to twelve days later and produces a real immunity.

The drawbacks to these methods are: 1st, that fowls are of too little value, to warrant inoculation in healthy flocks; 2d, that in infected flocks, where it is employed, the more susceptible birds are usually already contaminated, and a large proportion die in spite of it; and 3d, that it becomes a means of planting the infection in new localities (Kitt).

Treatment. The disease is so deadly that little can be hoped from medicinal treatment. It has been directed mainly to gastric and intestinal disinfection. Copperas and sulphuric or hydrochloric acid in the drinking water ½ to 1 per cent. of each is at once prophylactic and curative. Friedberger and Fröhner add fennel or peppermint, and give a tablespoonful every hour to an affected chicken. Other agents recommended are: carbolic acid (5;100) by the mouth or subcutem (Nocard), salicylate of soda, quinia (Cadeac), tannic acid (2:100), salol, naphthol, tar water, etc.

See Catarrhal Enteritis in Birds: Vol. II. p. 258, Bacillus Gallinorum; B. Coli Commune: B. of Duck Cholera: Spirillum Metchnikowi.

CHICKEN-PEST.

Definition. A deadly form of hæmorrhagic septicaemia caused by an invisible, filtrable microbe, and leading to hyperthermia, great prostration, dullness, stupor and early death with extensive petechiation, blood extravasation, and exudation in the bowels, mucosa, serosæ, liver, lungs, spleen, kidneys, etc.

Synonyms. Chicken typhus; Chicken typhoid; Exudative typhus; Vogelpest; Birdpest; Henpest; Kyanolophia.

History. Described by Centanni as prevailing in Italy in 1891, this deadly affection extended to the Tyrolean Alps, was found in Brunswick in 1901, and soon in Wurtenberg, Hesse, Belgium, Oldenburg, and North Prussia.

Cause. That the disease is most virulently contagious has been abundantly proved, and that the germ is self-multiplying is unquestionable, 4 cc. of a solution of virulent blood (1:125.000.000) proving fatal to a hen of 370 grams, and such successful inoculations from hen to hen for four or more removes losing nothing of the virulence (Maggiora and Valenti). Yet the most careful staining and the highest powers of the microscope have hitherto failed to detect the germ, which passes readily through a Berkefield filter. Maggiora and Valenti failed to pass it through a Chamberland K filter. It is evidently one of the infinitesimal, invisible microbes like those of rabies, lung plague, Rinderpest and Aphthous fever.

Virulent blood dried at room temperature and kept in the dark, remained virulent for 22 days, but not for 42. In dull (diffuse) light, virulence lasted 15 days, in bright sunlight, less than 40 hours. It is destroyed in half an hour at 158° F., in 4 per cent. carbolic acid in 15 minutes, in mercuric chloride (1:1000) in 10 minutes, and even in concentrated milk of lime.

Pathogenesis. The virus is deadly to chickens and turkeys, while ducks, geese, swans, pigeons, mice, Guineapigs, and rabbits are immune.

Symptoms. The affected chicken becomes listless, with ruffled feathers, blood-red or dark leaden comb, drowsiness, coma and atony. The first day the affected hen leaves the flock and is found apart, dull and apathetic, with slightly elevated tempera-

ture. On the second day the patient declines food, sits on its breast or abdomen, with head sunk between its wings, feathers erect, eyes closed or semi-closed, and when raised it moves unsteadily, or falls to one side, forward or backward. The comb and wattles are now usually violet or black, and the droppings still normal or slightly soft and green. The temperature varies from 108° to 113° F. Drowsiness advances into coma, the patient can no longer be roused, the breathing is stertorous or accompanied by audible plaints, and death usually occurs on the third day, preceded by a marked fall of temperature (86° F.) and perhaps spasms or paralysis. Some live only a few hours, others for a week.

Lesions. These are not very diagnostic. Local congestions and blood extravasations may be diffused over the mucosæ of the alimentary canal and air passages, but the bowels rarely show any such congestions or secretion as distinguish fowl cholera. Petechiation and blood extravasation of the serosæ, above all of the pericardium, pleura, and peritoneum are more in evidence. The lungs may show congestion and circumscribed areas of hepatization. The liver is enlarged, and like the spleen and kidneys hyperæmic or petechiated. Congestion and discoloration of the lymph glands are common. Subcutaneous exudations are met with. The localization of the lesions is however far from constant, and it is their hæmorrhagic character rather than their seat that is significant.

Diagnosis. The features which distinguish chickenpest from fowl cholera are mainly that chickens alone suffer, and particularly that mature ducks and pigeons are immune; that diarrhœa is absent; that there is no severe inflammation of the bowels, though petechiæ and blood extravasation may be present: and that death follows more tardily—on the second to the fourth day in place of on the first to the third. The failure to find the *bacillus choleræ gallineæ*, or any definite microbe in all cases in the blood is even more diagnostic.

Medicinal treatment has been eminently unsatisfactory.

Prevention may be sought by separating the sick and healthy, disinfecting the poultry houses and yards, furnishing fresh runs to the exclusion of the old ones, excluding from water-courses which may be suspected of exposure to infection, exclusion of

food or litter brought from infected places, and the keeping of the flocks carefully secluded from contamination by other poultry, or in summer through wild birds or insects. All infected birds should be promptly destroyed and boiled, burned or deeply buried, and all droppings and other materials taken from the infected yards dealt with in the same way. The absolute seclusion of an infected flock and the prompt destruction of all sick fowls, should be legally enforced, together with disinfection. The removal of birds from a recently infected flock for any purpose (sale, gift, exhibition or otherwise) should be made penal. Even when an entire flock has to be sacrificed, the eggs washed in sterilized water may be availed of to speedily build up a new flock of equal pedigree and excellence.

MOLLUSCUM CONTAGIOSUM IN BIRDS.

Synonyms. Epithelioma Contagiosum ; Acné Varioliforma ; Acneoid Epithiliosis ; Bird-pox ; Geflügelpocken.

Definition. A croupous or diphtheritic inflammation of the mucosa and skin due to an invisible, infinitesimal, filtrable microbe.

Contagion. This affection is virulently contagious to pigeon, chicken and goose, and is easily transferred by rubbing the morbid products on the delicate skin of the comb, wattles, eyelids, beak, etc., or on the buccal or nasal mucosa. In such an exposed situation the raw surface becomes the seat of accidental microbian infection, and the disease has been attributed to protozoa (gregarina, coccidia, blastomycetes). The experiments of Marx and Sticker (1902) have, however, definitely shown that the virulent material passes through a Berkefield filter though it is arrested by the finest porcelain filter. This ranks it with the invisible microbes of rabies, lung plague, etc., the demonstration of which under the microscope is not yet accomplished. It has great vitality and resistance, having survived complete drying at room temperature ; exposure to sunlight or to 8° F. for one week ; three hours at 140° F. ; or one week in glycerine. It perishes in two per cent carbolic acid. Passed from the pigeon through the system of the hen, it parts with its virulence for the pigeon.

Recovery from a first attack brings immunity against a second.

Symptoms. These resemble those of chicken diphtheria. In slight attacks there is inflammation with more or less production of false membrane in the mouth, nose, throat and eyes and at times in the bowels causing diarrhœa. When it extends to the skin, warty-like masses appear on the angles of the beak, nasal openings, eyelids, ears, comb, wattles or bare parts of the head, at first the size of poppy seed or millet seed, and later of a hemp seed, or even a pea, or cherry stone, imbilicated in the centre, unlike an ordinary wart, and of a variable color, reddish or yellowish gray or brown, and even in some recent cases, a pearly lustre. The individual nodule is definitely circumscribed and is easily enucleated by pressure between finger and thumb, the contents

escaping in the form of a thick creamy mass, which suggests the error that it was variolous. A nodule may stand isolated or discrete; in other cases a number aggregate together like a granulating surface; in still others they become confluent, and grow out in considerable masses, the surface having a conglomerate appearance like that of a strawberry. Under this exuberant growth it becomes rough, scurfy and more or less broken by fissures. The drying exudate may close and seal the eyelids, or obstruct the orifices of the nose or ears.

Pathological Anatomy. The first consideration is that this is a product of the dermis or rather of the deep layer of the epithelium (Malpighian layer) and does not encroach on the cutis vera or adjacent tissues, hence the ease with which it is enuncleated. The deep, or basal cells of these mucous layers are normal, but in the 3d or 4th stratum, the cells become granular, opaque and swollen, the nucleus is displaced or disappears, yet the outer membrane remains distinct and thickened and from this the cell gradually dries into a scaly mass which is desquamated. The form of the individual cell may be round, oval, or fusiform with attenuated ends. A careful dissection may show the clusters of altered epithelial cells enclosed in separate chambers converging above to a point in the center of the mass. It is at this point that drying and exfoliation takes place most rapidly, accounting for the umbilication.

Prognosis. In the milder cases, affecting the skin, a spontaneous recovery may take place, the morbid product slowly drying up and exfoliating to leave a healthy surface. In the mouth, nose and pharynx a recovery is more tardy, or the excess of the morbid product may cause suffocation and death, or its persistence may lead to a great impairment of health, and marasmus. The worst cases are those in which the disease extends to the crop, proventriculus, gizzard or bowels. There is dulness, prostration, and emaciation in spite of an inordinate appetite, ruffling of the feathers, dragging of the wings and tail, irritable bowels or diarrhœa, raucous cries, and death in three or four weeks (4 or 5 weeks after inoculation.—Bollinger).

Treatment. This has been carried out most successfully along the lines resorted to for fowl diphtheria (croup). Painting the affected part with a two per cent solution of creolin or carbolic

acid does well in the early stages, or half the water may be advantageously replaced by glycerine. Lysol, creosote, salicylic acid, or boric acid may be substituted. In advanced cases with extensive deposits the latter may first be scraped off, and then the antiseptic applied. Even glycerine alone, liberally applied has proved successful. For the enteritic form glycerine has been administered by the mouth, a tea- to a tablespoonful for a mature goose. In this last case borax, salicylate of soda, or salol might be tried.

Prevention. To exterminate or prevent the infection, avoid contact with strange birds, from neighboring yards, in exhibitions, or in shipping; remove sick birds from infected flocks, disinfect buildings and runs, or change the latter; divide up the flock in separate pens of three or four fowls each, and remove at once for treatment any sick fowl, afterward disinfecting the pen; take all possible precautions against diffusion of infection through the food or water. Generous feeding and a course of bitters, with copperas in the drinking water may be of material advantage.

DISTEMPER IN DOGS AND CATS.

Definition. Synonyms. Animals susceptible: dog, fox, jackal, hyena, wolf. Historic notes. Causes: contagion, inoculation, immunity, contact with sick, clothing, bedding, kennel: infection fixed—little diffusible, dogs at large, chill, domestication, high breeding, special breeds, shows, public conveyances, vegetable diet, debilitating conditions, catarrhs, change of climate, recurrent attacks, youth, native susceptibility, teething. Microbiology: micrococci, bacilli, mixed cultures, Shantyr's observations, Lignieres'. Virulent products—all secretions and exudates. Vitality: virus survives drying, freezing, dilution in water. Destroyed by disinfectants. Forms of distemper: catarrhal, opthalmic, cutaneous, gastro-hepatic, bronchitic, pulmonary, nervous. Duration 20 to 30 days. Mortality 20 to 70 per cent. Prognosis: unpromising conditions. Incubation 4 to 7 days. Symptoms: hyperthermia (103° to 104°), dulness, debility, anorexia, staring coat, tremors, seeking warmth, early fatigue, dry burning nose and footpads, irregular temperature, simultaneous congestion of all visible mucosæ; respiratory phenomena; sneezing, congestion of nose, discharge, blocking, snuffling, rubbing, cough, retching, vomiting; percussion flatness in lungs, in islets or along the lower part; auscultation râles, wheezing, crepitus, creaking, etc.; epiphora, swollen eyelids, weeping, photophobia, muco purulent discharge, sticking of lids together, opacities, vesicles,

ulcers; red, hot, buccal mucoso, costiveness, fetid diarrhœa, tenesmus, weakness. emaciation; skin eruption, on delicate areas, papules, vesicles with colored contents, pustules, sticky, greasy exudate; irritability, restlessness, taciturnity, depraved appetite, spasms, delirium, paresis, epilepsy, chorea. Lesions: inflammation, degeneration, ulceration on air passages, alimentary tract, lymph glands, kidneys, liver, cerebral and spinal meninges, leucocytic infiltration: offensive odor. Lignieres' views of microbes. Prevention: quarantine new dogs for 14 days, wash, disinfect all colors, etc., avoid shows and meetings, exclude street dogs, protect against mice, rats, birds, shut up all dogs during an epizoötic; separate a pack into small lots; seclude the sick and all belonging to them. Immunization: by lung exudate, by weakened cultures. Treatment: hygienic, dietetic, warm baths, antipyretics, antiferments, calomel, phenic acid, eliminants, expectorants, collyria, emetic, demulcents, bismuth, etc., pepsin, quinine, nerve sedatives, tonics.

Definition. A contagious, febrile affection attacking dogs (and by inoculation cats), and tending to local inflammatory and degenerative lesions in the mucosæ, lungs, bowels, liver, skin, kidneys, and nervous system,—a first attack usually immunizing against a second.

Synonyms. Contagious catarrhal fever: Dog ill; Bronchial Catarrh: Intestinal Catarrh; *Fr.* Maladie des Chiens, Maladie du jeune age, Typhoïde, Typhus des chenils, Variole du chien; *Ger.* Staupe; *It.* Cimurro,

Animals susceptible. Dogs, especially puppies and young dogs, and other members of the canine race, fox, jackal, hyena, wolf. Cats suffer from inoculation (Laosson), also apes (Cadeac). Old cats and dogs are often immune, also all animals that have passed through one attack of the disease.

History. The older English veterinarians quote the epizoötics in dogs described by Virgil, Aristotle and even Homer as probably distemper. Laosson attributes to it a canine epizoötic which prevailed in Bohemia in 1028. It appears to have been unknown in Europe in the earlier third of the 18th century though prevailing in Peru. According to Ulloa, it was introduced from Peru into Spain in 1735, whence, it spread into France (1740), Germany (1748), Ionian Isles, Greece (1759), England (1760), Italy (1764) and Russia (1770), Sweden and Norway (1815), Siberia (1821). Since that time it has prevailed in Europe and many dependencies of European nations.

Causes. Its advent in Europe as a new disease in the second third of the 18th century, its steady spread, its continued prevalence, and extension, concur with the infection of kennels and districts by the introduction of a sick dog, in demonstrating its purely contagious nature, and this implies a living microbe transferred from animal to animal. If any doubt remained it must be dispelled by the inoculations which have been constantly successful when made on young puppies, or dogs that have not previously suffered, and on cats. A first attack confers immunity.

Among *accessory causes* may be especially named *contact with the sick dog*, its *clothing* or *bedding*, and above all its *kennel.* A kennel may retain infection indefinitely, especially if there is a constant accession of puppies or susceptible dogs. On the contrary it is not readily carried on the clothes of attendants, and the inmates of a kennel often remain sound, though only separated by a yard from the infected one, and though cared for by the same attendant. Yet so universally diffused is the contagion that few dogs escape it until they are a year old.

The habit of letting dogs run at large, to meet in roads and fields is a most prolific cause, which might easily be done away with where distemper is prevalent.

A *chill* is a common condition, hence injudicious washing, swimming in cold water, exposure of house dogs to cold storms, or outdoors at night, sleeping in cold, damp cellars, on cold stones or metal plates, or in passages in a current of cold air are frequent factors. Yet it often spreads rapidly in the summer, the heat favoring the preservation and diffusion of the germ.

House dogs as a rule suffer more severely, as their systems are more sensitive to the cold, and the resisting power to invading microbes is lessened. Country dogs and those living in the open air are hardier and more resistant and often have the disease in a mild form.

High bred dogs suffer more severely probably largely from the greater protection and care lavished on them and their consequent diminished power of resistance. Newfoundlands, great Danes, pointers, pugs, poodles, spaniels and greyhounds may be named as especially liable.

The exposure to infection at *shows*, and in traveling by *rail*, *steamboat* or *other infected conveyance* must be considered as among the prominent causes.

An exclusively *bread diet* has been recognized as a predisposing cause, by reason of its lowering the stamina of the carnivorous animal.

Any condition which induces *debility* whether severe or continued disease, parasitisms, impure air, under feeding, improper food, rachitis, or scurvy, etc., must be admitted to operate in the same way. Any *catarrhal disease* of the nose or bronchia is especially conducive to the affection by weakening the mucosa and making an easy entrance channel for the germs.

Change of climate is a strongly predisposing condition, which not only hastens an attack, but even, at times, arouses anew the susceptibility in dogs that have passed through a first attack. Dogs that suffered in England have had a second attack in India, and some have even had a third attack when brought back to Europe. Four attacks within a year, in the same dog, and without change of climate, are recorded by Friedberger and Fröhner.

Youth is much more susceptible than age, even apart from the immunity which comes to the mature dog from a first attack. Yet some puppies are insusceptible from birth.

Microbiology. No one pathogenic organism has been proved to be the constant infecting agent, yet two classes of investigators have contended in favor of micrococci and bacilli respectively.

Micrococci, 1 to 3 μ in diameter, singly or in chains of 6 or 8 have been found in the blood, lungs, liver, kidneys and spleen (Semmer, Friedberger, Krajewski, Rabe, Mathis, Marcone, Meloni, Kitt).

Bacilli were found associated with micrococci by Semmer, Legrain and Jacquot, Laosson, Millais, Schantyr, and Galli-Valerio. As observed by the latter they were 1.25, to 2.5μ long, by 0.31μ broad and took the Gram stain. Cultivated on gelatine the colonies were waxy, lustrous points, which indented the gelatine without liquefying it.

Cultures of the mixed bacteria (cocci and bacilli) and their inoculation on young dogs produced the symptoms of distemper with subsequent immunity (Laosson, Millais). Legrain and Jacquot claimed immunity, as resulting from inoculations with the cocci in pure cultures. Galli-Valerio, using the mixed cultures produced all the symptoms (pulmonary and cerebro-spinal) of distemper.

Schantyr, ignoring the cocci, describes three bacilli which cause three different diseases (distemper, abdominal typhus, and typhoid), but the distinctions are not clear nor generally accepted.

Lignieres and Even, in Argentina, and dealing with the more susceptible high bred dogs (fox terriers, great Danes and carlins), found constantly in the blood of the dog, in the early stages of the disease, a long, delicate bacillus, non-motile and easily stained in aniline, but not by Gram's method. Inoculated on the Guinea pig it shortens, approximating rapidly in successive passages to a cocco-bacillus, and assumes the general characters of Lignieres's *pasteurella*. Different bacteria may be found in the lungs, bronchial and nasal mucus, tears and vesicles.

Lignieres's *pasteurella* is inoculable on the Guinea pig, mouse, rabbit, and dog, producing the symptoms of local or general infection. Two centigrammes, subcutem, in the *Guinea pig*, produced a local œdema; disappearing in four or five days and securing immunity. Five cc. subcutem proved fatal in 48 hours; intraperitoneal in 24 hours. In the *rabbit* 1 cc., subcutem, caused local œdema for 2 days and hyperthermia (104°) for 3 days; 1 cc. intravenously, caused fever for 3 weeks, diffluent blood and hæmorrhagic lesions in lungs, liver, kidneys, bowels, spleen and serosæ. In the mouse, 4 to 8 drops, subcutem, caused œdema, and recovery or death in 2 to 4 days. In such cases the microbe was found in pure cultures in the blood or inoculated tissues.

Inoculations of the pure cultures on dogs produced in different cases, gastro-enteritis, pneumonia, pleuro-pericarditis, and arthritis, in various combinations. If the animal survives it is immune.

Grown on *peptonized bouillon*, *neutral* or *slightly alkaline*, the bacillus forms, in 24 hours, small granular colonies which fall to the bottom, leaving the liquid clear. The addition of serum renders this more abundant without causing opacity.

On *pancreatic* bouillon the growth is very free without indol.

On *gelatine plates* there are fine punctiform colonies, transparent, but becoming white and opaque in 8 or 10 days. Similar colonies form in stab and streak cultures. The gelatine is not liquefied. Cultures in *milk* cause neither coagulation nor acidity. There is little or no growth in hay tea, on potato, nor in vacuum. The cultures have the peculiar odor of the pasteurella.

Virulent Products. Infection is present sooner or later in all the morbid animal products. The nasal mucus, bronchial exudate, saliva, tears, contents of the cutaneous vesicle, milk, contents of the bowels, and the blood have been successfully inoculated on susceptible subjects. Inoculations with blood failed with Bryce, but proved successful with Konnhauser, Krajewski, Even, Lignieres and Physalix, so that the first-named cases may be explained by a prior immunity, or by a too advanced stage of the case which furnished the matter. The virulence is not lost nor even diminished by drying at ordinary temperatures, freezing (10° F.), nor by moderate dilution in water. Prolonged exposure at ordinary temperature, however, reduces the virulence, which is greatly impaired in 15 to 25 days (Laosson), or, if dried, in three months and upward (Krajewski). Virulence is easily destroyed by disinfectants,—thorough washing (Menard) hypochlorous acid (Trasbot), chloride of lime, mercuric chloride, etc.

Forms of Distemper. This disease is extremely protean in its manifestations. Many cases, in country districts especially, are manifested by a slight fever, with a *catarrhal condition of the mucosa* of the nose, eyes, and throat. In other cases there may be simply a slight *conjunctivitis or keratitis*, and in still others a *cutaneous eruption*, papular, vesicular or pastular. In some instances there is slight *gastric* or *hepatic disorder* with inappetence, nausea, and vomiting or some irregularity of the bowels. In such cases recovery may take place in eight or ten days.

In the more severe forms, especially seen in house and city dogs, and in high bred and confined dogs generally, the fever is high and persistent and the disorders often predominate in particular organs, hence we see a *catarrhal form*, a *conjunctival*, a *bronchitic*, a *pulmonary*, a *gastro-intestinal and hepatic*, a *cutaneous* and a *nervous*. The severe cases may last for twenty or thirty days or even in their sequelæ for several months. Exposure to cold contributes greatly to a pulmonary attack even in inoculated cases (Lignieres).

The *mortality* varies greatly. In country dogs it may be below 20 per cent, whereas in city dogs and those kept confined in close quarters and in large numbers it may rise to 50, 60 or even 70 per cent.

Prognosis is especially unfavorable in severe cases; in those profoundly affecting the brain, lungs or liver; in high bred or pampered dogs; in the very young; in the debilitated, anæmic or rhachitic; in those that have been recently imported from another climate and are as yet unacclimated. Puppies from debilitated dams, or those raised in a large litter with insufficient nourishment, or older dogs confined to a purely vegetable diet show less power of recuperation. Profound prostration and offensive odor, from excess of toxins are always to be dreaded.

Incubation in inoculated cases varies from four to seven days (Krajewski). When contracted by simple exposure it may seem to have been extended to fourteen or even eighteen days.

Symptoms. The *earliest symptom* is hyperthermia (103° to 104° F.) This is accompanied and followed by prostration, dulness, impaired appetite, erection of the hairs along the spine, shivering, trembling, seeking a warm place, fatigue on slight exertion, hot dry nose, burning pads of the feet, sometimes taciturnity. Later the temperature may descend even to the normal or there may be alternations of rise and fall. One of the most characteristic symptoms of distemper is the implication of more than one set of organs, so that morbid manifestations referable to the nose, eyes, throat, stomach, skin and nervous system occurring in the same subject are to be especially noted. In enumerating the prominent symptoms caused by disease of one set of organs therefore, it is not to be implied that the absence of others referring to a different class of organs is to be understood. On the contrary a complication of several is especially significant of this disease, though the predominance in one class of organ will signify a special form of the disease.

Respiratory Symptoms. One of the earliest symptoms is usually sneezing with redness of the nasal mucosa, followed by a mucopurulent blocking of the nose, and rubbing of it with the paws. With the implication of the throat there is usually local tenderness and a hard, painful cough, which may be accompanied by retching or vomiting. The breathing becomes snuffling, especially in pugs and bull dogs, and rapid and even oppressed in case of implication of the smaller bronchia and lungs. The nostrils may become glued together, the discharge red or dark in color, vesicles and sores may appear on the mucosa, and the cough gets paroxysmal, small, weak and husky or gurgling.

Percussion of the lungs may reveal small areas of flatness from exudate or collapse, and in case of pleurisy and hydrothorax there is a lack of resonance up to a given horizontal line, varying in position according to the position of the animal and always keeping to that part of the thorax which may at the time be lowest. Auscultation will reveal various sounds according to existing lesion. There may be a loud blowing murmur over the large bronchia, or at points to which this sound is conveyed through consolidated lung. Or a coarse or fine mucous râle may be present indicating the second stage of bronchitis, or a line of crepitation around a non-resonant area indicating pneumonia, or there may be friction sounds or, later, creaking murmurs from false membrane. Wheezing and sibilant sounds are not uncommon, also sounds of the heart, bronchia or bowels, heard in unwonted situations to which they are conveyed through consolidated lung tissue. Dyspnœa may become extreme, with puffing out of the cheeks, labial soufle, and violent inspiratory action. Fœtor of the breath is common. Emaciation, marasmus, sunken, pale or dark red eyes, putrid diarrhœa and nervous disorders usually precede death.

Eye Symptoms. Conjunctival congestion is one of the earliest and most constant symptoms. Weeping, swollen eyelids and red turgid mucosa. Photophobia may bespeak keratitis. Soon the watery tears become muco-purulent, matting together the lashes and even the lids, during the night especially, so that they must be sponged to get them apart in the morning. The exudate may accummulate under the lower lid, or may become flocculent, and usually flows down the cheeks causing matting and even shedding of the hair. Vesicles exceptionally appear on the conjunctiva; more frequently it becomes cloudy and opaque, and at points near the centre degeneration of the epithelium leads to the formation of ulcers no larger than pinheads, but extending into the cornea and sometimes perforating it so as to allow protrusion of the membrane of Descemet or the escape of the aqueous humor. The formation and extension of the ulcers are favored by the general debility, the rubbing of the eye with the paws, and the infection of abrasions, by pus-microbes. This infection may extend to the lining member of the anterior chamber and even of the posterior with panophthalmia, but, in the absence of perforation, internal ophthalmia is rare. When the ulcers heal, white

citatricial spots, or black points caused by the adhesion of the uveal pigment remain.

Digestive and Hepatic Symptoms. Anorexia and vomiting may usher in the disease. Buccal congestion, dryness, clamminess and fœtor are marked symptoms and there may be some yellowness of the mouth and eye. The patient is at first costive, but diarrhœa often sets in early, with tenesmus, much fœter, mucus, froth and even blood, also abdominal pain and tenderness. The abdomen is habitually tense and contracted. The alvine flux may rapidly exhaust the animal, or it may continue for a month in dysenteric form with intense fœtor, weakness, emaciation and exhaustion. Ulceration of the mouth, gums and rectum, invagination, prolapsus ani, jaundice, septic pneumonia, paralysis, chorea, convulsions, or cutaneous eruptions are occasional complications. The abdominal type of distemper is especially fatal. Even in its early stages debility, prostration and even drowsiness are marked features.

Cutaneous Symptoms. Skin eruptions are observed in the great majority of cases, at some stage of the disease, and may remain as a sequel for a time after apparent recovery. Friedberger and Fröhner note cases in which the high fever and skin eruption are the only prominent symptoms, and recovery may be looked for with some confidence. The lesions are most patent on white skinned dogs with short hair, and on the more delicate parts of the skin (abdomen, scrotum, perineum, inside of the thighs and elbows) but they may extend over the whole body and even encroach on the mucosæ. They vary much in different cases and stages. There may be punctiform reddish spots, changing to hard elevated papules, and, in the case of a certain number, to vesicles and even pustules. Some vesicles may be small and pointed, but more commonly they are rounded and flat, and as large as a lentil or small pea, when first formed, the contained liquid exudate may be clear and transparent, but often it is reddish or even violet. The individual vesicles tend to speedily burst, and dry up, but others appear, and thus the eruption will continue for weeks, the skin meanwhile exuding a sticky, greasy, offensively smelling exudate which mats the hairs together. Itching is usually slight, yet in given cases excoriations and sores are produced with considerable moist discharge. It tends to

spontaneous recovery when the general health improves and appears to be little affected by local treatment.

Nervous Symptoms. These are shown more or less from the beginning. The great dulness, depression, apathy and weakness, which usually ushers in the disease, are indications of this. Drowsiness may be early shown. Even the early nausea and vomiting may be largely central in its origin. In some cases, however, the brain symptoms are more active and violent. The dog is restless and irritable, getting up and moving from place to place, starting from sleep, yelping, snapping, with twitching of the muscles of the face or limbs, rolling of the eyes, and excessive heat of the head. Krajewski even describes rabiform paroxysms, depraved appetite, spasms, furious delirium, fawning, or threatening, and finally where rabies is familar it would have been more satisfactory if inoculation had been made from such cases with results negativing rabies. Epileptic attacks may appear at any stage of the disease. Chorea, tonic spasms, paresis and paralysis are on the contrary habitually late manifestations and often seem to be sequelæ determined by toxin poisoning of the nerve centers, or degenerations of their structure, Choreic movements may be confined to the head, or a limb, or they may affect the whole body. Tonic spasms often affect the neck, turning the head rigidly to one side. Among other nervous disorders may be named, amaurosis with dilated pupil and atrophy of the optic nerve, deafness, anosmia, and dementia.

Lesions. These vary as do the symptoms. The nasal pharyngeal, and faryngeal mucosæ show congestion, swelling, infiltration, ecchymosis with vesicles, pustules and ulcerations. They are covered by a foul muco-purulent exudate. The same condition may often be traced to the final ramifications of the bronchial system. In pulmonary cases, the lungs show inflammation, inflammatary exudation and consolidation, collapse, splenization, œdema, and even suppuration in points or large areas. The bronchial lymph glands and often the pharyngeal are enlarged, congested and may be suppurating. The pleura over the congested lung may be the seat of exudation and false membrane, and a bloody serum may occupy the pleural cavity. The heart may show parenchymatous degeneration.

In the digestive organs there may be buccal congestion, with degeneration and the desquamation of the epithelium and formation of more or less extended ulceration. The stomach shows similar congestions and degenerations, the ulcer appearing particularly on the summit of the folds. Together with the intestines this often presents numerous petechiæ and ulcerations, and is covered by a foul but often tenacious mucus. The agminated and solitary glands are usually swollen and infiltrated, and the mesenteric glands are swollen, congested and infiltrated.

The kidneys are often congested, and show points of blood extravasation and tissue degeneration.

The meninges of the brain and cord are often inflamed, with infiltrations, false membranes, and especially exudation into the subarachnoid and ventricles. Centres of congestion and softening have been noted in the brain and cord with embolism of capillaries and softening and degeneration of their endothelium. Nocard and others have noted a leucocytic infiltration of the perivascular lymph spaces. In old standing cases sclerosis is an occasional feature.

In nearly all cases there is marked emaciation and a very heavy offensive odor comes from the skin, the tissues, the contents of the bowels and the exudates on the respiratory passages.

Lignieres alleges that in the early stages, the specific bacillus is found in the blood and viscera, but that later it is only exceptionally found and that other bacteria (streptococcus, etc.,) usually take its place. In the nasal discharge it may be found at times, but in the more tardily appearing cutaneous vesicles its absence is the rule, and in the brain matter and meningeal and ventricular fluids, in cases of paralysis or chorea, it is not to be detected by culture. Hence the case cannot always be diagnosed by a successful search for, or culture of the germ, and hence also the frequently unsuccessful inoculations with the blood, tears, liquid of vesicles, and even the lung tissues or nasal discharges.

Prevention. Distemper, like any other contagious malady may be excluded from a city or district by the simple expedient of shutting out animals that bear the infection. From a kennel or pack of hounds, new arrivals should be quarantined for a fortnight, until danger is past, and should only be admitted after a

good soapy wash. All clothing, collars, brushes and other material that came with them should be thoroughly disinfected. Dog shows and other meetings are to be avoided as far as possible and any animal that has returned from one should be quarantined and all his belongings purified. Dogs that run at large should be carefully excluded from kennels where valuable dogs are kept, and from all possible contact with them. Even mice, rats, and birds have to be considered if the disease exists in the near vicinity. When the disease exists in a district a sound sanitation would demand the shutting up of all dogs on their owner's premises, unless carefully led on chain and prevented from coming in contact with other dogs.

When the disease has broken out in a pack of hounds, or a populous kennel, the dogs should be separated into small lots of 3 or 4; temperatures of all should be taken twice a day; any lot in which one shows a high temperature should be instantly removed to a safe distance and placed in quarantine; and the enclosure where they have been and all their belongings should be thoroughly disinfected. The enclosures where the sick are kept must be carefully quarantined so that no infection may escape on food, water, brushes, utensils, clothing, attendants, cats, vermin, or even birds.

The dogs that are still healthy shonld have spacious, well-aired dwellings, open air exercise (as much as possible in the fine season), good but not too stimulating food (in part at least fresh animal food), pure water, and protection against undue fatigue, cold, icy baths, especially when exhausted, rain or snow storms and cold stone or metallic beds. In the cold season artificial heat in the kennel is desirable.

Immunization may be sought in various ways based on the use of the toxins and antitoxins on the one hand, and of a weakened type of virus on the other. Bryce (1882) and others inoculated with the blood and pulmonary exudate, and produced in three-months-puppies, local swellings mainly, with subsequent immunity. The mortality from the inoculation did not exceed 10 to 15 per cent. These losses imply that in certain cases the material inoculated conveyed the microbe of the disease, and the survivors acquired all that immunity which comes from a first attack. In the cases that show local lesions only, it may be presumed

that few microbes or none were inserted, while the results came mainly from the toxins or antitoxins. This would be entirely in keeping with Lignieres' observation that the blood and pulmonary lesions often failed to furnish the pathogenic microbe, as tested even by attempts at artificial cultures. The protection secured from the antitoxins alone is short-lived, terminating with the elimination of these elements, while that coming from the action of the toxins on the leucocytes, and the stimulation by these to the production of defensive products, is much more lasting and in ratio with the quantity of the stimulus introduced and the profundity and duration of its influence on the leucocytes. This may partly explain the occasional early exhaustion of the immunity and the re-infection of the animal within a year after inoculation.

Physalix working in Chauveau's laboratory has found the best results from the use of weakened artificial cultures. He cultivated the microbe of Lignieres in peptonised bouillon having 6 per cent. of glycerine, and allowed successive cultures to rest (without reseeding a new culture fluid) after they have attained to their full growth. The strength of the culture is in inverse ratio to the period that elapsed between successive cultures, leading up to this one, and the first inoculation is made with the weakest product that will produce a very limited local swelling which is fully developed in 48 hours, and disappears in a few days. Three or four inoculations are made successively with cultures of gradually encreasing potency, after which immunity persists for years. It is to be understood, that an overdose will overcome the immunity at any time ; also that the passage of a culture of lessened potency through a dog or Guinea pig will raise it to its original virulence. Physalix operated only on young dogs, that had not shed their milk teeth, and injected 2 to 3 c.c. of the culture as a dose with only 2.5% mortality. Others had less success.

Treatment. To secure good results in the treatment of distemper every attention must be paid to good hygiene. An open air life in summer, and a roomy. clean, well aired, warmed building in winter are most important. Chills, foul air, and filth generally are to be carefully guarded against. Food should be moderate in amount, easily digested and nutritious. Milk is especially good (sweet, skim or butter-milk) ; then biscuit and

milk, or in patients accustomed to animal food, lean meat, minced, scraped or pulped. Warm baths are often advantageous, but they must be given with great caution to avoid chill.

Medicinal treatment is largely symptomatic. An excessively high temperature (104° F. and upward) may be met by warm baths, or antipyretics—quinine, acetanilid, salicylate of soda, antipyrine, phenacetin, or even damp compresses to the sides. As a rule, however, it is not well to continue such agents as acetanilid, antipyrine or phenacetin longer than is absolutely needful to reduce excessive temperature.

Attempts have been made to check microbian proliferation by antiferments, such as quinine, calomel, creolin, phenol, lactophenin and phenacetin. The tonic action of quinine specially recommends it but like all bitters it is obnoxious to a dog with a delicate stomach. Calomel is especially recommended by Fröhner as a gastric and intestinal antiseptic and its indirect action on the liver renders it valuable in many cases. Creolin and carbolic acid exhaled from saturated cloths tend to disinfect the air passages and give tone to the mucosa. My colleague, Dr. P. A. Fish, gave carbolic acid, 2 per cent., and afterward 4 per cent. in normal salt solution, and in doses of ½ to 1 drachm, subcutem once and twice a day. In the initial stages, it seemed often to be of great value, quieting the nervous excitement, improving the general symptoms, and in some instances apparently cutting short the affection. Trasbot recommended strong infusion of coffee, and others potassium bromide, ergotin, or better hydrogen peroxide in spray or as a draught. Iodine trichloride (1:2000-5cc) subcutem is claimed to abort an attack.

Eliminative treatment. As in all depressing contagious diseases, we must favor elimination of the toxins, and in this case without risking any material encrease of debility. Calomel (7 grs.), sodium salicylate or benzoate (4 grs.), sodium bicarbonate (7 grs.), potassium iodide (4 grs.), chlorate (7 grs.), or nitrate (7 grs.) may be cited. Digitalis (1 gr.), strophanthus, or caffeine are especially recommended by their power of encreasing the tone of the heart when that has become weak or exhausted.

For the *respiratory* symptoms we may employ the antiseptic inhalations already named, or, in place of these, iodine or sulphurous acid. The nervous cough may be met by syrup of poppies, or anise, by morphia, or codeia. (Recipe: morphinæ

hydrochloras 0.1 gram: aquæ amygdal. amar. 10 grams, aquæ distill. 150 grams. M. A teaspoonful three or more times a day. (*Fröhner*). As expectorants, ammonium chloride (5 grs.), or acetate, senega, or apomorphia ($\frac{1}{10}$ gr.), may be used. In vigorous subjects ipecacuan ($\frac{1}{2}$ to $\frac{3}{4}$ gr.), or antimonial wine (1 drop). Hydrogen peroxide in doses of a teaspoonful is often useful. Among counterirritants and derivatives the warm bath and cold compresses are especially valuable, or tincture of iodine, or camphorated spirit, or equal parts of aqua ammonia and olive oil serve a double purpose as furnishing at the same time an expectorant inhalant.

Special pulmonary complications must be dealt with on general principles as advised for the special diseases, bearing in mind always the profound prostration and the need to avoid depressing agents.

For *conjunctival symptoms* bathing with tepid water to soothe irritation and remove adhesions and crusts, may be followed by a drop of aqueous solution of pyoktanin (1:1000) under each lid, twice a day, or cocaine (3:100), or silver nitrate (½:100); may be used. Fröhner advises creolin (½:100); Cadeac, cresyl (½:100); Müller, mercuric chloride (1:2000) or boric acid (1:40). Other Collyria may be substituted (see diseases of the eye). In violent inflammations atropia will find a place and in ulcerations boric acid, silver, pyoktanin, calomel, red precipitate, or hydrogen peroxide. When irritation and rubbing are persistent a cocaine solution may be dropped into the eye every few hours. When the ocular troubles persist during general convalescence tonics with good nourishment and hygiene are demanded.

With *digestive symptoms* the attendant vomiting will usually have cleared the stomach of irritant contents. In the exceptional cases it may be unloaded by apomorphia ($\frac{1}{100}$ gr. subcutem), or ipecacuan wine a teaspoonful by the mouth. More commonly a check must be placed on persistent vomiting by bismuth nitrate (3 grs.), laudanum (5 to 10 drops), creosote (5 drops), or chloroform (5 to 10 drops), and small pieces of ice. A derivative to the epigastrium is sometimes useful. The food should be of the simplest and most easily digested kind, milk, meat-soup skimmed of fat, meat juice, scraped or pulped raw meat. Demulcents like gum water, slippery elm bark, or decoction of marsh mallow may

be resorted to, and in case of extreme irritability nourishment may be given by rectal injection. As vomiting may be kept up by irritants in the intestines a tablespoonful of tincture of rhubarb may be required to be repeated twice a day until relief is secured. Calomel and chalk (1:12) in grain doses will sometimes serve a good purpose. Also dilute hydrochloric acid in water (1:60) in doses of a teaspoonful with pepsin, gentian, quinine or nux vomica will often contribute much to restore tone and function. Septic intestinal fermentations may be met with beta-naphthol, naphthalin, (7 grs.), chloral hydrate (10 grs.) lactic acid (buttermilk), or salol (5 grs.). The attendant fœtid diarrhœa may demand in addition opium or silver nitrate (½ to 1 gr.). The gastric secretion is usually suspended so that it largely passes into the intestines unchanged. Of course it should not be used along with muriatic acid.

The *skin eruption* is usually considered of little consequence, or, by some, beneficial (Cadeac). If treatment is desired it may consist in dusting powders, demulcent soothing dressings and perhaps stimulating liniments as found under *skin diseases*. It usually disappears with the elimination of the toxins and the restoration of vigorous health.

For the *nervous symptoms* treatment must correspond to the morbid phenomena. Extreme prostration may demand diffusible stimulants, coffee (7:100), sherry 1 dr., beef tea, meat extract, ether, camphorated oil subcutem, strychnia, electricity. Spasms and other indications of congestion may be met by cold to the head, and inhalations of ether, followed by potassium or sodium bromide (8 grs.), sulphonal (20 grs.), trional (15 grs.), chloral hydrate in mucilage, or hypnal (15 grs.). Paralysis must be met by tonics, stomachics, easily digestible, rich food, and good hygiene. Pepsin, muriatic acid, nux vomica (½ gr.), arsenite of soda solution (5 drops), arsenite of strychnia ($\frac{1}{20}$ gr.), orexin (3 grs.), strong coffee infusion, wine, and electricity may be tried, in addition to stimulant liniments. Chorea must be treated on the same corroborant plan. Cold douches after which the patient is carefully rubbed dry are sometimes successful, (see Chorea).

During *convalescence* and in all cases of debility and anæmia a similar corroborant treatment is demanded. Pulped raw meat, rich soups, stomachics, tonics including the preparation of iron,

and in extreme cases transfusion of blood or a normal salt solution may be resorted to.

In *cats* a parallel course of treatment may be pursued, allowance being made for the smaller size of the animal and the great susceptibility of the feline patient to phenol.

INFECTIOUS BRONCHIAL CATARRH. BENCH-SHOW DISTEMPER.

Under this name Glass describes an affection, milder than the usual distemper, but showing similar lesions and demanding an equivalent treatment. It is not self-limiting the same patient having suffered twice in the same year (an occurrence which is occasionally seen in distemper. The incubation is 3 to 5 days. Diarrhœa is invariably present from the first, and the fæces slimy and at the end of a week slightly bloody. The affection is characterized by the predominance of the digestive disorder, the absence of skin eruption, the free shedding of the hair in long coated animals, the ulceration of the gums, tongue and lips, and the low mortality.

Bacteriological research must be invoked to determine whether this is only a form of distemper or if it is one of a group of diseases which have hitherto been known by that name.

EMPHYSEMATOUS ANTHRAX.

Definition. Historic notes. Geographical distribution. Animals susceptible: Young cattle after weaning, sheep, goats, horses, asses and white rats, have local swelling; dog, cat, pig, bird and man immune. Immune animals succumb if injected with lactic acid, or proteous vulgaris, or violently exerted (sarco-lactic acid). Causes. Bacillus anthracis emphysematosa, 3 to 10μ by 5μ, stains violet with iodine, anærobic, sporulate in living body, hence seen as rod, club, and round, spore. Lives in exudate, not in blood nor on surface. Table comparing with anthrax bacillus. Vitality: resists drying, cold, 98° F., weakened by 139°, sterilized by 212° F for 20 minutes, by strong antiseptics. Lives in dense clay, hard pan, and water logged soils holding little oxygen. Accessory causes: lactic and other organic acids, overwork, potash salts, alcohol, salt, proteus vulgaris, micrococcus prodigiosa, low condition, debility, plethora, chills, change to warmth, youth, melting snows, freshets, drying of wet lands. Symptoms:

incubation a few hours, diseases 12 to 70 hours. Hyperthermia, swelling in loose connective tissue, shoulder, quarter, arm, thigh, neck, trunk, palate, base of tongue, pharynx, tender point, rapidly enlarges, spreads, crepitates, percussion resonance, finally cold, insensible, withered. On incision black, bloody pulp, or frothy. Peripheral gelatinoid exudate. Subsidiary lymph glands enlarged. Cases with deep seated exudate. Diagnosis: from malignant œdema and anthrax. Lesions: early decomposition, bloating, in swelling blood extravasations with gas bubbles and lymph exudate, muscle beneath dirty brown or black, breaking down when pressed, shows waxy or fatty degeneration, and many leucocytes and cell forms. Lymph glands and plexuses blood gorged. Extravasation may be in internal organs. Liver congested. Spleen rarely enlarged. Treatment: Chloride of iron internally, ammonia iodide and ol. terebinth externally. Scarify and use hydrogen peroxide or potassium permanganate. Antitoxins. Prevention: drain and till soil, apply quicklime to muck, exclude new animals just from infected districts, disinfect buildings, close infected wells and streams, seclude the sick, burn, cook or dissolve carcasses, or fence graves. Bleeding purgation, diuresis, uniform good feeding, setoning. Immunization by heated and sterilized culture; by toxins passed through a porcelain filter; by minimum dose intravenously; by injection into trachea; by inoculation on tip of tail; by inoculation with Pasteur weakened virus; by heat sterilized virus.

Synonyms. Symptomatic Anthrax; Black Quarter; Quarter Ill; Black Leg; Rauschbrand; Charbon Symptomatique.

Definition. An acute infectious bacteridian disease manifested by hyperthermia, lameness, and a localized, hot, painful swelling on the shoulder, quarter, leg, neck, trunk or elsewhere, tending to emphysema, and gangrene and when incised showing black extravasated blood, clotted or frothy.

This affection was long confounded with anthrax proper, but was differentiated by the observation of Wallraff (1856), Boulit-Josse, Vernant, Pfisterer, Feser, and others and finally Bollinger in 1875 found the motile bacillus. Arloing, Cornevin and Thomas (1879–84) thoroughly substantiated this position, and devised a method of immunizing by inoculation. Feser had however seen the motile sinuous rods in the exudate as early as 1860, and even produced the disease by the inoculation with mud from infected Alpine Pastures.

Geographical distribution. Emphysematous anthrax prevails in limited areas, and particular buildings in Europe, Asia, Africa, Australia and America, and in all climates from the tropics up to the sub-Arctic. It is however most prevalent in spring, summer and autumn. It is not uncommon at the breaking up of the winter snows. It is especially prevalent on damp, undrainep

land or on such as has dried up in the heats of summer, and has become less prevalent in many localities in connection with drainage and careful cultivation.

Animals susceptible. The disease is especially common in young cattle, from three months to four years of age. Calves fed on milk are rarely attacked, being in a sense carnivorous and sometimes immunized by toxins from the dam. Cattle over four years usually escape, having already become immune if kept in an infected locality. If brought from a non-infected place they are, at any age, as susceptible as the young. Sheep and goats contract the disease only exceptionally, but like the Guinea pig are easily infected by inoculation. Horses, asses and white rats have only a circumscribed swelling in the seat of inoculation. Ganter saw violent general symptoms as well in a horse, and foals and buffalo calves sometimes contract it casually. Carnivora and omnivora, (dog, cat, pig, bird, man), and the rabbit are virtually immune. Marck saw violent pharyngo-laryngitis and exudates in adjoining muscles, of the pig, and from this inoculated Guinea pigs successfully.

Immune animals may, however, be made to succumb under special treatment. In a rabbit inoculated at the same time with the microbe of black quarter and proteus vulgaris, or micrococcus prodigiosus, or if injected with a little lactic acid, the disease develops promptly and fatally. Over-exertion, producing sarcolatic acid, will also lay the system open to attack. The reduction of the vitality and resistance of the muscle of the rabbit by a contusion, bruise or lacerated wound, or by injecting it with acetic acid, potash salts, alcohol, or common salt, will render the inoculation pathogenic. Again, the introduction of the black quarter microbe into the aqueous humor of the rabbit, where there are so few defensive leucocytes, entails an active proliferation and a fatal result.

Causes. The essential cause of emphysematous anthrax is a rod-shaped germ, variously known as **bacillus anthracis emphysematosa, bacillus Chauvæi, Rauschbrand bacillus.** This is a rod-shaped microbe, with rounded ends, found singly or connected in pairs, or very short filaments. The bacilli are 3 to 10μ long, by .5μ broad, or when sporulating, 1.1 to 1.3μ. They form spores even in the body of the affected animal, often assuming a club shape by reason of the spore formation near one end. If the spore develops in the center they appear fusiform.

They take aniline colors readily, and iodine slightly, assuming in the last case a violet tint. The staining is unaffected by heating in melted balsam. The bacillus grows readily in ordinary culture media, (peptonized gelatine, bouillon, milk, etc.), but, being anærobic, only under the surface in stick cultures, or under a neutral gas or vacuum. It grows most rapidly at a temperature of 36° to 37° C. but also as low as 15°C. The bouillon at 37° C. becomes milky and opaque in 24 hours, and later it clears up, the microbes being precipitated as a fine white powder. In gelatine cultures liquefaction takes place in three days, and in twenty days the whole mass may be dissolved and the microbes precipitated to the bottom. Spores may form in the living body and as these are set free by the granular degeneration of the bacilli, the virulent exudate and cultures usually show the microbe in three different forms: 1st, the straight, motile bacillus of one thickness throughout its whole length; 2d, club-shaped or fusiform bacilli, the thickening of the end or median part being due to the endogenous formation of a refrangent spore or spores; and 3d, the free refrangent spores which have been set free by the degeneration and destruction of the sporulating bacilli. The microbe is not found on the surface of the living animal, nor in the blood, for in both the supply of oxygen is too abundant; it forms its colony under the skin, in the tissues, and above all, in the mass of extravasated blood or gelatinoid exudate which its irritation has produced and where air is lacking.

The most marked differential features of the microbes of anthrax and emphysematous anthrax are contrasted in the following table :—

Bacillus Anthracis.	Bacillus of Emphysematous Anthrax.
5 to 20μ × 1.25μ.	3 to 10μ × 0 5 to 0.6μ
Ends square or cupshaped.	Ends rounded.
Occurs singly in the living body.	Often in pairs or threes in body.
Long filaments in cultures.	No long filaments.
Nonmotile.	Motile (sluggishly).
Ærobic.	Anærobic.
No spores formed in living body.	Sporulates in living body.
Sporulates in air; in surface soil.	Sporulates in vacuo; deep in soil.
Bacilli only.	Bacilli; sporulating bacilli; free spore.
Multiplies freely in blood stream.	Dies in blood stream unless charged with toxins.
Rabbit very susceptible; man less so.	Rabbit, pigeon and man immune.
Produces no gas.	Gas producing.
Inoculation swelling very restricted.	Inoculation swelling very extensive.
Bacillus destroyed by putrefaction.	Not destroyed by putrefaction.

Resistance of bacillus of black quarter to physical and chemical agent. The microbe is possessed of great vitality. Thoroughly dried at a temperature of 95° F. it retains its virulence. The spores may be preserved indefinitely in dry soil, buildings, fodder, litter, harness, etc. Cold is equally harmless to it. It has been exposed to a temperature of 98° F. below zero without losing its virulence. Its virulence is lessened by exposure for an hour to 139° F., and is sterilized at a temperature of 212° for twenty minutes. The dried spores are virulent after six hours of the boiling temperature but are sterilized at 230° F. if maintained for the same length of time. Diffused in water the virus is sterilized in thirty-five minutes at the boiling temperature. Some waters at ordinary temperatures destroy the virulence in twenty-four hours ; others not for many months. It is destroyed by the more potent disinfectants, mercuric chloride (1 : 1000), silver nitrate (1 : 100), acid salicylic (1 to 2 : 100), acid carbolic (2 to 4 : 100), copper sulphate (20 : 100), boric acid (20 : 100), muriatic acid (1 : 2). Quicklime, copperas, zinc chloride, sulphuric acid and an alcoholic solution of phenol have proved unsatisfactory.

In clay soils, hard pans, waterlogged soils, and in some that are over manured so that the atmospheric air is excluded, it may be preserved indefinitely. Feser, Gotti and others have produced the disease by inoculating with the washings of infected marshy soils, and this is doubtless a common source of casual cases of the malady.

Accessory causes are important. The predisposing influence of lactic acid, of other organic acids, and of overwork have been already named. Potass salts, alcohol, common salt and the products of proteus vulgaris, or micrococcus prodigosus increase the susceptibility. Low condition, debility, or suddenly induced plethora have a similar influence. Sudden changes of weather, chills, and particularly the access of hot weather in spring, when the animal is changing its coat lays the system open to attack. Youth, after the period of suckling, and under three years old, seems to increase the predisposition, though this is largely the result of the absence of a previous exposure. Then impermeable, clay, wet, marshy soils, or those charged with organic matter are conditions of the presence of the microbe. It often appears in spring in connection with the melting of the winter's snows, the

occurence of freshets, and the washing out of soil infection which would otherwise remain buried. Also in advanced summer and autumn when swamps, ponds, basins, deltas, river bottoms, etc., are drying out and furnishing pasture. Pease records its great prevalence in the rainy season in the swamp districts of the Punjab and North Western provinces of India, and the same is largely true of our Gulf coast states.

Symptoms. Emphysematous anthrax develops suddenly, the incubation in experimental cases, lasting only for a few hours, (casual cases 1–3 days, Friedberger) and the whole course of the disease does not usually exceed ½ to 3 days. The local swelling may be the first observed symptom or there may be first febrile disturbance followed by the local swelling. *The swellings* show where the connective tissue is loose and abundant as on the shoulder, quarter, arm, thigh, neck, face, or trunk, and practically never where the areolar tissue is very spare and dense as on the end of the tail, or ear, or on the limb below knee or hock. They sometimes form on the palate, base of the tongue, or pharynx. The muscular system is especially liable to suffer, the looseness of the texture and the presence of lactic acid making a particularly favorable field for the propagation of the microbe. The comparative absence of muscle in the region below the metacarpus, the tail and ear is an important cause of immunity.

The *swelling* is at first very small and tender, but it increases rapidly, and in a few hours may extend to one, two, or three feet in diameter. At first smooth, rounded, pitting on pressure and destitute of crepitation on handling, it becomes softer and less sensitive and when pressed or kneaded it gives a crepitant sensation and sound, or it even appears to gurgle. When percussed the resonance is drumlike. Finally, the skin may become cold, insensible, and withered like a piece of parchment. When incised the tissues are found to be gorged with blood, and of a black or dark red color; they break down under pressure into a bloody pulp, and from the wound flows a bloody fluid which may be red in the early stages, black in the advanced, and frothy in the latest. Where the connective tissue is very loose and abundant, the bloody extravasation is surrounded by an extensive straw colored œdematous infiltration. The swelling is sometimes single, but more frequently several appear and become confluent. The lymph glands in the vicinity become greatly enlarged.

Fever is a constant condition as the swelling advances and sometimes it precedes the local engorgement. There is erection of the hair, with, it may be, distinct shivering, recurring again and again. Then general stiffness, dulness, prostration, loss of appetite and rumination, accelerated breathing sometimes attended by a grunt or moan, cyanosis and rapid pulse. The temperature usually reaches 104° F., and many rise to 109° F. The breathing becomes more and more labored and plaintive, colicy symptoms may set in, the prostration advances to complete adynamia, the patient can no longer stand, the temperature drops to 100° F., or 98° F., and death supervenes in from eight hours to two days from the first sign of illness.

In some cases the swelling may be invisible because it is situated deeply or it may perhaps be entirely absent, and the constitutional symptoms are the only ones observed.

Diagnosis. From *malignant œdema*, which it resembles in producing gas and crepitating tumors, emphysematous anthrax is distinguished by the greater length of the microbe, by its formation of spores at the pole and not in the centre of the bacillus, by the more sluggish motions of the germ, by the restriction of the germ to given infected districts instead of being generally diffused as in malignant œdema, by its not attacking man, rabbit, nor pigeon, which are subject to malignant œdema, by its deadly action on mature cattle, which are usually immune from malignant œdema, and by the abundant blood extravasation on the swelling.

From *anthrax* it is distinguished by the motility of the bacillus, by its polar sporulation and club shape, by its rounded ends, by its absence from the blood in the earlier stages, by the presence of gas and crepitation in the swellings, and by the deadly action of the infection on Guinea pigs, but not on rabbit, man nor pigeon. Anthrax is easily inoculable on a cutaneous sore or intravenously, whereas emphysematous anthrax is not.

Lesions. The carcass is liable to be bloated with gas and a reddish, frothy liquid often escapes from mouth nose and anus. Gas is particularly abundant in the substance of the tumor, and the skin covering it may be dry and crackling. An incision made into the swelling exposes a mass of blood extravasation and lymph exudate, the blood predominating in the centre so that it may appear clotted and black, and mixed with gas bubbles,

while the yellowish lymph forms the periphery of the tumor, yet streaked more or less with blood, or even pink throughout. The abundance of gas is usually in inverse ratio to the amount of œdema. The muscles beneath, or surrounded by an exudate, are of a dirty brown or black, and are disintegrated so as to break down readily under pressure of the finger into a blackish pulp. They are infiltrated with gas, crepitate under pressure and assume a golden yellow color on exposure to the air. The gas is comparatively inodorous immediately after death, being mainly carbon dioxide and carbide of hydrogen. Later it may show distinct and even offensive odor, from the formation of hydrogen sulphide, or lactic acid. The muscular fibres are easily teased apart, and show under the microscope masses of blood globules, leucocytes, lymph cells, free nuclei and granules, with, in some points, fatty or waxy degeneration of the fibres, or granular masses that are stained black by osmic acid. The bacillus is present in large numbers, and this with its absence from the blood immediately after death becomes characteristic. The lymph glands near the swelling are usually enlarged and gorged with blood. The lymph plexuses and vessels contain bubbles of gas.

The swellings may be subcutaneous, or submucous in the tongue or pharynx, but they occur also in the pleuræ, lungs, heart, pericardium, mediastinum, the peritoneum, the sublumbar connective tissue, and even the walls of the stomach or intestine. It is not uncommon to find a pink effusion into one of the serous membranes. The liver is usually hyperæmic, as may be also the kidneys, but the spleen is rarely enlarged. In this and in the integrity of the blood globules this affection differs from anthrax.

Treatment. This disease is so often speedily fatal, cutting off its victim in eight hours, often during the night, that no opportunity is allowed for treatment. Even in those that survive for two days, the affection must always be looked on as exceedingly grave, and as little amenable to treatment. Yet much depends upon the patient and the country. Dr. Phares in Mississippi found that it yielded readily in many cases to ½ oz. doses of tincture of chloride of iron every four hours, and a local application composed of equal parts of tincture of iodine, aqua ammonia and oil of turpentine. Galtier tells us that recoveries are frequent in Algeria, while they are rare in France. Tisserant gives the French

recoveries as 2 per cent. It is probable that in districts and countries where the malady is all but ubiquitous, the surviving animals are racially immune, or they have been largely exposed and in some degree virtually immunized at an early age.

Wallraff mentions a success from applying a tight ligature around an infected limb, above the seat of the tumor and freely scarifying the latter so as to liberally admit the air. For swellings elsewhere, scarifications and the free application and injection of peroxide of hydrogen or potassium permanganate (2 to 3:100) would be rational treatment. The same agent might be given by the mouth in doses of 2 or 3 ozs. at frequent intervals. Antiseptics and tonics have been freely employed, including phenol, salicylic acid, sodium salicylate, potassium iodide, quinia, alcohol, phosphorated oil, ammonia acetate, and as an eliminant soda sulphate, but with no very good result. Locally, scarification, antiseptics, and caustics, have been employed.

Another line of treatment which deserves to be further exploited is the use of antitoxins on infected animals. An immunized animal may be again and again inoculated at intervals of a week or two until it has been stimulated to produce antitoxin in large amount. Then after three weeks interval its blood serum or blood, may be sterilized by heat, the resulting coagulum washed in distilled or boiled water, and filtered, and the filtrate injected subcutem on the infected animal.

Prevention. This is most effectively secured by sanitation of the soil and buildings. Thorough drainage to secure perfect and constant aeration leads to destruction of the anærobic germ, or the suspension of its pathogenic quality. Thorough culture contributes largely to this sanitary aeration, while baking of the surface counteracts it. When thorough drainage is impossible it may be desirable to subject the land to gardening or to the production of crops that are to be used for human consumption and not for domestic animals. Kitt's suggestion, to soil, cattle on hay, produced on such lands, and to exclude from the infested lands all animals that by wounds or sores near the feet, or by raw gums from shedding of teeth, furnish infection atria for the poison, is insufficient, as stalled cattle occasionally suffer.

When an open porous soil maintains the infection by reason of the presence of an excess of decomposing organic matter, that

may be largely remedied by a free application of quick lime. This hastens the decomposition of the organic matter and after a year or two, when that has been largely disposed of, the good effects may be expected.

An important measure is to exclude from fairs, markets, and above all from clay or other dense wet soils into which they might convey the germs, all animals brought from infected soils.

Disinfection of the buildings where diseased and infected animals have been is an essential measure. Wells and streams receiving the drainage of infected lands must be carefully avoided.

Diseased animals must be carefully isolated, and all their droppings, and products of every kind disinfected.

The carcasses are best cremated or rendered under superheated steam under pressure, Solution in sulphuric acid may be employed. If none of these is available they may be deeply buried in dry porous soil, well apart from any risk of drainage into wells or water supplies. The area occupied by the graves should be fenced in so that no cattle nor sheep can gain access to it, and any vegetation grown on the graves should be burned. The danger of the germs being raised to the surface by soil water or earth worms must be recognized and any consequent evil guarded against. The carcass should not be cut open but buried in the hide, or if the latter is preserved it should be treated with a chloride of lime solution. If a carcass is opened for scientific purposes, great care must be taken to avoid the distribution of the bacillus in soil appropriate to its preservation. The meat should not be preserved for human consumption unless it has been cooked under pressure at a temperature of 240° F. The object is not to destroy any poison which would be fatal to man, but rather to prevent the spread of the spores on new soil and the extension of the area of infection.

The reduction or prevention of sudden plethora was formerly availed of to lessen the number of victims and it is well to still bear in mind that this has an appreciable though limited effect. As a means of reducing plethora a free bleeding was resorted to when the period of yearly prevalence approached, and no less when the disease had already appeared in a herd. I can mention an instance in which infection was carried on the fleam from the first animal bled (the sick one) and caused the fatal infected swelling

around the phlebotomy wound in the next seven animals operated on. Another objection to phlebotomy is the tendency to a rapid reproduction of blood, which the depletion brings about, and the supervention of a greater danger than before, in the course of a month or more. Purgatives and diuretics are somewhat less objectionable in this sense. Careful feeding to keep the animal constantly in good condition does something to obviate sudden plethora and its attendant dangers, and thus an allowance of grain or linseed cake through winter and early spring, or when the pastures are bare, will bring the animals through in fine condition, and ward off the danger that comes from a sudden access of rich aliment.

Another measure was the insertion of a seton in the dewlap. The theory was to counteract plethora but the benefit probably came rather from the formation of an actively granulating wound, which came in contact with the ground and received the bacillus but in which the abundance of air, and of active leucocytes checked the propagation of the germ and the occurence of a fatal infection. A certain grade of immunity was the natural result in many cases.

Immunization. As the first attack of emphysematous anthrax secures for the subject of it immunity against a second, we are furnished with a reasonable basis for the practice of artificial immunization. This has been attained by a variety of methods, the essential feature of each being the subjecting of the system of the animal to be treated, to the action of the toxins of the specific bacillus.

1st. A culture of the bacillus made in the thermostat at 42 C. (107.6° F.) so as to prevent the formation of spores is then sterilized by heating to 100° C. for one hour and then injected subcutem in a dose of 2 drams, to be repeated on the second day. This, like all the other methods named should be done by some one accustomed to bacteriological manipulation and the sterlization completed by superheating the neck of the vessel containing the mixture. Any germs escaping on the hands, instruments or other objects used will prove fatal in spite of all the appearance of precautions.

2d. Roux sterilized his cultures by filtering them through a porcelain (Pasteur) filter and using only the filtrate for injection. This requires even greater precaution in manipulation as what is

left in the filter is most virulent, and must be thoroughly sterilized to obviate dangers from its dissemination.

3d. Intravenous injection of a small quantity of virus, containing but a few bacilli produces no local swelling, but only a slight temporary hyperthermia and permanent immunity. The greatest care is necessary in the manipulation, to prevent any contact of the bacillus with the subcutaneous tissues or the walls of the vein. The virulent exudate swarming with bacilli is taken and a drop or two added to a normal salt solution, which is diluted and shaken in a stoppered bottle, until each drop contains but one, or at most two bacilli. Then the hands having been thoroughly washed with soap and warm water and rinsed in a 5 per cent. solution of carbolic acid, and the instruments having been boiled, the vein is raised as for bleeding, and penetrated by a short cannula and trochar, which after boiling has been dipped in the carbolic acid solution, the trochar is withdrawn, and the nozzle of the syringe containing the virulent solution is inserted through the cannula, so that its point is free in the centre of the blood stream, into which a few drops of the virulent solution are discharged. The nozzle is left in place for a few seconds to ensure the washing of any infecting matter from its point, when it is withdrawn, followed immediately after by the cannula. Great care should be taken to avoid any scratching of the inner coat of the vein with the cannula, trochar or nozzle.

4th. Another method of immunizing is by the injection of the virulent liquid into the trachea, and bronchia. This appears to bring it so directly in contact with the blood, that the microbes are destroyed as rapidly as if it were introduced into the blood stream direct. The injection is made between two tracheal rings, the manipulation being essentially the same as in the cases of the vein, the tissues being first perforated by a sterilized cannula and trochar, and the sterilized nozzle subsequently inserted through the cannula.

5th. Inoculation into the tip of the tail can be successfully employed, the coldness of the region and the scantiness of the connective tissues preventing any dangerous increase of the bacilli in the cooler season. In the heat of summer, however, this is to be avoided as dangerous. The tail is first washed with soap and water followed by a 3 per cent. solution of phenol. It is then

punctured with a fine trochar or needle, (sterilized), within two inches of the tip and in a downward direction and the instrument is moved slightly from side to side so as to form a small sac, and is then withdrawn. The sterilized nozzle of the hypodermic syringe is now inserted in the opening and a few drops of the virus injected into the sac. When the nozzle has been withdrawn the thumb may be placed on the external orifice and the end of the tail manipulated to diffuse the virus in the connective tissue. This is usually followed by an insignificant swelling, and a slight rise of temperature. Should the swelling exceed the size of a duck's egg or if others appear higher up on the tail, they may be freely scarified and covered with a carbolic acid bandage. Or the tail may be amputated above the highest swelling and the stump treated with antiseptics.

6th. The virus prepared by the Pasteur institutes, that of Arloing, Cornevin and Thomas, is the most extensively employed. Forty grammes of the diseased muscle are dried rapidly at 32° C (90° F.) and triturated in 80 grammes of water. This is divided in 12 equal parts and put on plates in two thermostats, six at 100° C. (212° F.) and six at 85° C. (185° F.) where they are kept for six hours, when it forms a dry, brownish powder. One tenth of a gramme (1½ gr.) of this powder is dissolved in five grammes of distilled or boiled water and will furnish ten doses. The animal to be protected is first injected in the tip of the tail or elsewhere with the virus prepared at 100° C., and ten days later with that prepared at 85° C.

By the use of this method in hundreds of thousands of animals on infected lands, the mortality has been reduced to less than one tenth of its former amount. It is attended by the one danger which is not always duly appreciated, that unless its use is restricted to herds on ground that is already infected, it endangers the infection of new districts. The spores are not absolutely sterilized at 85° C. Arloing and Cornevin and later, Nocard and Roux have shown that the addition of lactic acid to the liquid which has been weakened for inoculation, restores it to its former virulence, making it a most deadly agent. Galtier says that the virus weakened by heating to 100° C. for seven hours until it will no longer kill a mature Guinea pig, will still kill a new born Guinea pig and acquire all its original virulence in the act. Also that the

injectton of large enough doses will not only kill the full grown Guinea pig, but at the same time restore the microbes to their former virulence. While recognizing the great economy of the judicious use of such weakened virus, we cannot but condemn the reckless sale by the Pasteur institutes of their products, to be used on animals on all kinds of lands, the uninfected as well as the infected. A great and valuable prophylactic measure should not be used in such a way as to increase the area of prevalence of the disease which is to be prevented, and also the yearly demands for more of the preventive agent. This may appeal to the business instinct, but this should ever be held subordinate to sanitary considerations. The danger might be avoided by making the state the sole distributor of such prophylactic agents, but in any case their use should be forbidden, and as far as possible prevented, upon dense and wet soils that are not yet contaminated by the bacillus.

7th. Kitt secured immunization by inoculating once only, with dried virus which had been subjected for six hours to steam at 100° C.

8th. In different outbreaks, I have taken the blood from the sick animal, or one that has just died, and heated it for over one hour in a water bath, at 100° C., then broken up the coagulated mass in well boiled water, filtered the liquid and used the filtrate for inoculation in doses of 2 drams, repeated the second day. Great care is taken in keeping the whole mass at 100° C. for the requisite length of time ; then in heating the upper part of the vessel, which was above the contents and the water so as to char anything adhering to it ; to see that hands, instruments, and all articles used have been thoroughly sterilized ; and to dip the hypodermic nozzle in carbolic acid before each injection.

It is not claimed that this method is perfect, since severe, advanced cases may have bacilli and even spores in the blood, and the latter would not be sterilized but only weakened. It has, however, several manifest advantages that may be held to more than counterbalance this danger.

a. It almost infallibly secures the toxins of the disease prevailing in the particular herd, thus escaping the danger of using the weakened virus of emphysematous anthrax on some other disease (anthrax, Wildeseuche, Barbone, etc.), which has been

mistaken for it, and which may not be prevented by this purchased product.

b. During life the blood of emphysematous anthrax is usually free from the microbe, and even where that is present it is liable to be in very small numbers, so that we secure either the pure toxins, or if a few germs are present they are so scanty, that, weakened as they are by heat, they are without danger to the animal operated on. I have never had occasion to note evil results.

c. There is no danger of the spread of the bacillus to new territory, as we secure the material from a herd in the already infected territory, and use it only on the animals on the same land.

The certainty of results with this method, and the comparative absence of danger of injury to the animal operated on, and of all risk of the extension of the area of infection appeal to me so strongly, that I would not willingly resort to the purchased products, except where it proves impossible to secure the virus on the spot.

ANTHRAX.

Definition. Synonyms. History and geographical distribution. Etiology: Bacillus anthracis. Susceptible animals: small rodents, sheep, horse, camel. ox, goat, deer, stag, man, swine, dog, white rat and bird when chilled, frog when heated; young most susceptible, races long exposed to infection are least. Soils, wet, dense, impermeable, basins, swamps, rich river bottoms, deltas, rich meadows, below tanneries, alkaline soils, wells with surface drainage; season: wet, hot and dry, late summer and autumn; flies; infected buildings, harness, vehicles, fodder, litter, butchers' knives and wagons, surgical instruments; insolation; privation of water; plethora, starvation; overwork, exhaustion. Bacillus anthracis; rod, non-motile, 5 to 20μ by 1 to 1.5μ, square ends, isolated in blood, often filamentous in cultures, sporogenous, ærobic, stains easily in aniline and iodine, killed at 131° F. (spores at 203° to 282° F.), action under chemical disinfectants, or septic ferments; air favors sporulation and survival; in shallow graves, water. Infection atria, ingestion, inhalation, inoculation, insects, placenta. Forms: fulminant, internal, febrile, local,—external,—gloss-anthrax, pharyngeal, hæmorrhoidal, subcutaneous. Lesions: blood normal in fulminant, dark, diffluent, crenated or disintegrated globules in prolonged cases, tissues brown or yellow, heart pale; liver enlarged, softened, pale, hæmorrhagic; spleen enlarged, blood-gorged, ruptured; lymph

glands hyperæmic; serosæ congested, petechiated, hemorrhagic; lesions embolic and like the blood swarm with bacilli. Toxins, ptomaines. Incubation 1 to 6 days. Symptoms: *internal cases*: hyperthermia, constitutional disorder, mucosæ dusky, brownish, yellowish, bleeds, bloody urine, rectal mucosa congested, blackish, colics, pharyngeal anthrax, blood diffluent, black, broken down red globules, bacilli, abortions, death in 12 to 48 hours; fulminant cases in cattle and sheep. Local (external) anthrax, cutaneous, swellings; gloss-anthrax; pharyngeal, hæmorrhoidal; sheep and goats; horse; swine; dogs; cats; birds. Differential diagnosis: deductions from symptoms, conditions of blood, animals attacked, environment or conditions of life, presence of the bacillus. Examination for bacillus. Post mortem lesions; gelatinoid, bloody exudate, petechiæ, blood-gorged spleen, lymph glands, and liver, diffluent blood, bacilli in capillaries. Inoculation intravenously. Cultures. Prognosis. Mortality often 70 per cent.

Definition. Anthrax is an acute infectious bacteridian disease occurring casually in the herbivora and omnivora and, under favorable conditions, communicable to carnivora, birds and batrachians, Its special features are the presence of the *bacillus anthracis* in the diseased parts, the destruction of red globules, the arrest of hæmatosis, the occurrence of capillary embolism, extravasations and exudations, and of necrotic processes in the affected parts, and a sanguineous engorgement of the spleen.

Synonyms. Malignant pustule; Splenic apoplexy; Splenic Fever; Charbon; Miltzbrand; Woolsorter's Disease; Malignant Carbuncle; Contagious Carbuncle.

History and Geographical Distribution. As anthrax prevails in damp, undrained lands where agriculture is backward, it is not wonderful that it can be traced to near the dawn of human history when the whole race lived under primitive conditions. Moses records its ravages on the bottom lauds of the Nile (Ex. ix. 9), Homer, on the plains of Troy (Iliad, Bk. 1st), Ovid, Plutarch, Dionysius, Livy, Lucretia, Columella, Virgil, Pliny and the Arabian physicians all show a familiarity with a disease of this nature. Later, Heusinger collects evidence of its prevalence in certain areas in all parts of the world from the equator to the Arctic circle. The mortality was often very high, thus Kirchner records the death of 60,000 people in a single epidemic in 1617, in the vicinity of Naples, Placide-Justin, that of 15,000 in St. Domingo in six weeks in 1770, and on the Russian and Siberian Steppes it is not uncommon for hundreds of thousands

of domestic animals and thousands of human beings to suffer in a single year.

The geographical distribution of the disease is largely influenced by soil and climate. On, dense impermeable clays and hardpan subsoils, on river bottom lands, dried lake basins and deltas, rich in organic matter, and with the air driven out by water or gaseous fermentation products, the germ is preserved and propagated if once introduced. Thus it is common around the mouth of the Nile, and along the occasionally inundated banks of the Vistula and Danube, the Spree, Oder, Elbe, Rhine, Eure, Loire, Seine and Marne, and in England in the Fen district. On the rich, undrained, black soils of Siberia it is extremely prevalent and fatal. In the rich Genesee Valley, N. Y., the writer has seen 200 cattle in one herd and three human attendants attacked in the course of a fortnight, and in different meadows receiving the drainage of tanneries, the affection prevails every summer and autumn. It is much more prevalent in the rich lands of the Southern States and a widespread and deadly epizoötic prevailed in Louisiana in 1896. Where the soil is favorable, the germ may be preserved indefinitely, even in mountainous districts, and near Los Angeles, Cal., where the disease was introduced in imported sheep some years ago it has become permanently domiciled, on the dry ranges which have moist lands (Cienegas). When an outbreak occurs, the herds or flocks are usually moved to higher soil, and the carcasses being left unburned and unburied the infection is spreading year by year (McGowan, Morrison).

Etiology. Nothing is more certain than that the disease is due to the introduction into the blood or tissues of the bacillus anthracis or its spores. These microbes are always found in the anthrax lesions and in the blood of the victim in the advanced stages. When grown in bouillon cultures to the hundredth generation they retain their virulence unabridged and determine the same lesions in the animals inoculated. When the infecting culture has been passed through a Pasteur filter the virulence is lost with the removal of the bacilli. The fresh anthrax blood containing bacilli but no spores when subjected to compressed oxygen (50 atmospheres) becomes non-infecting. The same liquid when boiled proves non-virulent.

Certain conditions, however, contribute to the propagation and reception of the bacillus and these may be considered as accessory causes.

Animals susceptible. The receptivity of the animal exposed is of first importance. Young animals are the most susceptible. The small rodents, the mouse, Guinea pig and rabbit are susceptible in the order named, followed by the sheep and horse and these again by the camel and ox. Among the wild herbivora the goat, deer, and stag have a high susceptibility. Man is less susceptible yet contracts the disease readily by inoculation, inhalation or ingestion. Swine and dogs are comparatively little susceptible, yet they often contract the disease by eating the carcasses or discharges of anthrax animals. White rats and birds are held to be insusceptible, yet the latter contract the disease readily when the vitality of the system has been reduced by immersing the body in water, or giving antipyrine. A similar result is observed in the otherwise immune frog if the body is heated above the normal cold blooded temperature. The receptivity may vary, however, in the same genus and species. The Algerian sheep is virtually immune from anthrax, perhaps because its ancestors have been so constantly exposed that only the insusceptible strains survived. Swine, birds and carnivora may have similarly acquired a fair measure of immunity by the survival of the fittest. Apart from this, however, a flesh diet is to a certain extent protective, thus Feser's rats if fed vegetable food proved susceptible to inoculated anthrax, while if fed on animal food they were comparatively immune.

The animal that has survived an attack of anthrax is thereafter strongly immune. This serves to partly explain the apparent immuuity of animals bred in an anthrax district, the young animal becoming habituated to infinitesimal doses of the toxins, conveyed in the secretions of the uterine glands or mammæ.

Soil is a factor so far as it preserves and propagates the bacillus. As already stated, soils that are naturally wet by reason of their impermeable character, their position on or near the water level, their conformation in basins which dry out in late summer or autumn, are especially favorable to preservation of the bacillus. Again soils that are especially rich by reason of an excess of decomposing vegetable and animal remains, or because of excessive manuring, tend to preserve and multiply the microbe. Rich,

flat meadows below tanneries or abattoirs, and irrigated from these or occasionally overflowed are especially dangerous to stock placed upon them. Soils with an alkaline reaction from the lime, potash or ammonia present are very favorable to anthrax. Wells receiving surface drainage are common factors in carrying infection.

Season is a contributing factor in various ways. Damp seasons sometimes bring the germ to the surface of the soil by the gradual elevation of the water level, or by causing inundations and the deposition of the bacillus on areas of pasture or forage that were previously free, or finally by bringing the earth-worms to the surface and leading to the deposition in their casts of the bacillus brought from the infected graves or retentive subsoils. Dry seasons are, however, the anthrax seasons *par excellence*, as they dry up swamps, fens, ponds, lakes, basins, deltas and bottom lands, and render them available for pasture. The germ-laden mud of these drying lands is also raised in dust and deposited on the vegetation to be taken in by the animals. Again on the drying basins and bottom lands the tempting green vegetation is often pulled up by the roots with adherent, infecting mud. For the above reasons, even in an anthrax region the malady is most prevalent in the late summer and fall, and in certain valleys like those of Corsica the stock is considered safe until the dry autumn weather demands their removal to the mountains.

Another reason for the summer epizoötics is found in the transference of the germ by *flies*. House flies, horse-flies, blow-flies, mosquitoes, etc., carry the bacillus on their feet, mandibles or piercing apparatus, and even in their stomachs (Bollinger) and transfer it from one individual to another. It is worthy of note that the great majority of cases of local anthrax in man occur on the habitually uncovered parts of the body (face, neck, hands and arms) and start from a centre like the bite of an insect.

Stables, *stable utensils*, *harness*, *shafts*, *poles*, *fodder* and *litter* are familiar bearers of the virus. Butchers' knives and wagons and surgical instruments are further media of contagion.

Certain conditions of the animal system expose it to attack. The fever and constitutional disturbance which are caused by *the extreme heats of summer and autumn* are strongly predisposing, especially if alternated with frigid nights as seen on the Siberian Steppes. *Privation of water* raises animal temperature and thus

the *drying up of the customary drinking places* becomes an important factor. *Plethora* and *starvation* are alike predisposing, probably by lessening the resisting power of the system. *Overwork* and *exhaustion* predispose, as Roger showed by making the immune white rat turn a wheel until worn out and then successfully inoculating it. The addition of lactic acid to the virulent liquid (1:500) greatly encreases its potency (Arloing, Cornevin and Thomas) and the further addition of fermentescible sugar (and rest) enhances this still more (Kitt). The production of lactic acid by muscular overexertion is thus a potent accessory cause in fatigue. In milk ducts of the susceptible it proves fatal: in those of the immune it lives indefinitely, killing slowly by toxine poisons and marasmus. (Nocard).

Bacillus anthracis. This was first demonstrated in anthrax blood and exudates by Pollender and Brauel in 1849 and 1850 but as they failed to find it in all cases they concluded that it was not the essential cause. Davaine who found the bacilli in 1850 suspected that they were pathogenic and by 1863 he had shown that blood which contained no bacilli was non-virulent, while that in which these organisms were present was constantly infecting. Klebs and Tiegel in 1871 filtered anthrax blood through an earthenware vase and found that the clear filtrate (bacillus-free) was non-infecting.

The *bacillus anthracis* as found in the blood is a nonmotile rod-shaped organism, 5 to 20μ long by 1 to 1.5μ broad with ends apparently square, but really slightly cup shaped as seen in the stained specimens when two have remained connected end to end. Under favorable conditions a clear hyaline envelope may be seen around the bacillus. Though usually isolated in the living blood, yet in bouillon cultures the bacilli grow out into long flexible filaments, made up of separate segments which are easily distinguished in the stained specimen. In all cultures out of the living animal body, in the presence of air and at a suitable temperature spores form endogenously in the bacilli, exceptionally polar, and are set free by the granular degeneration of the latter. In peptonized bouillon this may occur in four days at 14°C., or in eight hours at 37° to 40°C., but not above 42°C. (Schreiber. Centr. S. Bact. 1896). Sporulation never occurs in the living animal body.

The bacillus is ærobic, yet it will grow at the bottom of a stick culture in solid media. It fails to grow in an atmosphere of CO_2,

H, or N. It stains readily in aniline colors, and also by Gram's method. It grows freely on a variety of culture media (blood serum, aqueous humor, urine, vegetable infusions, milk, meat bouillon, peptonized gelatine, potatoes, etc.) at a temperature of 20° to 38° C. Growth ceases below 12° C., and above 45° C. Growth is most active in neutral or slightly alkaline media, and is arrested by that which is decidedly acid.

Action of physical and chemical agents on the bacillus anthracis. The bacilli survive a temperature of —45° C. (—49° F.), but they perish in 10 minutes at a temperature of 100° C (212° F). A temperature of 55° C. (131° F.), proves fatal if sufficiently prolonged. The spores are much more resistant. They have survived—130° C. (—202° F.) and though they may die in 10 minutes in liquid media kept at 95° C. (203° .F), yet when old and dry it may require several hours at 140° C. (282° F.) to sterilize them.

Even the bacilli are comparatively resistant to ordinary disinfectants, and, as spores form in the body very rapidly after death, and in virulent products, it is always best to assume their presence. In a moist medium Cl. 44.7% destroyed the spores in 3 hours: HCl (1 : 1100) in 2 hours: HgCl (1 : 1000) in a few minutes: HgI_2 (1 : 20000) in 2 hours: Malachite green (1 : 14,000) in 2 hours: Methyl violet (1 : 5000) in 2 hours: Aseptol (1 : 10) in 10 minutes: Carbolic acid (4 : 100) with HCl (2 : 100) in 1 hour.

Action of septic ferments on the bacillus anthracis. Rapid putrefaction in the anthrax carcass which has not been opened tends to speedy granular degeneration and death of the bacillus anthracis, so that the blood and tissues may be no longer infecting after six days in summer. In such cases the irrespirable gaseous products of decomposition drive out the oxygen without which the bacillus cannot live. If, however, spores have already formed or if air is freely admitted, the infection survives in spite of decomposition. The search for the bacilli may thus be fruitless as soon as decomposition is well advanced and the material can only be virulent through any spores that may have formed. Eventration serves to retard sepsis and admit air to form spores, and salting operates in a similar manner.

The infected hides, the nasal, buccal, kidney and bowel discharges, and spilt blood and exudates mingling freely with the

air tend to form spores and to preserve and propagate the contagion. On the contrary prompt and deep burial, without opening the carcass and before spores can have formed will usually ensure its destruction. The main danger in such cases comes from infecting matter (adherent to the surface of the body) which sporulates easily. This serves to explain the great danger of working in anthrax hides, leather, horn, wool, hairs and bristles. It also explains sporulation and preservation of infection when the virulent excretions, blood, etc., mix with the surface layers of soil. This may happen at a greater depth (3 or 4 feet) in a very porous soil and where the temperature is sufficiently elevated (above 14° C.) It may even occur in water. The dried spores are mostly carried in dust, hay, fodder, and running streams.

Since 1892 anthrax has prevailed along the banks of the Delaware river for a distance of 40 miles in N. J. and Del., destroying from 70 to 80 per cent. of the farm stock. The great morocco industry on this river draws infected hides from India, China, Russia, Africa and South America, and the spores are carried and distrubuted by the tides.

Infection-Atria. Infection may occur by a variety of channels as: 1st, by *ingestion*, giving rise most commonly to anthrax of the mouth, throat or intestines; 2d, by *inhalation*, giving rise to pulmonary anthrax (wool-sorter's disease): 3d, by *inoculation* through contact of abrasions, wounds, etc., with infecting bodies, including surgical instruments: 4th, by flies and other insects: and 5th, by transmission to the fœtus in utero. This last form is very rare in the larger animals, but has been repeatedly seen in Guinea pigs, rabbits, goats and even in one case (Pangalli) in man.

Forms of Anthrax in Domestic Animals. In the lower animals anthrax manifests itself differently according to the seat of invasion and the amount of the virus. The worst forms, seen especially in cattle and sheep, are so sudden that they have been called *apoplectic* or *fulminant*. Without premonitory symptoms there is sudden loss of appetite, trembling, haggard expression of face, uneasy shifting of the feet, irregular movements backward or to one side, dyspnœa, cyanosis, plaintive cries, convulsions, ejection of blood by the nose or with urine or fæces, and death in a time varying from a few minutes to four hours. The

second type is the *anthrax fever*, known also as *splenic fever*, *splenic apoplexy*, or *internal anthrax*. In this form there may be prodromata, especially in sheep, excitability, restlessness, and above all, a rise of temperature of often 3° or 4°. There may be distillation of drops of blood from the nose, eyes or ears, the mucosæ become congested, and in sheep this may show on the finer parts of the skin, as inside the forearm or thigh. Tremors, erection of the hairs, dulness, prostration, lagging behind the flock or herd, insensibility of the loins to pinching, inappetence, ardent thirst, grinding of the teeth, colics, tympany, mucus coated, bloody or liquid fæces, bloody urine, tumultuous heart beats, dyspnœa, dark, congested mucosæ, amounting to cyanosis, and spasmodic contractions of the muscles of the back, neck or eyes. If blood is drawn it may appear abnormally dark in color and very slow to brighten under the action of the air, it may have a thick, tarry appearance, and form a very loose clot. Death, (usually in coma or convulsions) will supervene, in sheep under 24 hours, in cattle in 2 to 5 days, and in horses in 1 to 6 days. The third type is the *local* or *external anthrax*, assuming in cattle the special forms of *gloss-anthrax*, *pharyngeal anthrax*, *hæmorrhoidal anthrax*, *cutaneous* and *subcutaneous anthrax*. In horses most often same forms appear, in the tongue, throat, neck, shoulders, withers, flank or thigh. These swellings have a firm or doughy feeling, are comparatively and sometimes wholly insensible and show a marked tendency to necrotic changes. When incised they show extensive blood extravasation, or a pale, straw-colored exudate mixed with sanguineous lines or patches, and manifest no tendency to suppuration, nor to emphysematous crepitation. These features distinguish them from phlegmon and emphysematous anthrax. When suppuration ensues it is tardy and indolent and is, on the whole, a favorable indication. In all cases the bacillus may be found on microscopic examination of the exudate.

Lesions. Putrefaction of the carcass is usually rapid. In the very rapidly fatal cases the changes in the blood and tissues are often little marked, and after the removal of the enlarged, engorged spleen and infiltrated internal organs, the carcass might often be placed on the market without much suspicion. In more prolonged cases the blood is profoundly changed, being very dark, not subject to rapid æration, and incapable of coagulating

firmly or at all. The red globules are crenated or otherwise distorted, adhere to each other in irregular masses, and have parted with much of their hæmoglobin which diffused in the serum stains the intima of the blood vessels and other white tissues. The leucocytes are relatively very much encreased. The bacteria are easily found in the intervals between the globules. The heart is often soft, discolored, as it were parboiled, with the endocardium deeply stained and the contained blood dark and diffluent or liquid. The liver is usually enlarged, softened, friable, and as if parboiled, with many hæmorrhagic patches. The spleen is materially, often enormously enlarged, irregular in outline from extreme engorgement with blood, and in exceptional cases even ruptured, An encrease to two or three times the normal is common. The bacilli are present in great numbers in the spleen, alike in the pulp, in the blood vessels and in the trabeculæ. The lymph glands are almost always hyperæmic, hypertrophied, and softened, especially in the vicinity of the localizations of the tissue lesions. They may be merely petechiated, or they may seem like a mass of black blood, and under pressure may break down readily into a sanguineous pulpy mass. Like the spleen they are favorite centres for the accumulation of the bacilli. The marked alterations in these glands will often indicate the channel by which the infection entered the body. The serosæ are usually hyperæmic, with many hæmorrhagic points and even extensive exudations, and they often enclose a sanguineous liquid The hyperæmia and points or patches of extravasation are to be found in any part of the body in which the bacilli have been colonized, thus they are common in the tongue, the throat, the lungs, the stomach or bowels, the mesentery, the omentum, the skin, the connective tissue, or the muscular system. There may be bloody or gelatinoid exudation, but there are always the capillary embolisms, by irregular masses of blood globules, and bacilli. These embolisms, the arrest of hæmatosis and the destructive action of the toxins on the red globules go far to account for the extreme fatality of the disease.

Morbid poisons. Hoffa found in anthrax cultures a ptomain which killed with anthrax symptoms. Hankin obtained a deadly albumose which in small doses procured immunity. Brieger and Fraënkel separated a toxalbumin, and Martin too, a protalbumose

and a deuteralbumose together with a ptomain. Marmier separated a toxin which did not give the reactions of the albuminoids, albumoses, peptones nor alkaloids. This was not poisonous in small doses, to animals possessing immunity—natural or artificial. It was weakened but not destroyed by 110° C., and was rendered harmless by treatment with alkaline hypochlorites. Immunity could be induced by its use in small nonfatal doses. This is present in the bacilli and being soluble in water can be secured from these by diffusion in watery fluids and especially so if aided by heat.

Incubation. The implanted bacillus begins at once to multiply in the tissues, but the encrease is at first slow and the resulting morbid phenomena slight, so that there appears to be a period of incubation. In experimental inoculations in which this can be certainly noted it extends from one to two and even three days in the rabbit and Guinea pig, from two to four days in sheep, and from three to six days in horses and cattle. It may be shortened by giving an overdose and especially if this is introduced intravenously, the chemical poisons apparently acting at once. In young animals, too, the period is shortened, unless they have been rendered refractory by a milk diet or otherwise.

Symptoms. These vary according to the species affected, and the seat of the disease, general or local, *internal* or *external.* In the latter case the febrile and constitutional disturbance is delayed. In the *internal* cases the fever is early and of a high type. Even before the animal appears to be seriously ill, while still keeping with the herd and showing life and vigor, there may be high temperature, 104° to 106° F., pulse and breathing accelerated, heart beats tumultuous, tremors or shivering or perhaps only staring coat, anorexia, and grinding of the teeth. Later there may be drooping head and ears, dulness, a disposition to lie, apathy, stupor and somnolence. Nervous excitement and delirium have been noticed. The patient becomes weak, especially behind, comatose, and the temperature declines to, it may be, 97° or even 94° F., prior to death. The visible mucosæ become dusky, brownish or yellowish, and streaks of blood may appear in the nose or elsewhere. The urine may be red, the fæces covered with mucus, or blood streaked, and the rectal mucosa of a violet tinge, or blackish and blood gorged.

In this the vulvar mucosa often participates. Local swellings may appear in the tongue or pharynx, even if not on the surface, or colics indicate implication of the digestive organs. The examination of the blood early reveals the presence of the bacillus, and as the disease advances, its black, incoagulable, tarry, or coffee-grounds appearance becomes characteristic. Pregnant animals are liable to abort. Death occurs in 12 to 48 hours. Most cases are fatal at the beginning of an outbreak, while later the great majority often recover.

Fulminant cases (Anthrax acutissimus) occur mostly in cattle or sheep, in high condition, the victim being found dead in pasture or stall, without previous observation of illness, or, if seen during life there is the sudden attack, leaving food, muscular tremors, anxious expression of countenance, hyperthermia, dyspnœa, dark red mucosæ, it may be streaks of blood on them, plaintive cries, rolling of the eyes, spasms or coma, and death in a few minutes to one or two hours. In *sheep* there may be separation from the flock, pawing, stretching, shaking of head, turning in circle, dyspnœa, falling, convulsive struggling, passing of blood by nose, kidneys or bowels and death.

Local Anthrax in Cattle. Symptoms. In *cutaneous anthrax* the circumscribed swellings appear suddenly, and may grow to considerable dimensions on different parts of the skin, head, neck, breast, shoulders, abdomen, axilla, sheath, udder, or flanks. There may be one or many, and they lack the acute early tenderness, and later crepitation of black quarter. On white skins they are dark red, or violet, and when incised, show a gelatinoid, bloody, non-suppurating mass, abounding in bacilli.

Gloss-anthrax implicates the whole, or it may be the roots only, of the tongue and the fauces. There is profuse salivation, perhaps bloody, intermaxillary and pharyngeal swelling, anorexia, and the tongue is found protruding, swollen, violaceous, vesiculated or with rounded nodules, or sloughs and ulcers, with lardaceous or blood stained bottoms. Hyperthermia and constitutional symptoms are present.

Pharyngeal anthrax is manifested by swelling of the throat, profuse salivation more or less marked with blood, complete dysphagia, attending on the marked febrile and constitutional symptoms already described.

Hæmorrhoidal anthrax, a common complication of the constitutional disease, is manifested by infiltration, blood extravasation, violet discoloration and often enormous swellings of the rectal mucosa, seen mainly during straining, but sometimes also as a constant protrusion.

Local Anthrax in Sheep and Goats. External anthrax swellings may form on the face, throat or udder with the general characters of those of the ox, but this form is much more rare than in cattle and horses. The usual form is the internal one, with engorgement of spleen, liver, and perhaps some other internal organ, and it is relatively very fatal.

Local Anthrax in the Horse. *Symptoms.* In anthrax districts, tumors form in the seats of inoculations on sores, insect bites, or sometimes without apparent local cause, as a result perhaps of a general infection. The infiltration takes place suddenly into or beneath the cutis, mostly on the head, tongue, throat, neck, breast, shoulder, inguinal region, mammæ, croup, or thigh, encreases rapidly, but without crepitation, or suppuration, the incision showing the general characters described in the ox, and early attended by the constitutional disorder. The general fever is often later in developing, less intense, and, on the whole, less fatal than the purely internal forms.

Anthrax in Swine. *Symptoms.* Swine are, on the whole, less susceptible than cattle, and on the strength of his laboratory experiments Pasteur denied their susceptibility. The mistake gained a wide acceptance, yet experienced practitioners knew that they were occasionally infected by eating the carcasses or droppings of anthrax cattle. I had seen a number of swine die in common with cattle in an outbreak at Swineshead, Lincolnshire, in 1863, and a like occurrence took place in East Lothian, and similar cases are reported by McFadyean, Trombitas and Von Ratz, while Crookshank and Perroncito have respectively inoculated the pig with success. In my experience at East Lothian a shepherd skinned an anthrax bullock and then castrated several litters of pigs, all of which died of anthrax. Much doubtless depends on the condition of the animal as regards food (flesh or vegetable), the presence of lactic or other organic acid, the coincidence of infection with anthrax bacillus and one of those conditions, which habitually enhances its virulence. There

may be named venesection (Rodet) or its counterpart anæmia, the presence in the blood of inert powders—precipitates (Bardach), fatigue—sarco-lactic acid (Charrin and Roger), starvation —dyspepsia (Canalis and Morpurgo), privation of water (Pernice and Allesi), the products of previous or coincident illness (Galtier).

The pig, infested by ingestion, suffers especially from pharyngeal and intestinal anthrax. There is marked swelling of the throat with stiffness, dysphagia, champing of the jaws, salivation, frothing about the lips tinged with blood, dark, violet discoloration or ulceration of the fauces and tonsils, retching, vomiting, hoarseness of grunt, extension of the swelling to the face, with petechial spots and patches, diarrhœa with frothy or bloody fæces, great muscular weakness, a disposition to lie, it may be actual paraplegia, with the usual accompaniments of hyperthermia, constitutional disorder, cyanosed, or dusky brown, reddish or yellowish mucosæ, and the black, incoaguable blood, with destruction of the red globules.

Anthrax in Dogs. *Symptoms.* In the outbreak at Swineshead, Lincolnshire, in which the pigs suffered, one shepherd dog contracted the disease from eating the carcasses. It took the pharyngeal and intestinal form, with dysphagia, vomiting, bloody diarrhœa and high fever. Straus found that young puppies were very susceptible and old dogs refactory probably because of flesh diet and exposure. The excision of the spleen encreased the susceptibility to 76 per cent. instead of 20 per cent. (Bardach). Cornevin saw 5 dogs die the same night out of 7 that ate from an anthrax carcass. Much depends on the previous exposure and existing condition of the dog.

Cats have been observed to suffer under similar conditions. At Geneseo in 1877 I found that a cat and three young horses died from licking the blood from a stoneboat on which an anthrax hide had been carried.

Anthrax in Birds. *Symptoms.* Birds (chickens) are naturally less susceptible than swine, yet they succumb readily to inoculation, when the body has been cooled by partial immersion in cold water. Caplewsky found that, apart from artificial chilling, young pigeons of certain breeds were easily infected, and Œmler successfully inoculated small birds, sparrows, finches,

canaries, yellowhammers, redbreasts. The larger birds are more resistant but succumb readily if dosed with chloral hydrate or antipyrin (Wagner). Birds of prey seem to be immune.

In chickens the disease is very acute, of rapid progress and fatal. A few hours after inoculation they are seized with dulness, debility, sunken head, drooping wings and tail, ruffled feathers, and dark red or black discoloration of comb and wattles. Dark colored anthrax swellings may appear on these last, on the eyes, tongue, palate or feet, and the obstruction of breathing may cause general cynosis. Weakness is extreme, the bird staggers, or is unable to rise, has violent tremors or convulsions, with bloody diarrhœa, and perishes after a few hours, or a day's illness. The presence of anthrax in the locality, or in other species, will be to some extent a safeguard against confounding chicken cholera, entero-hepatitis, Birdpest, or malignant œdema with this affection. The crucial diagnosis is based, as in other animals, on the discovery of the characteristic bacillus.

Differential Diagnosis. The suddenness of the attack, hyperthermia, dusky, cyanotic, petechiated mucosæ, the escape of blood from mucous surfaces, the dark, tarry blood, brightening imperfectly on exposure to the air, its comparatively loose coagulum, the crenation and destruction of red globules, the staining of the serum with hæmatoidin, the leucocytosis, the engorged, enlarged liver and spleen, and the gelatinoid or bloody swellings, not gasogenic as in black quarter or malignant œdema, together present a picture which is strongly suggestive of anthrax. If the malady affects domestic animals generally, is especially virulent in cattle, sheep and horses, and attacks even man; if the district is subject to anthrax, or of a rich, damp soil which would favor the preservation of the bacillus anthrax; if it is in the line of watershed from stock-markets, abattoirs, tanneries, rendering works, glue factories, packing houses, sausage factories, or phosphate works; if forage or new stock has been introduced from an anthrax district; and if the outbreak has taken place with a high soil-water level, or during a dry, hot season, the case for anthrax will be strengthened.

The final tests are known by the microscope and inoculation. To discover the bacillus a power of 400 to 500 diameters is desirable. From the living animal take a drop of blood, exudate or

hæmorrhagic extravasation, make a thin film on a cover-glass by drawing across it the straight edge of another one, dry the film, then pass it three times through the alcohol flame, film downward, stain in anilin dyes, clear with acetic acid and examine. The bacillus is large (5 to 20μ by 1 to 1.5μ), nonmotile, of uniform thickness throughout, and sharply cut off at the ends. The bacillus of blackquarter is shorter, often club-shaped because of spore in one end, has rounded ends, is flagellate, motile and gasogenic. That of malignant œdema is much thinner, has rounded ends, sluggish movements and is gasogenic. The bacillus subtilis (hay bacillus) is short, thick, with rounded ends, each bearing a flagellum and is motile. The proteus vulgaris (common septic saprophyte) is small, short, with rounded ends and very active movements. If the subject is dead we may examine the blood, or the scraping from the cut surface of the spleen, liver, kidney, congested lymph gland or other part bearing the lesion.

If we find in the carcass exudates, gelatinoid or bloody (especially the latter), petechiæ, dark, uncoagulated blood, brightening little on exposure, blood-gorged spleen, congested or hæmorrhagic condition of one or more internal organs, muscle or connective tissue (particularly of the lymph glands), if the muscles of the loins, quarters, thighs, diaphragm, or elsewhere, are soft, as if parboiled, salmon-colored, clammy, friable, or if reddish, yellowish, brownish, with petechiæ, and capillary embolism, the case will require critical examination. If the first examination fails to show bacilli, repeat it from different lesions until thoroughly satisfied of their absence, or until another cause for the condition has been discovered. The blocking of the capillaries with bacilli in the various lesions is a most important point, never to be overlooked.

In case of uncertainty, inoculation of a Guinea pig, mouse or rabbit should be made. The blood or scraping from the seat of a lesion is made into an emulsion, if necessary, and injected subcutem. A single bacillus will destroy a mouse or Guinea pig in from one to four days, and the blood shows the characteristic bacilli.

One must, however, preclude the possibility of septic bacteria excluding or obscuring the bacillus anthracis, by taking the inoculating material from the blood of the living animal, or from

the same, or the tissues as shortly after death as possible. Fifteen hours may be altogether too late for inoculation. To exclude the anærobic bacteria of black-quarter, malignant œdema, and septic affections, make an emulsion of the suspected material in sterilized water, filter through a boiled cloth, and inject a strong dose into the auricular vein of a rabbit. The anærobic bacteria perish in the blood, and, if anthrax bacilli are present, they are found in pure cultures.

In inoculating suspected water or infusion of forage the intravenous method should be adopted. Another resort is to make two artificial cultures, one in free air, and the other in an atmosphere of nitrogen or carbon dioxide. The bacillus anthracis develops in the first, the anærobes in the second.

Prognosis. Mortality. Fulminant cases are uniformly fatal. Acute intestinal cases are usually fatal in 70 to 90 per cent. of the animals attacked at the beginning of an outbreak. Toward the decline most cases may recover. In a herd of 200 head, at Avon, N. Y., in 1875, 40 fat bullocks died in two weeks, and 50 more showed a marked hyperthermia, yet under a change of pasture and antiseptics, all but two of the latter recovered. As serving to identify the disease, three attendants suffered from malignant vesicle, but recovered.

SUPPRESSION AND PREVENTION OF ANTHRAX IN HERDS.

Less simple or easy than in plagues. Germ survives in soil and water. Extinction not always possible. Killing: conditions demanding it; when unwarranted. Kill without shedding blood or opening carcass. Body burned; if buried, five feet deep, in porous soil, distant from wells, ponds and rivers. Fence graves, burn grass. Disinfection of hides, litter, fodder, manure, excretions, stables, etc.: of bodies that must be moved, of buildings, yards, utensils, etc. Isolation of unaffected on porous soil; surveillance. Sales interdicted, milk, butter, cheese. Immunization: by toxins which stimulate leucocytes to form defensive products: anti-toxins. Eosinophile cells, action of spleen and liver. Mellituria. Protection by a minimum dose, by enfeebled virus—modes of lessening potency, Pasteur's "vaccin," its drawbacks and dangers, its technique; by soluble toxins in sterile solution, author's experience, apparent failures, advantages. Drainage—æration of land. Prevention of importation and diffusion. Therapeutic treatment.

Prevention of anthrax in animals is equally important for the sanitation of herds and human beings. It involves the purging from the anthrax bacillus of the infected lands and drinking supplies, and in this respect the disease is much less amenable to thorough and speedy extinction than is a simple plague in which the germ does not live and multiply outside the animal body. In some localities the extinction of the germ may be confidently counted on and secured; in others this may be impossible, and other measures of protection must be resorted to.

Killing of the Sick and Disposal of the Carcass. This is not always so imperative as in the obligatory parasitic infections, since the destruction of the sick still leaves the germ present in the soil and water. If, however, the infection has just been introduced, on hitherto uninfected soil, by the arrival of new animals, and, if the new location is in any way favorable to permanent colonization by anthrax bacillus, and if the diseased and suspected animals cannot be kept secluded so as to absolutely exclude these dangers, or again, if the diseased herd or its remnant is to be moved on to another locality, slaughter is the obvious sanitary resort.

The animal should be killed on the premises to avoid the danger of scattering the infectious discharges in transit; it must not be bled nor cut open, as the admission of air determines the formation of the resistant spore; and the carcass must be burned, boiled, or rendered in superheated steam under pressure, or finally dissolved in strong mineral acids. If buried, it must be in open, porous soil, far apart from any well, pond, river or bank where the liquids may leach out, and the body must be wholly covered to a depth of at least five feet. The graves must be well fenced in from all stock, for a number of years, and no forage grown on them can be safely fed to animals, as the bacillus can be brought to the surface by earth worms or soil water. I have known cattle to become infected by licking the fluid which escaped above a stratum of clay, on the deep bank of a river, at some little distance from where an anthrax carcass was buried in the surface sandy loam. A covering of coal-tar. chloride of lime, or of sand charged with sulphuric acid is an admirable precaution.

As a measure of economy the skins may be removed, if at

once, on the spot, plunged for 12 hours in a 5 per cent solution of carbolic acid, or cresyl, or creolin or in a 2 per cent solution of sulphuric acid, and, if the knives and other instruments used are placed in boiling water for half an hour. The same will apply to fleeces.

Disinfection. All litter, fodder, manure, urine, and other excretions, or products; all stalls, feeding troughs, sheepfolds covers, halters, harness, wagons, poles, shafts, and other objects used about the animals, or soiled by them or their products, should be disinfected by burning, flaming or scalding (boiling), when applicable, by one of the above disinfectants, by mercuric chloride (5:1000), by formalin, or other potent antiseptic. Extensive dungheaps, too wet to burn, may be sprinkled freely with strong mineral acids, or mercuric chloride in solution, piled in compact mass, covered with chloride of lime and finally with a thick layer of earth, and fenced in from all stock.

If carcasses must be moved to the grave, rendering works, or elsewhere, they should be sponged with carbolic acid solution, formalin, or mercuric chloride, and each of the natural openings firmly plugged with tow or cotton soaked in the same material, so that no infecting matter may drop on the way. They should on no account be dragged on the ground, but carried on a wagon or stoneboat, which should be afterward carefully disinfected. Men or animals, entering an infected place, should be disinfected on leaving: especially hands and feet.

All roads, yards and pastures, where the sick have been, and, above all, where manure, saliva or urine has fallen, should be subjected to thorough disinfection, or the surface layer removed and deeply buried. When available a concrete or asphalt floor should be placed in the buildings.

Isolation. Movement from Infected Ground. It has long been known that the movement of an infected herd from the contaminated pasture to another, will often at once check the development of new cases. In Sardinia and Auvergne the flocks and herds were yearly moved on the approach of autumn, from the rich valley, and bottom lands, to the drier hill pastures, to avoid or lessen the decimation that otherwise inevitably overtook them. This is in keeping with the enzoötic nature of the malady which arises more from the microbe preserved in the

soil than from the sick animal direct. Two precautions are necessary in making such change of locality: 1st, Animals already infected should not be moved on to such new pasture; and 2d, The pasture to which the stock is moved should be entirely free from the impermeability (clay, hard pans), and saturation with water (swamps, basins, low bottoms) which would ensure the permanent preservation of any microbe planted there. Elevated, sandy, argilaceous or loamy soils are to be selected. To these the animals of the infected herd, which by their appearance and thermometry may be pronounced sound, should be removed and kept under careful supervision, especially as regards thermometric tests. Any showing symptoms of anthrax should be at once taken back to the infected herd. If they have stood in stalls, for milking or otherwise, these should be disinfected, and they should be carried in wagons or driven by unfrequented roads. Their droppings should be carefully disinfected.

If, in the absence of anthrax symptoms, animals must be kept in the infected lot, or returned to it, they should be immunized.

Interdiction of sales. No animal in the infected herd which shows a rise of temperature, should be sold even for slaughter. No animal should be sold for stock purposes until the disease has completely subsided. Any animal, in the infected herd, which shows no hyperthermia nor other sign of anthrax, may be sold for immediate slaughter, subject to a critical expert examination of the cadaver for anthrax. Milk, the product of an infected herd, and butter and cheese made from such milk, should not be used as food. If such members of the herd as show no hyperthermia or other symptom of anthrax, can be held apart as a separate herd, in a disinfected place, and under careful thermometric observation, their dairy products may be used.

Immunization. A number of different methods have been practiced of rendering animals refractory to the bacillus anthracis, but all are apparently based on the production in the system of defensive products, as the result of a nonlethal poisoning with anthrax toxins. It is true of anthrax as of many other infections that a first attack protects against a second. In all animals there is a certain measure of defensive power against the

bacillus anthracis, amounting in some cases to virtual immunity, and in others having very little effect. The object in immunizing is to stimulate to the encrease of these defensive products in quantity or power until an ordinary dose of the bacillus will fail to colonize the tissues or the blood. In considering this subject a clear distinction must be made between the simple bactericidal and the antidotal or antitoxic products found in the serum of immune animals, and the toxins which are produced by the bacilli. The soluble antitoxic and bactericidal agents found in the serum of the immune, may be employed for therapeutic purposes to preserve life in an animal which has received a lethal dose of the bacillus anthracis, but, as these are rapidly eliminated from the system, their protective power is very short-lived, and if some bacilli survive the period of their presence and potency, or if the bacilli are introduced into the system later, the animal may fall a victim to anthrax as if no such protective agent had been used. Behring showed that the blood-serum of the white rat proves fatal to the bacillus anthracis, but Metchnikoff pointed out later that it must be brought in contact with the bacillus in order to prove effective, whereas if the serum and bacillus were injected at different parts of the body no protection was obtained. The antidotal or bactericidal action of the serum of an immunized animal acts at once, whereas a permanent immunity cannot be established before about fifteen days. The serum of the immune animal contains the following elements antagonistic to anthrax: *Antitoxin* or *leucomain*, which may be poisonous to the bacilli, or chemical antidotes to their products: *globulicidal principles* which distort or disintegrate the blood globules and release their contents, including the bactericidal nucleins: *precipitins*, *agglutinins*, etc. All such agents, when injected into the system, are present only for a limited time, and while they may be subservient to a temporary immunity, they can give no permanent protection and must be considered mainly as therapeutic agents.

A permanent immunity must depend on a stimulation of the system to the production of these defensive agents *de novo* or in encreased quantity. This must be done by exposure of the tissues to the toxins of the bacillus anthracis, and is accomplished slowly. All the tissues that engage in the production of the de-

fensive agents can not be certainly stated, yet certain indications show that the eosinophile cells of the blood are presumably important factors in this work. The spleen as the seat of extensive blood changes, and as preëminently the seat of election of internal anthrax is probably involved. The dogs from which Bardach had removed the spleen were found to be three times as susceptible to anthrax as were the dogs that had not been operated on. Leo's rats, in which he had produced mellituria by the administration of phloridzin were found to be much more susceptible to anthrax. This together with the habitual implication of the liver in internal anthrax, suggests the existence of a certain protective power in the products of the healthy liver.

The practical problem for the sanitarian is to develope the habit of producing defensive products without imperilling life.

By Minimum Dose. Chauveau and Colin secured this in the larger animals by intravenous injection of a minimum dose,—one or two bacilli. This is more lasting in effect if a second and stronger dose is injected some days later.

By Weakened Virus. This has been secured by heating the defibrinated blood to 55° C. for ten minutes (Toussaint); Pasteur, Chamberland and Roux accomplished the same end by making anthrax cultures at 42° to 43° C. in presence of air; Chauveau by subjecting the virulent culture for 8 days to oxygen under a pressure of 8 atmospheres at a temperature of 38° C.; Chamberland, Roux and others have cultivated the bacillus in weak antiseptic bouillons as phenic acid (1:600 or 1200), bichromate of potash (1:2000 or 5000), sulphuric acid (2:100).

Other methods have been followed, as growing the bacillus in the blood or serum of immune animals (dog, chicken, pigeon, white rat, frog).

Of these different methods that of Pastenr has been most extensively adopted. The temperature of culture (42° C.) prevents the formation of spores and the duration of exposure to air gradually lessens the virulence until in 12 or 13 days it is not fatal to the Guinea pig and after 31 days it fails to kill the young mouse. Thus preparations of varying grades of virulence, and adapted to the varying susceptibility of different animals, are secured. The protective inoculation is made by preference in spring, when there is less chance of complication by a coincident accidental in-

fection. It is to be avoided in animals at hard work, in advanced gestation, in full milk, in extreme youth or in ill health. To secure the best results it should be repeated with a stronger preparation 10 or 15 days after the first injection. The acquired immunity lasts a year or over and is probably perpetuated by new and nonfatal doses taken in casually on the anthrax pastures. Hundreds of thousands of live stock in all parts of the world have been treated in this way with the result of reducing a mortality of 2, 5 or 10 per cent. to insignificant proportions. It can only be safely adopted on anthrax lands, as elsewhere it may lead to the stocking of new areas with a malignant germ which in young and susceptible animals reacquires its original virulence.

It can never be safely ignored that we are dealing with the living seed of a most deadly infection. Though robbed of a large part of its virulence by artificial culture at 107.5° F., yet many accidental conditions contribute to a relapse to its original potency, and when it has once killed a victim the renewed virulence is usually persistent. If the virus, employed for protective purposes in cattle and sheep, is inoculated on Guinea pigs of 1 to 30 days old, from these to those of several months, and from these last on sheep, the virulence is constantly and persistently enhanced. The same is true of the microbe which is inoculated on a succession of pullets of steadily encreasing ages (Roux and Chamberland), or on a succession of pigeons (Metchnikoff). The germs reinforced in potency in any such way are liable to be the starting points for dangerous infections in animals and permanent contamination of soils and waters. Fortunately an occurrence of this kind is rare, yet with a wide application of the Pasteurean inoculation the opportunities are great, and with the free sale and distribution of the enfeebled anthrax "vaccin", the evil may grow indefinitely. The method departs from the ideal one, which aims at a final extinction of the disease, and accepts in place a more temporary protection of the generation which makes up the herd or flock at a given time, with no consideration for the generations that are to come after. Eradication of anthrax cannot always be secured, yet every effort should be made to attain it, and above all to check its infection of new land.

Technique. The weakened virus (1st "vaccin") is sold in tubes holding enough for 100, 200 or 300 sheep. Of this ⅛th

cc. is injected subcutem on the inner side of the thigh of the mature sheep, and 12 or 15 days later a similar dose of the stronger preparation (2d "vaccin"). For the ox or horse double the amount (¼th c c.) is used, being injected behind the shoulder, and on the side of the neck of the respective animals. The dose is graduated in the different subjects according to the size and age, yet a considerable latitude is permissible. The syringe must be disinfected before and after inoculations by a 5 per cent. solution of carbolic acid, or by boiling, and the nozzle should be dipped in strong carbolic acid immediately before and after each insertion. This will greatly obviate infection of the liquid used and of the wound by any virulent germs lodged on the surface of the skin. The liquid to be injected should be used as soon as possible after preparation, and if kept should be in a dark cold place, and if the tube is once opened the whole of its contents should be used the same day,—never kept over. The second, stronger preparation should never be used until the system has been prepared for it by the use of the first.

By the Soluble Toxins in Sterile Solution. In 1884 in an outbreak of anthrax at Skaneatles, N. Y., I drew blood from an anthrax cow, subjected it to 212° F. for 30 minutes, dissolved out the soluble toxins in boiled water, and injected the product subcutem, in a dose of 2 to 4 cc. according to size, into every apparently healthy member of the herd, excepting one, which was left as a check. The check animal died of anthrax which all the others escaped.

Since that time I have personally used it in every herd where opportunity offered, and with equally good results. In an outbreak near Elmira, Dr. Moore adopted it in a large dairy herd, and the disease was at once arrested.

In several experimental cases at the N. Y. S. V. College, the outcome was not so satisfactory, and in a herd in Oneida Co., N. Y., it is said to have failed to check the disease.

Notwithstanding these untoward results in other hands, I am still confident that we have in this a measure of no little value, and worthy of application in suitable cases. A certain percentage of failures in immunization are to be looked for. Even cowpox vaccination is not always protective against itself: I knew one man who was successfully vaccinated every three years in a com-

paratively long series. Many habitually self-limiting diseases relapse in particular individuals. Even in the case of anthrax, excess of glucose or lactic acid in the system, and the lack of some unknown influence of the spleen are respectively destructive of immunity. After the Pasteurian inoculation a certain number of inoculated animals are lost, it may be between the first and second injection, or it may be "two or three months" after the latter (Galtier). We must also bear in mind that in an infected herd or flock, there are almost always a certain number already infected at the time of the protective inoculation, and as the protective conditions are slowly established, through the action of the leucocytes, it is unreasonable to expect that serious illness and death can be obviated in such animals.

In inoculation with the Pasteur lymph, the bacillus is held not to enter the blood, a position supported by the researches of Bitter, Perroncito, Wissokovicz, Lubarsch, Metchnikoff, Chamberland and Roux, so that the resulting immunization must come from the toxins. Add to this that Chauveau (1885) conferred immunity on a sheep by injecting intravenously, anthrax blood, defibrinated, and sterilized by heat; Arloing obtained immunity in the sheep by injecting, subcutem, the clear supernatant liquid from old bouillon cultures of anthrax, from which all bacilli had been precipitated; Roux and Chamberland obtained the same result by using the pulp of an anthrax spleen, treated with essential oil of mustard, so as to destroy the life of the bacillus, and then evaporated in vacuo to remove the essence. Small doses proved more effective than when the splenic pulp had been filtered or sterilized by heating to 58° C.

The advantages of using sterilized toxins are numerous:

1st. As the material can be derived from a case of the outbreak in hand, there is no risk of using the anthrax protective inoculation for black quarter, hæmorrhagic septicæmia or other disease which is so often confounded with it.

2d. There is no danger of the sudden enhancing of the potency of an enfeebled microbe on account of some condition of the animal inoculated, as no living microbe is employed.

3d. There is no possibility of planting the anthrax bacillus on new soil, as is so liable to take place in using the weakened but still vital microbe.

4th. There is no necessity for the care and cost of holding the inoculated animals apart by themselves, under official veterinary control for 15 days, of witholding their products from market, or of disinfecting the place where they have been kept. On the contrary, the animals inoculated can be treated in every way as if no such injection had been made.

Thorough Drainage and Aeration of Land. The most complete and permanent method of eradicating anthrax is by thorough æration of the soil. In dry, sandy or gravelly soils, having a good natural or artificial drainage, and not underlaid by an impermeable damp stratum, the bacillus is never permanently found, and if introduced, is slowly robbed of its virulence by the action of the oxygen. When a soil can be well and permanently ærated by thorough underdrainage, a few years suffice to rob it of its infecting property and render it salubrious. In many localities, however, this is actually and economically impossible, so that the owner is thrown back on the alternatives, of abandoning the soil for stock keeping, or of immunizing all the animals placed on it.

Prevention of Importation of Anthrax. To prevent the introduction of anthrax into a country or district, the usual control must be exerted on trade in cattle and their products, as in the case of other infectious diseases. Live stock coming from an anthrax-infested country or district must be excluded, or admitted only after quarantine of 6 to 10 days and the disinfection of the surface of the animal. Dried hides, horns, hoofs, hair, wool and bristles are even more dangerous, as they are liable to hold the microbe in the spore form which will survive indefinitely and plant the disease widely. The recent great extension of the disease along the Delaware River, in connection with the morocco factories, which draw their hides from the most virulently anthrax regions (India, China, Russia, Africa, South America) is a strong case in point, and nearly every tannery planted on a favorable soil is an example on a smaller scale. Disinfection of all such products on arrival is essential. But this should be thorough and no question of trouble nor expense should stand in the way. If the trade cannot stand the expense, it has no right to exist where it is, threatening as it does, ruin, local and ultimately general, of agriculture on which all other industries are

based. Similar control is demanded of live stock products from infected regions in America.

The control of home markets, stockyards and abattoirs is no less important. Fortunately the disease is short-lived and deadly, and is much more easily discovered and arrested than in the case of plagues with prolonged incubation and frequently occult form (glanders, tuberculosis). An inspection of the various markets, and the detention of herds that have shown anthrax infection would do much to limit extension. This would entail the disinfection of the infected places, cars, boats, harness, clothing, and other things, and of the skins of the healthy animals of the infected herd.

The Therapeutic Treatment of anthrax in animals must in the main follow in the same lines given below for the human being; locally, antiseptics (mercuric chloride or iodide, Luzol's solution, hydrochloric acid, phenic acid, iodized phenol, creoline, cresyl, oil of turpentine, formalin, salicylic acid, scarification, excision of the primary sore or swelling with antisepsis, antiseptic injections into the swelling). Internally, there have been employed, dilute phenic acid, creolin, terebene, calomel, quinine, hydrochloric acid, bichromate of potash, tincture of iron chloride, etc. (See below).

ANTHRAX IN MAN.

Causes: infection from animals and their products, from soil, by flies, by dust. An industrial disease, of workers among animals and animal products. Wounds as infection atria, ingestion, anthrax, inhalation. Lesions: malignant vesicle, anthrax oedema, intestinal anthrax: pulmonary anthrax. Symptoms: malignant vesicle, œdematous anthrax, intestinal, pulmonary. Prevention. Treatment: caustics: antiseptics, excision of nodule and subsidiary glands, mercurial ointment, iodine, sodium bicarbonate. For intestinal anthrax: emetic, oleaginous purgatives, potassium iodide, sodium salicylate, iron muriate, heart stimulants. For pulmonary anthrax: inhale chlorine, iodine, bromine, phenic acid, eucalyptol, oil of cinnamon, sterilized cultures of prodigiosus, pneumo-coccus of Friedländer, bacillus pyocyancus, staphylococcus aureus, or streptococcus: blood serum of immune animals: blood serum (sterilized) of anthrax cattle.

Causes. Anthrax in man is usually the result of contamination by infected animals or their products. It is quite possible that man, like animals, may be infected directly from the soil or

water, or from the same source through the medium of flies or windblown dust, yet undoubted cases of this kind are rare or unrecognized. The animal origin of the disease, as regards man, makes this largely an industrial affection, attacking shepherds, cattlemen, horsemen, farmers. drovers, butchers, veterinarians, tanners, and workers, in hides, wool, hair, bristles, furs, hoofs, bones, rags, felt, glue and even leather. The sound skin is sufficient protection, but the slightest abrasion may form an infection atrium. Workers in tanneries and those who live near them are notoriously subject to anthrax. The hides must of course be drawn from an anthrax region. Russian, Armenian, South American, Australian and African. Hides have an especially bad reputation. *The British Medical Journal*, May 21st, 1898, records cases occurring in postal clerks who had to handle foreign parcels bound with strips of hide. Proust records cases from handling Chinese goat skins (Bull d. l'Acad. de Med. 1894). Infection may also occur through leather made from infected hides as proved experimentally on Guinea pigs.

Hair has long been recognized as a frequent medium of infection and outbreaks among brushmakers have been recently recorded by Gerode, Sarmont, and Chauveau (Compt. Rende de l'Academie des Sciences, 1893), Trousseau reports twenty cases in Paris all contracted from South American horsehair. Wool from infected countries is often dangerous and has given rise to special names for the disease (wool sorters', ragpickers', which may develop in the lungs from inhalation of the dust. In the same way those who handle bones about fertilizer, glue and rendering works are particularly exposed. The agency of insects in man is undoubted. In 60 cases recorded by Dr. Bell, 54 were on the face, two on the hands, one on the wrist, and one on the fore arm. This is mainly due to blood-sucking flies, yet Heim incriminates the Coleoptera as well (Compt. Rend. de Soc. de Biol. 1894). Wounds of all kinds contribute to inoculation, hence, the presence of burdocks, thorns, thistles and the like in the matted wool or hair is often a direct cause of infection.

The infection may be transferred on surgical instruments, and in these days of hypodermic medication the greatest care is necessary to prevent accidental inoculation with the needle.

As in animals man suffers from ingestion and inhalation of

the bacillus; and sometimes widespread mortality comes in this way. Meat just killed may be thoroughly disinfected by the secretions of a healthy stomach, yet the bacillus may pass through in an envelope of fat, in an undigested mass, or during a fit of indigestion, and infect the intestines. The spores are proof against the gastric juice, and as they are produced in a few hours after death the meat of an anthrax animal must always be considered as exceedingly dangerous.

Man is much less susceptible than some animals and the disease, wherever inoculated tends to remain for a time localized, as in the skin, lungs or bowels. The forms of the disorder are malignant carbuncle (pustule), malignant (anthrax) œdema, intestinal anthrax and pulmonary anthrax.

Lesions. The morbid histology is in the main the same as described in animals. In the protracted cases there is the same dark nonærated blood, forming a loose coagulum, the crenated or distorted blood globules aggregated in irregular masses, the escape and solution of the hæmoglobin so as to stain the white tissues, the enlargement of the spleen which is gorged with dark blood, and the hyperæmia of the liver and lymph glands. There is in the affected tissues and usually in the blood, the characteristic large bacillus anthracis.

In the *malignant carbuncle* (vesicle) there is first a minute, firm central dark nodule like an insect bite with a lighter colored areole, and showing not only hyperæmia, but blocking of the capillaries, and minute areas of extravasation. Somewhat later the dark centre is surmounted by a small vesicle, beneath which the tissues are becoming necrotic, and the area of congestion and extravasation has extended and thus the local disease advances by a constant invasion of new tissue which in its turn becomes the seat of coagulation necrosis. On microscopic section the central necrotic part shows the cells of the rete Malpighi separated by a finely granular coagulum, and the papillæ are greatly swollen by serous and hæmorrhagic exudate. The cell nuclei are necrotic and no longer take a stain. The capillaries are gorged with red globules and bacilli. In the surrounding tissues there is much congestion and exudation, with numerous points of extravasation, but the abundant multinuclear cells retain their staining power.

In *Anthrax Œdema* which appears in parts like the eyelids,

neck and forearm where there is an abundance of loose connective tissues and a scanty blood supply, there is no hard central nodule, but a diffuse soft infiltration, with points or patches of a yellow or reddish color. The capillaries are congested with minute emboli and extravasation and there is an excessive and rapidly spreading exudation. It shows a great tendency to early general infection and may end in vesication and local gangrene or in favorable cases in resolution.

In *Intestinal Anthrax* (intestinal mycosis) the lesions are usually concentrated on the small intestines, while the stomach and large intestines in the main escape. The walls of the bowel are of a dark red, and greatly thickened by exudation and extravasation, which also mixes with the ingesta giving it a dark bloody tinge. At intervals on the mucosa are nodular hæmorrhagic swellings from the size of a linseed to a pea, with commencing necrotic changes or the formation of sores. The mesenteric glands are swollen, infiltrated and hæmorrhagic and like the other lesions abound in bacilli. Hyperæmia and engorgement of the liver and above all of the spleen are the rule.

In *Pulmonary Anthrax* (wool sorter's disease) a sanguineous liquid is found in the lower trachea and bronchia, and not infrequently in the pleuræ and pericardium. The bronchial glands are swollen, hyperæmic and often hæmorrhagic, and exudations and extravasations may be found in the mediastinum and lungs. Lesions of the intestines and spleen are common, and in all alike the bacilli are found.

In certain cases the anthrax lesions are found in the brain, or any part of the body, but in all they show the same general characters and the same specific microbe.

SYMPTOMS : DIAGNOSIS.

Malignant Vesicle (pustule). Symptoms vary somewhat but are in the main as follows : An itching papule appears in the seat of inoculation, which might be mistaken for an insect bite, but for the dark red color of the centre. Occurring on an uncovered portion of the skin, in an anthrax district or near a factory where anthrax products are likely to be used, this should at once create suspicion. Soon the dark centre is covered by a small vesicle, with clear contents which later become bloody.

Within 24 or 48 hours the vesicle dries up, becoming firm, resistant and brownish red or blackish gray, and apparently gangrenous. The swelling has meanwhile extended to ½ or ¾ inch in diameter and a row of fresh vesicles may appear which in their turn give place to a necrotic slough. In this way extension may take place, the sore retaining a more or less rounded form, and necrosis extending from the center in every direction. The necrotic mass, however, remains firmly adherent to the adjacent tissues until separated by the work of suppuration which ensues in favorable cases. The disease is attended with more or less fever, chill, hyperthermia, nausea, diarrhœa, with aching of head, back, and limbs and unfavorable cases may merge into acute and fatal general anthrax. The mortality is about 20 per cent., though in special epidemics it has reached 80 per cent. (with the pustule on the face 25 per cent.; on the lower limb 5 per cent, Norris). The prognosis is favorable with a free concentration of leucocytes, a moist condition of the wound and above all a liberal invasion of pus cocci. It is unfavorable when the wound is dry, when the drying slough remains firmly adherent and when the adjacent lymph glands become implicated. In nonfatal cases it may be difficult to find the bacillus.

Anthrax Œdema. This is less easily diagnosed than malignant vesicle, and appears where the connective tissue is loose, abundant and little vascular, from direct local inoculation, or as a concominant of internal anthrax. It is a flat, rapidly extending swelling, with the skin comparatively unaltered, though at points yellowish or reddish discoloration indicates congestion and extravasation. Not being limited by firm tissues nor aggregations of accumulating leucocytes it tends to a speedy general infection with all the febrile manifestations of that condition. Thus chills, nausea, hyperthermia, dusky reddish or brownish mucosæ, cephalalgia, rachialgia and profound prostration assist in diagnosis. The bacilli in the blood and exudate would serve to confirm the conclusion.

Intestinal Anthrax. Here again the ingestion of anthrax products, and the simultaneous attack of a number of people who have eaten such materials will often assist in diagnosis. There may have been for some days indications of local bowel lesions, such as chilliness, elevation of temperature, nausea,

headache, and giddiness. Suddenly these become more violent, there is vomiting and sanguineous diarrhœa, extreme anxiety and debility, cyanosis, dyspnœa, and it may be the appearance of petechiæ on the skin and mucosæ or even of local swellings. In some cases there are convulsions or other symptoms of nervous disorder and in others extreme prostration and collapse. The bacillus is not always to be found in the circulating blood, but may be detected in sanguineous excretions, or by cultures.

Pulmonary Anthrax. (Woolsorter's disease). Here again the occupation of the patient assists in diagnosis. For two to five days prodromata similar to those of intestinal anthrax may be noted. The difficulty in breathing, dyspnœa, cough, cyanosis and sense of constriction of the chest are especially diagnostic. Suddenly all these symptoms are aggravated, respirations become 30 to 40 per minute, the pulse 120 to 150, the temperature 104° to 106°, and there is a frothy bloody expectoration in which the bacilli may be detected. There may be indications of intestinal, cerebral or nephritic lesions, and bloody discharges. Death usually occurs in 12 to 48 hours from collapse, or coma, from asphyxia or in convulsions. The few recoveries are tardy and tremors and spasms persist for a length of time. In the most favorable cases the disease does not proceed beyond the initial stage.

PROPHYLAXIS AND TREATMENT.

Prevention is the most important consideration and this will include all that has been stated above with regard to the restriction of the disease in flocks and herds, the drainage and improvement of anthrax lands, the seclusion, destruction, deep burial or cremation of carcasses without autopsy or incision, the disinfection of stalls, secretions and all contaminated products, and the suppression of all traffic in anthrax products—meat, milk, blood, guts, bones, horns, hoofs, hair, wool, bristles, etc., or the thorough disinfection of the same. Above all, is the adoption of personal precautions. No one should handle anthrax animals, nor suspected products who has any sore or abrasion on hands or face, or such sore may be temporarily covered with a film of albuminate of silver, or the hands may be washed with a solution of mercuric chloride (1 : 500), or chloride of lime (1 : 200). If

persons must work in wool or textile products which are open to suspicion a respirator is an obvious precaution, and this may be disinfected by live steam at intervals.

Treatment of malignant pustule is mainly surgical. At the outset the thorough destruction of the dark central point or nodule with a red hot needle or powerful caustic will be sufficient. Even when the pustule is fully formed, its free excision with as much of the surrounding infiltrated tissue as can be safely accomplished and the free application of caustics will usually succeed. Potassa fusa, or zinc chloride (1:3), or mercuric chloride or iodide in powder with or without calomel, or pyoktanin, or formaline, or iodized phenol may be named as especially applicable. Injections of carbolic acid (5 or 10:100) into the indurated centre and infiltrated periphery have proved very successful. In the case of Kaloff, when the excision of the nodule followed by the local use of carbolic acid solution, failed to prevent implication of the inguinal and pectoral glands, violent fever, prostration, and diarrhœa; the excision of the affected glands and the free use of phenic acid solution (5:100) in the adjacent tissues led to speedy improvement. Some surgeons make a crucial incision of the pustule and apply caustics freely. Muskett has been successful in excising the nodule, filling the wound with ipecacuan powder and giving the same agent internally. Many mild cases, or those that occur in refractory systems will however recover spontaneously or under a less drastic treatment. In the anthrax districts of Russia mercurial ointment is rubbed on the sore, and the application of tincture of iodine or iodized phenol to the raw sore or incised nodule and surrounding infiltration is often successful.

Camescasse has claimed great success by incising the swelling, applying tincture of iodine freely, and then wrapping in cloths kept wet with a solution of 5 drachms of bicarbonate of soda in a quart of tepid water.

When systemic reaction has set in it is desirable to have resort to general medication as for internal anthrax.

Treatment of Anthrax Œdema must follow the same rule. Free incisions into the œdematous tissues with the application of antiseptics, solution of mercuric chloride or biniodide (1:1000), or the injection of the whole infiltrated area and around it with the

same agents, with phenic acid (5 or 10:100), or with pyoktanin (1:1000) will prove useful, and as in the malignant pustule the surface should be kept disinfected by a compress wet in solution of the mercuric chloride or biniodide, carbolic acid, iodized phenol, formalin or pyoktanin. If the pain of these applications is very acute cocaine will be demanded or even ether. If ether is applied to the surface its evaporation will cool the parts and retard the proliferation of the bacillus. Under other conditions cold water, pounded ice or snow may be applied.

Treatment of Intestinal Anthrax. When anthrax flesh has been eaten, or when there are symptoms of incipient intestinal anthrax, the first resort is an emetic of ipecacuan, followed by an active oleaginous purgative to clear the *prima viæ* of bacilli and their toxins. To these may be added potassium iodide, pyoktanin, sodium salicylate, quinine or tincture of muriate of iron, by way of keeping in check the multiplication of bacilli. To counteract depression and heart failure digitalis, strophanthus or strychnia with alcoholic stimulants may be resorted to.

In *Pulmonary Anthrax* the same principles are applicable. The patient may be made to cautiously inhale gaseous chlorine, iodine or bromine or a solution of iodide of potassium in an atomized condition. The vapor of carbolic acid, eucalyptol, or oil of cinnamon may be tried.

The irritable stomach may be soothed by oxide of bismuth, with milk, beef tea and other bland nutritive or stimulating draughts.

The grave character of internal anthrax, however, is such that resort may be had to one of the various anti-toxins, antidotal cultures, serums, and immunizing agents that have proved useful in different hands. Unfortunately such agents do not seem to act in the same manner on all genera, and what has been effective in one of the lower animals may fail in the human being. Thus Roger found that sterilized cultures of bacillus prodigiosus retarded or obviated anthrax in rabbits, but hastened its progress in the Guinea pig.

The sterilized cultures of the pneumococcus of Friedländer (Buchner) or of the staphylococcus pyogenus aureus, (Pawlowsky) when injected subcutem have proved antidotal to anthrax. Emmerich has successfully used erysipelas serum subcutem in the treatment of anthrax. (Münch. Med. Woch.

1894). The sterilized cultures of the streptococcus erysipelatos therefore offer themselves as promising curative agents. The same is true of the sterilized cultures of the bacillus pyocyaneus (Woodhead and Cartwright-Wood), B. Coli Commune, and B. Enteritidis (*Gilruth*).

The blood serum of animals that are naturally immune (frog, white rat, pigeon, dog), is bactericidal and to a certain extent antidotal to the bacillus anthracis, but that of an animal which is naturally susceptible but which has been artificially immunized has proved much more potent. In the experience of the writer this potency attaches no less to the blood of an animal in the advanced stages of the disease. In adult cattle he has found the symptoms of anthrax subside under two successive daily doses (4 cc.) subcutem of the sterilized blood serum of one of the herd which had just died. Relapses were observed several days after the serum treatment was abandoned. In experiments on rabbits two check animals with anthrax inoculations died at the end of 3½ and 4 days. Of six inoculated with anthrax and injected from one to three times with sterilized (anthrax) blood serum one recovered, and the others died, one at the end of the 7th, two of the 6th and two of the 5th day. In this connection it may be stated that successful treatment by leucocytes is claimed, (Pawlowsky) and that one of the effects of serum treatment is the destruction in part of the globules and the release of nuclei, and in this we may have an explanation in part at least of the therapeutic action of the serum from the infecting and immunized animal.

Protective serums may be utilized by hypodermic injections daily or every second day for a week, giving time for the disposal of the bacilli present in the system. In the intestinal anthrax they may be given by the mouth and injected into the peritoneal cavity. In pulmonary anthrax they may be introduced into the trachea, bronchi and pleural cavity.

GLANDERS.

Synonyms. Definition: Acute, infectious, microbian disease, often localized in lymph glands and plexuses of nose and air passages, etc.; with hyperplasia, degeneration, necrosis, liquefaction. Affects solipeds, and, by inoculation, man and all domestic animals save cattle, chickens, and (usually) swine. Geographical distribution and historic notes: known in Ancient Greece; now where solipeds live and fresh subjects are exposed; Central Europe; great horse trade and movement; war, Franco German, Napoleonic, Afghanistan, American Civil War, Boer War. Unknown in Australia. Susceptibility: solipeds, Guinea pig, rabbit, goat, cat, dog, pigeon, sheep, and swine in low condition. Cattle, chickens, white and house mice, linnets, chaffinches, and frog immune. Microbe lives in frog in water at 86° F. Cause: Bacillus mallei. Accessory causes: trade in solipeds, mingling of sound and sick, crowding, common feeding and drinking troughs or buckets and racks, debility, low condition, starvation, overwork, damp, dark, draughty stables, carriage in transports or cars. Insular quarantined lands—Australia, Tasmania, and New Zealand exempt. Bacteriology: Bacillus Mallei; 2 to 5μ by 0.5 to 1.4μ, non motile, ærobic grows in ordinary culture media, stains tardily but is easily bleached. Spores uncertain, easily killed by disinfectants, preserved in stables, does not grow in infusion of hay, straw or horse manure, lives 15 to 20 days in water; infection by coitus, and through placenta, by ingestion, by blood transfusion, through dust; microbe in all lesions and discharges (unless sometimes in milk, sperm, etc.). Infection-atria: skin wound, mucosa, hair follicles, lungs. Forms: Acute, chronic, nasal, pulmonary, cutaneous (farcy), orchitic, arthritic, abdominal, occult. Symptoms: nasal; incubation, 3 to 5 days, languor, weariness, stiffness, horripilation, tremors, inappetence, thirst, hyperthermia, epiphora, snuffling, nasal discharge, serous, viscid, agglutinating, purulent, swollen alæ, violet mucosa, elevated spots and patches with central ulceration, may become confluent, and involve submucous tissues; submaxillary lymph glands swollen, nodular, not tender, non-suppurating, swollen (corded) facial lymphatics, from nose, eye or lymph glands; swellings, cutaneous and lymphatic in skin of limb or body, ulcers (farcy buds), deposits in throat or lungs; chronic cases; slow, indolent, persistent, nasal discharge—unilateral (or bilateral), viscid or not, nodules on mucosa with whitish centres or points; red areolæ, later ulceration, puckered white cicatricial lesions, submaxillary swelling, nodular, large or small, insensible; cutaneous cases; arthritis with lymphangitis, skin engorgement, corded lymphatics with ulcerating nodules, sanious discharge, intermuscular abscesses. Occult cases: lesions in internal organ; —cough, leucorrhœa, enlarged testicle, low condition, weakness, lack of endurance. Diagnosis: inoculation of male Guinea pig in flank or peritoneum,—ulcer and orchitis, cat, dog, old soliped; mallein test,—swelling,

involving lymphatics, fever, 1.5° to 2.5° F. and upward above normal, at 10th to 18th hour, lasting two days; agglutination. Lesions: cell proliferation in nests infibrous stroma, pea upward, central degeneration, fatty debris, ulcer or abscess, hyperplasia of lymph vessels, on nasal mucosa like sand-grains, peas, patches, centre grayish or yellowish, blood extravasation, necrotic degeneration, ulcer with ragged edges; fibroid degeneration—cicatricial lesion; lesions in guttural pouch or tube, larynx, trachea, bronchia; lungs—peribronchial, lobular or interlobular inflammation, cell proliferation in foci, degeneration—nodules—and caseation; skin,—cell proliferation, degeneration, rupture, fibroid hyperplasia of lymphatics, exudates in connective tissue; dependent lymph glands congested, hypertrophied, cell proliferation, caseation; lesions in pharynx, spleen, kidney, heart, brain, testicle, scrotum, mammæ, vagina, uterus, joints, bones; bone fragility. Glanders in swine, sheep, goat, rodent, dog, cat.

Synonyms. Malleus, Equinia, Farcy.

Definition. An acute infectious disease caused by the bacillus mallei, which tends to localize itself in the lymphatic glands and plexuses, especially of the nose and upper air passages but also in other parts of the body, where it produces a progressive hyperplasia, with a strong tendency to degeneration, necrosis, and liquefaction. It occurs casually in horses, asses, mules and other solipeds, and is communicable to man and all domestic animals except the bovine races, chickens, and, under ordinary circumstances, swine.

Geographical Distribution. Glanders (Malis) appears to have prevailed in asses in Greece as noted by Aristotle. Its contagious prevalence in horses is recorded by Absyrtus in the time of Constantine, and again by Vegetius Renatus in 381 A.D. At the present time its existence is almost coextensive with the equine family, but its prevalence is in a direct ratio with the facilities for the infection of fresh subjects. In the central countries of Europe where the equine population is greatest and where there is the most extensive trade and movement among horses it secures the greatest relative number of victims. War with its constant opportunities for infection, in crowded cavalry and artillery stables and the successive changing of place, tends greatly to enhance its ravages. Thus in the German army it rose from 966 to 2058 per 100,000 per annum in the year of the Franco–German war; in Spain it was practically unknown until the Napoleonic war in the Peninsula, but after this it proved a veritable scourge; in Hindostan it was hardly known until the Sepoy rebellion yet

its ravages greatly hampered the army movement in Afghanistan in 1879; from the United States it entered Mexico in army horses in 1847, and became very prevalent in the armies during the Civil War of 1861–4, and was widely scattered over the whole country on the sale of the army horses and mules. Since that time, as before, it has been most prevalent in the car stables of our great cities, though it has also gained a wide extension in many great horsebreeding establishments in the Rocky Mountain region, where however it proves much less destructive than in the East.

It is unknown in Australia, whence it is excluded by a rigid system of quarantine.

Susceptibility of Different Animals. Horses, asses and mules are the most susceptible, and it is only exceptionally that the disease is contracted casually outside the class of solipeds. The Guinea pig and rabbit are susceptible to glanders in the order named and the former is especially available for experimental diagnostic inoculations. The goat, cat and dog sometimes contract the disease from living in stables with glandered horses, but infection is much more certain when they are inoculated. The pigeon is also susceptible. In the dog the disease is rarely fatal, but the ulcerations tend to heal in 14 days and recovery ensues. In sheep and goats too, many cases recover though in other cases an internal infection takes place followed by death. Swine are comparatively insusceptible, but they may be successfully inoculated when in ill health and low condition (Spinola, Cadeac and Malet). Cattle and chickens have uniformly proved refractory even on inoculation. White and house mice and rats, have proved immune, also linnets and chaffinches and the frog at ordinary temperature. If however the frog is placed in water at 30° C, he may be successfully inoculated and, though it does not prove fatal, the bacillus may be found in the blood and tissues after a lapse of 50 days.

Etiology. As already stated this disease is due to the presence of a microörganism, the *bacillus mallei*. Many secondary causes, however, contribute to its propagation. The activity of movement and commingling of horses has been already noticed. Crowding in close yards where the animals bite each other, snort out the virulent discharges on each other and eat and drink from the same troughs, leads to a rapid extension. Even on the west-

ern ranges where the disease tends to be mild, Billings observed a deadly extension when yarded during winter storms. Debility from chronic ill health, starvation, overwork and damp, dark, draughty stables, is so conducive to the disease that it was at one time considered as the sole cause. Close confinement in impure air is at once a cause of increased susceptibility and a means of concentration and transmission of the poison. Hence confinement, between decks, of military and other horses, carried by sea, is a source of wide extensions. On the other hand insular places from which strange horses are excluded or into which they are admitted under careful inspection and quarantine have succeeded in preserving immunity. Australia, Tasmania and New Zealand are examples.

Bacillus Mallei. Christot and Kiener claimed to have found a bacillus in the lesions of glanders in 1868. In 1881 bacilli were found by Bouchard in a glander abscess in man, and these were cultivated *in vitro* and inoculated in a number of animals, by Capitan and Charrin in 1882. Independently in the same year (1882) Löffler and Schütz discovered the bacillus, cultivated it *in vitro*, and successfully inoculated it on animals. The microbe is rod shaped, 2 to 5μ long, by 0.5 to 1.4μ thick, the same length as the bacillus tuberculosis but thicker. It is non-motile, ærobic (facultative anærobic) and grows readily in a variety of culture media at a temperature of 37° C. On neutral bouillon of the flesh of horse, ox, calf or chicken with or without peptone, it grows readily, producing cloudiness in one or two days. In peptonized gelatine it forms a whitish flocculent mass. On glycerine agar with milk it forms in 48 hours a milk white layer, changing to yellowish brown. On potato it forms long slender filaments, in yellow, viscous, glistening colonies, changing to fawn and darker. It grows best at 35° to 39° C. and growth ceases below 25° C., and above 42° C. It stains tardily in aniline colors, and not at all by Gram's or Weigert's, but will readily take Kuhne's stain prepared as follows : take of phenic acid in solution (5:100) 50 cc., absolute alcohol 10 cc., and 1 to 2 grammes methylin blue. The stain is very easily bleached by acid, differing in this from the bacillus tuberculosis. For decolorizing Löffler recommends 10 cc. distilled water, 2 drops of strong sulphuric acid, and 1 drop of a 5 per cent. solution of oxalic acid. Sections should be

left in this not longer than 5 seconds. The bacillus often appears granular, and unequally stained in its different parts. It may be difficult of discovery in old standing lesions of horses, but comes out clearly in recent lesions of experimental cases in Guinea pigs and donkeys.

Baumgarten claims sporulation but this is uncertain.

The bacillus has only limited power of resistance to destructive physical and chemical agents. It is killed in 10 minutes at 55° C., in 2 minutes at 100° C., or by mercuric chloride solution (1:5000), or by phenol (5:100), or by permanganate of potass (1:100). In warm dry air and sunshine it is sterilized, in thick layers in 2 months (Peuch), in moderate layers in 4 to 15 days (Galtier), and in very thin layers in 3 days (Cadeac and Malet). In moist, cool air and in the shade it is much more resistant. In stables it may remain virulent for three or four months, and thus the disease has often reappeared among the newly introduced horses after a stable has been abandoned for a length of time. The microbe does not grow in infusions of hay, straw or horse manure, and it is doubtful if it can maintain an active saprophytic existence. Its vitality and virulence, however, persists in putrefying materials for 14 to 24 days, and in winter from 15 to 20 days. Hence it is largely propagated through drinking troughs and occasionally through ponds, lakes and sluggish streams. Again the virus is likely to be preserved in and transmitted by rotten or even sound woodwork, as of mangers, racks, buckets, shafts and poles, and by harness, halters, blankets, combs, brushes and rubbers. Sometimes direct transmission takes place in snorting or coughing, or by the animals biting or licking each other. As the virus is spattered on surrounding objects, the walls, stable utensils, soiled fodder and feed, and even the attendant's clothes may be the medium of transmission. Contagion during copulation is not unknown, nor infection of the fœtus *in utero* from a diseased mother. Carnivora (dog, cat, lion) fed on the diseased carcasses have become infected. Experimentally infection has been conveyed by administering, by the mouth, balls containing the virus (Renault, Coleman, etc.), and again by transfusing the blood from a bad case of glanders into the veins of a sound horse (Viborg, Coleman, Renault, Hering, Chauveau, Nocard). Transmission through the air on dust is counteracted by the speedy destruction of virulence on dust, and horses often stand side by side

in adjacent stalls for months without communicating the infection. Such escapes may, however, in some of these cases, be attributed to the immunity secured by a previous exposure and slight attack. That the germs may be exceptionally conveyed through the air appears to have been proven by Viborg and Gerlach, who collected the floating dust in a stable containing infected animals and successfully inoculated with it.

The microbes are especially found in the visible lesions of glanders, in the mucous, cutaneous and subcutaneous swellings and abscesses, in the swollen lymph glands, in the nodules and ulcers of the mucous membranes and skin, in the morbid discharges, from the nose, eyes, pharynx, guttural pouches, larynx, trachea and bronchia, and in the discharge of farcy buds and abscesses. They are not necessarily distributed through all the tissues, and in chronic cases, with strictly local lesions, the infection appears to be often confined to these or nearly so, and the contamination of other animals is slow and uncertain. When, however, the disease is acute and advanced, or generalized, every part must be looked upon as probably infecting. Thus virulence has been shown in the blood, the exhalations of the serous membranes, saliva, the aqueous humor (Cadeac and Malet), the tears, (Viborg), the muscles, and the bones. Galtier says the milk, sperm, bile and intestinal mucus are non-virulent, also the vaccine lymph raised on glandered animals, but much must depend on the grade and stage of the disease and no one would care to run unnecessary risks with these liquids.

Infection Atria. Inoculation on a skin wound or abrasion, is a most effective mode of transmission, but the virus undoubtedly enters in certain cases with the air, food or water, or by accidental lodgment of a speck of the virus on the mucosa of the nose or eye or other natural opening. Through the healthy mucosa the bacillus may enter by penetrating the soft epithelium, or entering the mucous follicles, but it will find the way smoothed for it, if there has been friction, abrasion, desquamation or congestion. The skin in its healthy state is usually resistant, but Babes has conveyed infection by rubbing on the virus mixed with vaseline, and without displacing the epithelium. The channel of entrance was the hair follicles. Injected into the blood, intravenously, or into a serous cavity, the bacillus infects with great certainty and promptitude.

There is ample evidence that primary lesions appear not only

in the skin and nasal mucosa, but also in the bronchia, intestines, and other parts. Much time and ink have been wasted in attempts to prove that pulmonary lesions are always secondary, and doubtless many are so, but the cases in which the lesions are confined to the bronchia or parenchyma, and the many parallel cases of direct infection of the lungs by other diseases (tuberculosis, lung plague, contagious pneumonia, influenza, etc.,) furnish conclusive enough evidence that the germ may be inhaled and colonize this part first. Cases of inhalation bronchitis, are equally corroborative, and not only may the grosser solids be aspired, but infecting droplets can easily gravitate down when from congestion or inflammation the action of the tracheal and bronchial cilia, is suspended.

FORMS AND LESIONS OF GLANDERS IN SOLIPEDS.

Glanders appears in two primary forms—*acute* and *chronic*, and each of these is further divided according as the lesions are exclusively or mainly seated in one part of the body or another. The generic term *glanders* is habitually used to designate that form in which the lesions are situated in the nose, the nasal sinuses, and the sub-maxillary lymphatic glands—*nasal glanders*. When the principal leisions are situated in the lungs and lymph glands of the chest, the case is one of *pulmonary glanders*. When the skin and subcutaneous lymphatics are more prominently affected it is known as *farcy or cutaneous glanders*. When the skin and nose are simultaneously affected the name *farcy glanders* is sometimes applied. But as the bacillus may enter by very varied channels the primary lesions may appear in still other organs. Thus in stallions the first symptom is often a glanderous orchitis. In other horses it may be a glanderous arthritis, and in still others infected by ingestion it may be an abdominal infection.

Symptoms of Nasal Glanders in Solipeds. Acute. After an incubation of three to five days the subject shows prostration, weariness, stiffness, erection of the hair, and even tremor or shivering, inappetence, thirst, hyperthermia, rapid pulse, weeping eyes, the discharge becoming purulent, snuffling breathing, and a discharge from the nose, at first serous, with a remarkable viscidity which tends to glue together the long hairs or even the margins of the nostrils. This discharge may be reddish, greenish, or brownish and may become distinctly purulent and opaque. The *alæ nasi* are swollen, hot and painful, and the mucosa red,

congested, thickened, with a blackish or violet tint especially along the median part of the *septum nasi.* On these, violet patches appear on the second or third day, pronounced elevations of very varying size indicating the centres of active hyperplasia. They are usually yellowish or grayish, surrounded by a deep violet areola, and may become confluent, forming patches. The centre of each undergoes rapid degeneration, forming a rounded ulcer with salient edges, a yellowish base, more or less pointed or streaked with red, and a viscid sero-purulent or bloody discharge which may concrete in crusts or scale. The whole septum may become one continuous ulcer with excavations of various depths surrounded by hyperplastic elevations, and involving not only the mucosa, but even the cartilage and leading to perforations.

From an early stage of the attack the submaxillary lymphatic glands and the investing connective tissue become swollen, forming a mass of firm bean, or pea-like nodules, with no excessive heat nor tenderness, and with little disposition to suppurate and discharge. If this has lasted for some time the glands often become more firmly attached to adjacent parts (maxilla, tongue) by the contraction of the exudate.

The swelling of the alæ nasi also often extends to the skin of the face, and firm, rounded cords formed by the swollen lymphatics stretch upward toward the eye, or the submaxillary glands. Upon the turgid lymphatics may appear more or less rounded nodules from the size of peas to hazel nuts, which, unlike the submaxillary glands, tend to soften, burst and discharge a viscid, glairy, sanious liquid.

At the same time the morbid process is liable to show itself in the cutaneous lymphatics of one limb, usually a hind one, in the form of firm cords, with degenerating or ulcerous nodules (farcy buds) and pasty patches. Or the throat or lungs may become involved, with local swellings, violent cough, dyspnœa and fever. The swellings of the cutaneous lymphatics usually follow the course of the veins, in the hind limbs the branches of the saphena, and extend from below upward, and the first nodules may be on the fetlock or hock.

Symptoms of Chronic Nasal Glanders in Solipeds. The chronic form of the disease follows an indolent course, and local symptoms are often so slight or equivocal that the true nature of the malady is unsuspected. If the patient is well fed and cared for and not overworked, the malady may run a course of three, five

or seven years, and the victim may pass through many hands leaving infection in every stable it occupies. Diagnostic symptoms, more or less clear, may be obtained from the discharge, the lesions of the mucosa and the submaxillary glands.

The *nasal discharge* may be bilateral, but if confined to one nostril is strongly suggestive of glanders. It may be profuse or scanty, continuous or intermittent, of a yellowish, purulent tint, or greenish, or grayish and with a special tendency to viscidity. In some indolent cases the nostrils may be clean but if there is any matting of the long hairs, or adhesion of the alæ nasi, the case is specially suspicious. If it is sanious, flocculent, or bloody it is all the more characteristic, and suggests the supervention of an acute attack.

The *lesions of the pituitary membrane* are varied. Hyperæmia of a purple or violet color is common, especially along the septum, and the mucosa is liable to be somewhat tumid or œdematous. Nodules the size of a pin's head, a pea or larger appear inside the inner ala, or on the septum or turbinated bones, and at first red from extravasation and, as it were vesicular, become grayish, whitish or yellow with points of red and surrounded by a deeply congested areola. Larger nodules forming in the submucosa approach the surface and stand out the size of the tip of the finger and with the same general character as the smaller. Sooner or later these degenerate and form ulcers which bear a resemblance to those of acute glanders but are less angry, and when small and solitary may be taken for simple erosions. In other cases they become thickened and indurated with sharply defined projecting margins, and a yellowish base with points or lines of red. The presence of red, black, green, or brown crusts may also be noted.

Another lesion frequently observed in indolent cases is a cicatricial white spot or patch in which the hyperplasia has become partially developed into tissue and shows no tendency to ulcerate. The mucosa may even be drawn or puckered around the cicatrix, making the illusion all the more complete.

The *submaxillary swelling* is even less sensitive than in acute glanders and produces the same sensation as of an aggregation of small, hard, pea-like masses with no tendency to ulcerate.

Symptoms of Cutaneous Glanders (Farcy) in Solipeds. Acute cutaneous glanders has been already referred to under nasal glanders. The chronic type is often less characteristic, yet may be detected by careful observation of the symptoms. The main

symptom may be the swelling of a joint with more or less engorgement of the limb from attendant lymphangitis. There can usually be detected around the margins of such swellings firm, tender cords, representing the larger lymphatic vessels and often branching in their course. In the absence of the engorgement, or when it is slight, these *cords* may be the main evidence of the disorder, and in the hind limb usually follow the course of the flexor tendons on the inner side of the digit, metacarpus and thigh. At intervals along the line of the *cords* appear nodular masses (farcy buds) varying in size from a pea to a hen's egg, and showing a great disposition to soften and discharge a glairy, sanious or more or less bloody liquid. The inner sides of the fetlock and tarsus are favorite seats of these nodules but they may form at any point. On the trunk also the corded lymphatics and nodules follow the lines of the veins and lymphatics, and here there may be the complication of large intermuscular abscesses often in connection with the groups of lymphatic glands.

Latent or occult glanders is often met with, the indolent, specific lesions being confined to some internal organs, like the larynx, lungs or womb, or to the testicles, the nasal diagnostic symptoms being absent. A chronic cough, with a slight purulent discharge from the nose, a chronic leucorrhœa, a swollen testicle, or simply a persistent low condition or weakness without apparent cause, may be the only indications, and special means of diagnosis are demanded.

Special Means of Diagnosis. In occult cases, the disease may be identified by inoculation, or by the mallein or agglutination test.

Inoculation is best performed on a very susceptible animal. If the suspected discharge from the nose, vagina, open sore or preferably from a freshly incised nodule is inoculated subcutem in the flank of a male Guinea pig, or better in the peritoneum, there develops a local ulcerous sore and on the second and third day a violent orchitis in which pure cultures of the bacillus can be obtained. The caseous and purulent centres are found not only in the testicle but along the line of the spermatic cord, affecting the tunica vaginalis and connective tissue. Death usually follows in four to fifteen days. The cat and dog can also be utilized, inoculation being made on the forehead. Old wornout, but otherwise healthy asses, and even horses make very available subjects, inoculation in the nose speedily developing acute glanders. In

the absence of a good subject the suspected animal is sometimes availed of, scarifications being made in the nose and the morbid product rubbed in freely. The rapid development of ulcerous wounds is characteristic. If, however, the case is chronic, and if a fair measure of immunity has been acquired this test may prove misleading.

Test by Mallein. Mallein is the sterilized and concentrated toxic product obtained from a pure culture of bacillus mallei in a peptonized glycerine bouillon. When injected hypodermically in a small physiological dose this has no effect on a sound horse, but in one affected with glanders it develops in several hours an extended swelling in the seat of inoculation, hot, tense and painful, which continues to enlarge for 24 to 36 hours and does not subside for 4 or 5 days. From the margin of the swelling, swollen lymphatics may often be traced running toward the adjacent lymphatic glands. There is also decided dulness, prostration, inappetence, staring coat and tremors. The body temperature rises 1.5° to 2.5° and upward from the eighth hour after inoculation, attaining its maximum from the tenth to the eighteenth hour and subsiding slowly to the forty-eighth to the sixtieth.

Mallein must be used under precautions like tuberculin. It must be obtained freshly prepared from a reputable maker. If preserved for months its force may be largely lost. The animal to be tested should be in his customary environment, and not just arrived from a railroad journey nor other cause of excitement. He must not be fevered as any rise of temperature is then equivocal, and a fall of temperature, which sometimes occurs in the febrile system under mallein, is no sure evidence of glanders. Reaction sometimes fails in advanced cases of glanders, but in such a case other symptoms are usually diagnostic so that mallein is superfluous and should not be misleading. The greatest care should be taken to prevent infection from the syringe, nozzle, skin, hands, etc., as other infections may give rise to local swelling and hyperthermia (see tuberculin test). If a first test leaves the matter in doubt, the animal should be secluded and tested again in a month (some prefer 3 months).

Test by Agglutination : Serum-test. In this a culture of Bacillus mallei is made in nonglycerined, peptonized bouillon. After 2 or 3 days growth, this is sterilized by heating to 60° C. for two hours. It is then diluted in carbolized, physiological salt solution, to an extent of not less than 1:10, nor more than

1:1500. The dilution is mixed in equal proportions with the pure blood serum of the suspected animal and placed in an incubator at 37° C., for 24 to 30 hours. By this time the serum of a glandered horse will have caused agglutination of the Bacillus mallei and the precipitation of the opaque bodies, leaving the supernatant liquid clear and translucent. The blood-serum of a healthy horse has no such effect, the liquid retaining its opacity throughout. The test is not always available to the average practitioner, as it requires laboratory apparatus and facilities, but, when the means are at hand, it may be employed to confirm the results of the mallein test, or independently in cases attended with marked fever and thereby ill adapted to the mallein test.

Pathological Anatomy. The colonization of the bacillus mallei in a tissue usually determines a concentration and multiplication of leucocytes, so as to form rounded nests of small lymphoid cells in a scanty fibrous network. These may be miliary or by aggregation they form masses the size of a pea or larger, which bear a close resemblance to the neoplasms of tuberculosis. As in tubercle the central cells of the group, degenerate, forming a granular fatty debris, and constituting an ulcer or abscess. In certain cases with a proliferation of fibrous tissue a cicatricial material is developed. Another characteristic lesion is the occurrence of hyperplasia in the walls of the lymph vessels so as to constitute firm tender cords, and the infiltration of the adjacent lymphatic plexus.

In the *nasal mucosa* the bacilli form prolific colonies at different points of the membrane and submucosa with the active production of lymphoid cells, followed by granular fatty degeneration and ulceration. Hence may be found different lesions representing the different stages. First there may be miliary deposits with clear contents and standing out like grains of sand. Then there are the larger pea-like nodules with congested vessels and minute hæmorrhages, but made up largely of the nests of lymphoid cells. These may bear on the surface a distinct blood extravasation, or the epithelium may be raised from the corium layer by a liquid exudation. The more advanced nodules show the centre light colored, grayish or yellowish with a distinct granular degeneration of the cells. Later still the degeneration involves the superficial layers and epithelium and an open ulcer is formed with a strong tendency to extend in depth and width. The formation

and degeneration of numerous foci of cell proliferation gives the ulcer a very uneven outline. The continuous growth of fresh centres of proliferation may cause marked elevations between the ulcers, constituting extended patches, or the entire nasal mucosa may be thickened as the result of the morbid deposit. The cicatrices resulting from the apparent healing of deep or extensive ulcers or from a fibroid transformation of the neoplasm consist of condensed connective tissue with small scattered nests of lymphoid cells and bacilli. In chronic cases the bacilli are very scanty.

The mucosa of the *Eustachian pouches and tubes, the larynx, trachea and bronchia* often present lesions similar to those of the pituitary membrane.

The *lungs* are usually marked in chronic cases by circumscribed lobular pneumonia, interlobular and peribronchial inflammations and miliary or larger areas of degeneration resembling tubercles. These may begin as a minute congestion and ecchymosis, which later shows in the centre a translucent or gray mass of lymphoid cells, with a surrounding area of congestion. Later still this central mass becomes yellowish and caseated from granular and fatty degeneration and this gradually extends so as to involve the whole area of the nodule. The peripheral portion may condense into a fibroid envelope, but usually this is less smooth and evenly rounded than in the case of an inspissated abscess or bladder-worm. The bacilli are found in the affected tissue but not always abundantly.

In *cutaneous glanders* the lesions may begin in the papillary layer by active congestion and infiltration and proliferation of lymphoid cells which cause an eruption of rounded papules like small peas that degenerate and soften and form superficial ulcers. When the derma is mainly involved the inflamed area becomes the seat of larger hard nŏdules which are at first deeply congested, with capillary thrombi, minute extravasations and rapid cell proliferation; later on section they show numerous caseated centres with a dense fibroid framework and surrounded by an area of active congestion and capillary hæmorrhage; later still the caseation and softening has caused rupture of the investing epithelium and the discharge from the ulcerous cavity of a yellowish, glairy, grumous liquid (open farcy buds). Sometimes the nodule undergoes fibroid induration and fails to ulcerate, becoming the counterpart of the cicatrices in the nose. When the infective inflammation extends to the subcutaneous connective tissue, diffuse

engorgements and extensive swellings occur from the general infiltration of the abundant lpmph plexuses. Lymphoid cells accumulate in the perivascular sheaths and lymph plexuses, the walls of the lymphatic trunks running out of these swellings become swollen and indurated and at intervals mostly on the seat of the valves there is the proliferation of small round cells to form farcy buds. In chronic cases the fibroid thickening involves the skin, subcutaneous connective tissue and walls of the lymph vessels binding the whole into one dense resistant mass, more or less studded with corded lymphatics, firm nodules, and ulcerous sores.

The *lymphatic glands* in the line of circulation from the infected centers are constantly involved. Hepertrophy, congestion, serous infiltration, and rapid cell proliferation are present and a section will usually show caseated or caseopurulent centres confined by the outer dense fibrous envelope. Exceptionally, these necrosed contents will escape through an ulcerous opening, forming a deep cavity which is slow and difficult to heal. In the vicinity of these glands and in the loose intermuscular connective tissue abscesses of the size of an egg or an orange or larger are sometimes met with.

Nodules and ulcers are found on the pharyngeal and intestinal mucosa, similar to those of the larnyx.

The spleen, and less frequently the liver, may be the seat of caseating nodules exactly comparable to those of the lungs. Glanders of the kidney is rare.

Nodules have been seen on the ventricular endocardium and one case of nodules of the choroid plexus (Boschetti).

In stallions, glanderous, caseating foci in the *testicle* and dropsy of the scrotom are common, while mares may have similar formations in the mammary glands or ulcers of the vaginal or uterine mucosa.

Infiltrations of the joints and other synovial cavities are not uncommon and glanderous infiltration of the bones with caries is also found. In chronic cases, fragility of the bones is marked, and the blood contains an excess of leucocytes. These may be traced to disease of the bone marrow, as well as of the spleen and lymphatic system.

GLANDERS IN SWINE.

The healthy, vigorous pig is practically immune. Experi-

mental inoculations have uniformly failed to produce the disease. Exceptions must be made when inoculation is made into the aqueous humor, in which there is comparatively little resistance by leucocytes. Sacharoff succeeded in giving the disease in a fatal form to a young pig in this way. To weak and debilitated pigs, on the other hand, the disease may be conveyed as shown by Spinola, and Cadeac and Malet. The symptoms were engorgement of the tissues in the seat of inoculation with the formation of glanderous nodules, which undergo molecular degeneration and ulceration; swelling of the lymph vessels extending from the infected point, and of the adjacent lymph glands; the formation of glander nodules in the lungs, liver and spleen, and of nodules and ulcers on the nasal mucosa.

GLANDERS IN SHEEP AND GOAT.

Casual glanders is uncommon in the small ruminants, yet it has been seen in goats that fed on the soiled provender left by glandered horses (Ercolani, Trasbot, Mesnard). Sheep have suffered after inoculation (Renault, Bouley, Gerlach, Bollinger, Croker, Peuch, Galtier) showing nodules and ulcerous swellings in the seat of inoculation, with extension in some cases to the nasal mucosa and lungs, and a fatal result. In other cases the lesions remain localized and the disease progresses to recovery.

GLANDERS OF RODENTS: RABBIT, GUINEA-PIG, MOUSE, RAT.

The *guinea-pig* is especially susceptible, and like the rabbit and mouse may contract the disease by eating the soiled fodder of glandered horses. It is usually selected for inoculation for diagnostic purposes, because in the male, the insertion of the virus in the peritoneum determines a rapid swelling of the testicle and cord, with a glistening, violet engorgement and tension of the scrotum, suppurative adenitis and death in three to six days. In other situations the inoculated part swells rapidly and ulcerates, the adjacent lymph glands become swollen and tender, similar lesions form in other parts and notably in the nose which discharges profusely a whitish, sticky fluid, the breathing is hurried, and emaciation is marked. Death occurs in a few days and ulcers, and abscesses are found in the lungs and other internal organs, as well as in the nose. In less susceptible subjects or under smaller doses of the virus the local swelling and ulceration in the

seat of inoculation are slight, but there persist engorgements of the adjacent lymph glands, swelling (cording) of the lymph vessels adjacent, and nodules and abscesses, (cutaneous, subcutaneous, intermuscular, intravisceral), arthritis, emaciation, dyspnœa and death in two to four months. In some cases with a very small dose of the poison, there is no local swelling, and no generalization nor subsequent manifestation of the disease.

In the *rabbit* the lesions are less certain and often less marked. There is sometimes no swelling in the seat of inoculation, in the neighboring lymph glands, nor elsewhere, and the rodent might have been supposed to have escaped, only that successful inoculation of the ass may be made from the tissues inoculated (Galtier). In such a case a certain immunity of the rabbit must be inferred. In less resistant rabbits, or with a larger dose, an ulcerous swelling forms in the seat of inoculation, the adjacent lymph glands become engorged or even purulent, corded lymphatics intervene, and nodules and caseous degenerations appear in the lungs, nose, spleen, liver, and other organs.

The *hedgehog*, *ground squirrel*, the *field mouse*, *house mouse*, and *mole* have been successfully inoculated.

The *frog* immersed in water at 30° C., forms a good culture ground for the bacillus which may be found in its blood, in pure cultures, from the second to the fifty-fifth day. These cause no local lesion, nor obvious, constitutional disorder. It seems possible that, in summer, the infection may be propagated by frogs in the drinking water.

GLANDERS IN CARNIVORA.

All carnivora are liable to contract glanders by eating the flesh of glandered horses, asses and mules, and this has been noted especially in menageries. *Lions*, *tigers*, *bears* and *wolves*, have shown the ulcerous lesions in the nose, and the nodules in the lungs, spleen, liver, kidneys and elsewhere. The carcass of the diseased horse is, however, often devoured without evil result, and even when the carnivora become affected the disease is not always fatal.

In the *dog*, experimental glanders has been closely studied by many observers. Casual glanders has been contracted by living with the glandered horse ; by licking his nasal or other discharges,

and by eating his flesh. The disease has also been conveyed from dog to dog by licking each other. In many cases even inoculated glanders produces only a local ulcerous inflammatory lesion with or without hard swelling of the adjacent lymphatics, and engorgement of the lymph glands. After a rather tardy granulation and cicatrization, the symptoms subside and the animal is restored to health. Yet such benignity does not depend on any lessened virulence of the bacillus, for an inoculation of the discharges on the ass produces acute and fatal glanders.

GLANDERS IN MAN.

Recognized by Lorin 1812. Causes : infection from soliped, man less susceptible ; infection from man, clothing, stable bucket, inhalation, etc. ; industrial disease ; native immunity. *Symptoms :* incubation ; mistaken for carbuncle, small pox, measles, erysipelas, anthrax ; anamnesis ; anthrax focus has darker center, no caseation, no corded lymphatics ; nodules and ulcers in nose, swollen submaxillary glands and lymph vessels, general illness, diarrhœa, vomiting, dyspnœa, mental derangement, stupor, coma, internal deposits, bloody sputa, fœtid breath, hepatic pain, icterus, muscles, bones, bowels, typhoid, pyæmic, osteo myelitic, or acute tuberculous symptoms. Death in 3 days to 4 weeks. Chronic cases, cutaneous, muscular, osseous, skin nodules in group or chain, glandular swellings. Diagnosis from pyæmia by lack of chills, and the sanious pus ; from syphilis by futility of potassium iodide, and history ; inoculate ox or white mouse ; find bacillus. Lesions : as in horse, more early coagulation necrosis, ulceration, abscess ; pus more viscid than in pyæmia, walls of abscess more irregular, lymphoid cell proliferation more abundant and extended (glands, spleen, liver, lung, nose, etc.), history ; distinguished from variola, rötheln, and erysipelas by the many miliary or pea-like neoplasms with cellular caseating centres ; lymphoid deposits in bone marrow, with friability.

Until the early part of the present century glanders in man was not traced to its origin in the soliped. Lorin in 1812 recorded a case in which the human hand had been accidentally inoculated from handling a horse suffering from farcy. Soon other cases were put on record by Waldinger and Weith, Muscroft, Schilling, Rust, Sedow, and a host of followers. Later Rayer, Tardieu, Virchow, Leisering, Gerlach and Korányе have thrown much light on the subject.

Etiology Man is manifestly less susceptible than the soliped,

considering the great number of exposures relatively to the victims. Yet the infection of man is altogether too common to be lightly passed over. The infection is almost always derived directly or indirectly from the horse, yet a number of cases have been derived from the human being through handling the dishes, towels or handkerchiefs of a patient, dressing his wounds, or performing a necropsy. Other cases like that of Dr. Hoffman of Vienna, came from handling artificial cultures of the bacillus mallei.

Glanders is preëminently an industrial disease, attacking persons of the following occupations: hostlers 42, farmers and horse owners 19, horse butchers 13, coachmen and drivers 11, veterinarians and veterinary students 10, soldiers 5, surgeons 4, gardeners 3, horse dealers 2, policeman, shepherd, blacksmith, employe at a veterinary school, and washerwoman, 1 each.

The modes of transmission are essentially the same as in the animal. In the great majority of cases there has been the direct contact of the infecting discharges with a wound of the human victim. Handling the diseased horse with injured hands, giving him a bolus and scratching the hand on the teeth, examining the nose, sleeping under a blanket which has been used on a glandered horse, removing the dressings of such an animal or performing a post mortem examination on him are familar examples. The particles scattered by the diseased animal in snorting, will infect the mucous membrane of the eye or nose, and all the more readily if these are already sore or abraded. Infection of man by ingestion has been discredited mainly because the carcasses of glandered horses have often been eaten with impunity; but this may be largely accounted for by cooking, the bacillus being destroyed by a temperature of 131° F. Carnivora such as dogs, cats, lions, polar bears and prairie dogs have been infected by feeding. Men also have been infected through drinking from the same bucket after a glandered horse. After making full allowance for the inimical action of the gastric juice, we must admit that this has often failed, and there is the added danger of abrasions of the lips, mouth and throat and of the entrance of the microbe into the tonsillar follicles and gland ducts. Still other cases are recorded of men sleeping in stables, but not handling horses, who contracted glanders, presumably, through

the dust-borne bacillus inhaled. The bacillus, is however, so readily destroyed by thorough desiccation that this mode of transmission is exceptional. Some men are immune to glanders, and suffer only when predisposed through a course of ill health, and yet a large proportion of the cases on record have been in strong hearty men.

Symptoms of Glanders in Man. In man as in the horse, glanders occurs in the *acute* and *chronic forms*. In the *acute* supervening on an external inoculation, *incubation* is from one to four days. When it enters through other channels it may seem to extend to a week or more.

When a skin abrasion has been inoculated it will show in a few days a soft inflammatory swelling or a firm nodule with a puffy reddish areola, and it may be mistaken for a carbuncle. In not a few cases the small nodule has been mistaken for small pox. In my experience a horseman on a ranch on which over a hundred horses showed glanders, died of an ulcerous skin affection which was variously supposed to be a malignant small pox and measles, though neither malady was known to exist in the district. Other cases are confounded with gangrenous erysipelas. The absence of these other affections from the locality, and the fact that the patient was employed about glandered horses, should go far to correct such mistakes. The early supervention of ulceration is further diagnostic, and discovery of the bacillus mallei in the products will be conclusive. From anthrax it is easily distinguished by the absence of the dark centre of the sore in the early stages, and of the large sized bacillus anthracis. The caseation or liquefaction of the necrotic centre further distinguishes it from the characteristic anthrax slough, and the thickening and induration of the lymphatic walls are not present in anthrax. Sometimes the inoculated case proves mild and recovers in two or three weeks with healing of the ulcer, but in other cases there is an extension to adjacent tissue and a general infection with the supervention of nasal glanders.

There is a spread of the erysipelatoid inflammation and swelling, and the formation in such newly invaded tissue of nodules and ulcers in successive crops. In acute cases too, the nasal mucosa becomes involved with the formation of the nodules and ulcers that are so pathognomonic in the horse. The discharge is

then somewhat sticky and often tinged with blood. In acute cases according to Senn, the nose may be completely destroyed and deep facial ulcers may be formed in a week. The submaxillary glands are enlarged and painful and the facial lymphatic vessels leading from these to the nose may be red, thickened and tender. Suppuration and ulceration of the glands may ensue. Headache, prostration, nausea, inappetence and vomiting with diarrhœa usually supervene. Then follow dyspnœa, wakefulness, troubled dreams, anxiety, nocturnal delirium, stupor and coma. The pulse may rise to 120 and the temperature to 104° F.

There may be various complications as deposits in the lungs with pain in the chest, weak cough, aphonia, bloody expectoration and offensive breath; or the morbid process may take place in the liver or spleen with pain in the hypochondrium and much prostration and even icterus; or the muscles, bones, joints or testicle may suffer and the symptoms may suggest typhoid fever, pyæmia, osteomyelitis, or acute general miliary tuberculosis. The bacillus can usually be detected in the blood.

Acute glanders may prove fatal in three days or it may be prolonged for two, three or even four weeks.

Chronic glanders in man usually confines itself to the cutaneous muscular and osseous systems. It may take on an indolent type with the formation of skin nodules in groups or chains which remain hard and show no tendency to soften nor ulcerate. The adjacent lymphatic glands may become enlarged and indurated and the affection strongly resembles tuberculosis of the skin. Later when the nodules have softened and formed irregular and obstinate ulcers, with swollen lymphatic glands, the disease is easily mistaken for syphilis. From pyæmia and septicæmia it is usually to be distinguished by the comparative absence of chills, and by the more sanious character of the pus. From syphilis it may be distinguished by the futility of a course of potassium iodide, and the general history of the case and probable exposure of the patient, and for tuberculosis the same principles will apply. In case of uncertainty, inoculation may be resorted to on the horse in suspected syphilis and on the pig when there is suspicion of tuberculosis. Or conversely the ox may be employed for the latter disease as he is altogether insusceptible to glanders. As a last resort the discovery of the bacillus may be made or the mallein test may be adopted with the concurrence of the patient.

Pathological Anatomy and Diagnosis. This is fundamentally the same as in the horse. The bacillus and its toxic products act on the infected tissues to produce clusters of lymphoid cells in a fibrous stroma after the manner of tuberculosis. Like that disease it also tends to affect primarily the lymph channels and glands, showing a particular tendency to the respiratory mucosa and has a great disposition to early coagulation necrosis, ulceration, suppuration and abscess. The giant cell of tuberculosis is not a prominent feature in glanders, and the disposition to suppuration is greater especially in the human being so that the disease often resembles pyæmia. As in solipeds, however, the glander abscess has somewhat more sanious or glairy contents and the investing wall is not smooth and regular, but uneven and ulcerous from the successive softening and discharge of the clusters of degenerating lymphoid cells in the adjacent tissue. The pallor of the adjacent tissues from exudation and from the presence of numerous nests of lymphoid cells, the thickening of the efferent lymphatics, and the presence of numerous lymphoid neoplasms in the adjacent glands and tissues, and often in the internal organs such as the liver and spleen and in the nasal mucosa or lungs, together with the history of the patient's exposure to glanders, serve to diagnose from pyæmia. From smallpox and rötheln the skin lesions are distinguished by the presence of a central coagulation necrosis bathed in a glairy seropurulent fluid, and by the infiltration and thickening of the efferent lymphatic trunks. It differs from erysipelas in the same way by the presence in the affected tissues of the small hard lymphoid masses of embryonal tissue, and in a more advanced stage by the granular fatty debris resulting from their fatty degeneration. The presence in the affected tissue of these miliary or pea-like neoplasms in all stages of development from the primary congestion, through the embryonal tissue to the coagulation necrosis and caseation or softening is characteristic of the lesions of glanders. The sanious, sticky or glairy pus is especially noticed in the newly opened abscess, as after exposure to the air it is speedily infected with pus microbes, and the discharge becomes less serous and more creamy. Another characteristic of glanders in man is the frequent implication of the bone marrow, and the formation of the lymphoid deposits in the cancellated tissue until the bone may be reduced to a mere friable shell. Even

when the disease is localized in the nasal mucosa it extends rapidly, not only to the skin and muscles, but also to the cartilage and bones of the face, so that deep, wide, perforating and destructive ulcers are common. The enlarged ends of the long bones of the limbs are favorite seats of the lesion, and the synovial membrane of the joints and the articular cartilage often bear centres of lymphoid proliferation. Though usually small the intermuscular neoplasms may form abscesses as large as a hen's egg. The affected muscle appears pale, degenerated and granular with foci of lymphoid cell growth. The swelling of the lymph glands is usually less than in the soliped though the same in character. The pulmonary neoplasms are histologically almost indistinguishable from tubercle, though the comparative absence of the giant cell, the different staining qualities of the bacillus, and the coincident lesions in the upper air passages, with the cord like infiltration of the walls of the lymphatics may assist in diagnosis. Diagnostic inoculation may be made on the basis of the susceptibility of the ox and white mouse to tuberculosis, and their insusceptibility to glanders; also the partial insusceptibility of the soliped to tuberculosis and his marked susceptibility to glanders.

The characteristic nodules and abscesses may be found in different internal organs such as the stomach, intestine, liver, spleen, kidneys, testicles and brain, especially in acute cases, whereas the lesions of the skin and nose are more common in chronic cases.

PROPHYLAXIS AND TREATMENT OF GLANDERS.

Extinction. Exclusion, in Australia, New Zealand, English army. Occision of infected, and disinfection of stables, harness, vehicles, utensils, manure, and other infected things, mallein diagnosis; attendants should avoid handling suspicious horses, except with sound hands, and disinfect latter. Sheep living in horse stables, tested before slaughter, or inspected after. Malleinization on Plains and in high, dry air, in secluded herds; less hopeful elsewhere. Mallein test for all solipeds from glanders districts, and imported horses. Treatment illegal in many states, so that justice would require extinction with indemnities. Successful on high tablelands and mountains. Demands careful segregation and disinfection. Acute cases always hopeless; chronic skin cases more promising. Antiseptic injection of unbroken nodules (carbolic acid, potassium permanganate, also

of open sores (mercuric chloride, iodized phenol, cupric sulphate, saturated, zinc chloride), excise nodule. For delicate mucous surfaces Lugol's solution of iodine or iodized phenol diluted. Tonics: arseniate of strychnia, copper biniodide, baryta nitrate, iron sulphate, sulphites, bisulphites, hyposulphites, phenic acid, open air life at pasture, or thorough ventilation, sunshine, moderate exercise, generous, partly grain diet. Mild cases in separate herd, in secluded, wide pasture with rich diet, including grain, and shelter in clean, comfortable shed at will, under tonics and antiseptics, tested by mallein at intervals, to be restored individually to work after two or more tests without reaction. Serum of immune animals subcutem. Treatment in man: surgically as in the horse, excision, curetting, antisepsis, abscesses opened and disinfected; iodoform insufflations, antiseptic gases, sprays and solutions. Internally; tonics, sulpho-carbolates, iron muriate, iodides, phenic acid, quinia, strychnia, arsenite, stimulants, serum treatment, pure air, out door life, rich, digestible food

Radical efforts at prevention must look to the extinction of the disease in the soliped, and its complete exclusion from Australia and New Zealand shows that such a result is not unattainable. In the English army where every glandered horse is at once killed and all pertaining to him disinfected, the disease is now virtually unknown except in the case of newly purchased horses or regiments operating in the field. In the French army which formerly lost 9 per cent. per annum from glanders, now under similar precautions loses but .5 per cent. A law providing for the prompt destruction of every glandered soliped and the safe disinfection of carcasses, stables, harness, vehicles, utensils, fodder, litter and manure that have been exposed to contamination, if enforced, would soon eradicate the disease. But this law should provide efficient machinery for its enforcement, and, under suitable safeguards, an appropriate indemnity for the owner. With the use of the mallein or serum in all infected studs, as a diagnostic agent, the campaign can be made sharp, short and effective, instead of waiting as in the past for the slow development of occult cases.

The greatest and most fundamental error in veterinary sanitary legislation is the lack of a guarded indemnity for the animals killed. I strongly urged this fact on the committee of the N. Y. legislature in 1898 but to no purpose. A bill was passed forbidding all indemnity for glandered horses, and an impetus thus given to the spread of the disease is daily bearing fruit in our great cities especially, disastrous to the health of the horses,

and a constant menace to that of humanity as well. The law makes it the duty of veterinarians to report all cases of glanders, but in great horse establishments such a report would stop the use of the whole stable, at a loss of thousands of dollars per diem, and put a sudden end to the employment of the reporting practitioner by the firm or corporation in question. The owner of one or two horses can afford to report, the loss of these and their work does not mean absolute ruin, but the owner of hundreds can not safely report. For owner and practitioner alike the alternatives are presented of obedience to the law with personal ruin on the one hand, and the surreptitious dealing with cases of glanders and the preservation of their livelihoods on the other. Whatever may be said as to the constitutionality of the law which destroys private property without compensation under the right of eminent domain, this is certain, that, as applied to animal plagues, this course is unjust, oppressive, and not only useless, but positively injurious, in that it drives the owners of animals to such courses as favor the spread of the plague in place of restricting it. To-day in New York City glanders is extensively prevalent, but large horse owners dare not adopt the legal measures for its extinction, with the certainty of great loss or ruin staring them in the face as the result. It should be further considered that any law is at once bad and vicious in its tendency which places before the citizen the alternatives of disobedience with profit, and obedience with loss or ruin. Such a law is the worst possible economy because in preserving the infection, it not only perpetuates the disease and its attendant losses for all time, but perpetuates forever the official expenses of keeping it in check, when a prompt extinction of the infection would once for all time abolish all loss and all outlay for surveillance.

Until provision can be made for the enforcement of our laws against glanders, all who handle horses should be warned of the danger of working about strange solipeds, or those that show suspicious symptoms, while they have any sores on the hands, and in case they must run any risk of contact with infection, to promptly wash hands and face in a solution of hydrargyrum chloride (1 : 2000), or carbolic acid (2 : 100). Animals of susceptible races (sheep, etc.) which have lived with glandered horses should be made to pass the mallein test before they can be put upon the market for human food.

In the high, dry altitudes of the Plains and Rocky Mountains, where most cases of glanders are mild and the majority recover, resort may be had to malleinization provided the patients are kept safely secluded from all other horses. In some horses with a native tendency to immunity, the oft repeated inoculation with 0.5 cc. of mallein will render the animal refractory to the infection. Animals that have recovered from casual attacks show the same immunity. Among those who have experimented with mallein may be named: Straus, Schneidemuhl, Semmer, Bonome, and Vivaldi, Mowry and Michel, Schweinitz and Kilborne. Sacharoff apparently secured immunity in the horse by inoculation with virus modified by passing through the cat. Straus found that dogs which had received mallein intravenously could be made immune against intravenous inoculations, but, as Galtier had already pointed out, were still susceptible to cutaneous inoculations. Finger, experimenting on the rabbit, found that immunity only resulted after a long series of inoculations. The frequency of successful auto-inoculations in chronic cases of glanders in the horse would tend to discredit the alleged value of single injections of mallein, so that a long series is necessary if we would aim at good results.

Altogether attempts at immunizing the equine population generally, are not hopeful where it is dense, where they must be kept stabled, where the climate is moist and where glanders is deadly, or tends to persist in the chronic form for years in the same animal.

Considering the prevalence of the disease in a mild form on many of the western breeding ranches it is well to test all horses arriving from the west. The same applies to solipeds imported from abroad.

TREATMENT OF ANIMALS.

In the majority of the states the treatment of a glandered horse is prohibited by statute. Yet without providing definite machinery for the administration of the law, and without idemnities for horses disposed of, such laws are largely inoperative. On the other hand treatment is quite successful on the pastures of our dry table lands and mountains. It can, however, be sanctioned only when careful segregation and disinfection are provided for.

Acute cases of glanders are hopeless in any region, but chronic cases and especially such as have the lesions confined to the skin are much more hopeful.

The unbroken nodules may be injected with carbolic acid solution (1 : 200), or permanganate of potash (1 : 60). The open sores on the skin may be treated with the same solutions, with mercuric chloride (1 : 2000 to 1 : 5000), with iodized phenol, with cupric sulphate (saturated solution), or with chloride of zinc. A primary nodule may be excised and the sore treated with antiseptics. When the lesions are very extensive the less poisonous agents should be made use of, or tincture of iodine may be substituted. The nose lesions may be treated by the weaker solutions of iodine or of iodized phenol.

Benefit also comes from a course of tonics the most successful of which have been arseniate of strychnia, biniodide of copper, sulphate of copper, nitrate of baryta and sulphate of iron. The sulphites, bisulphites and hyposulphites and phenic acid are desirable adjuncts. An open air life at pasture is the ideal condition. Otherwise thorough ventilation, sunshine, moderate exercise and nourishing easily digestible food, including grain, are very important.

When conditions are favorable and an absolutely secluded pasture can be secured, with shelter from storms, and where civic or state authorities do not take effective measures to stamp out glanders, nor compensate owners for animals killed, mild, chronic and cutaneous cases and occult ones that have reacted to mallein without showing any other symptom, may be subjected to treatment. They should have an open air life, a generous diet, including grain, perfect cleanliness and pure air in the shelter-shed, an antiseptic and tonic medication (sulphites, tonics) may be given, and every three months a new mallein test may be applied. If the individual horse passes two successive tests without reaction, and shows no other indication of glanders, if his general health appears perfect and his condition good, he may be returned to work as a sound animal. With effective measures of extinction in force on the other hand, and indemnity for the slaughtered animal, any such measure would be entirely unwarrantable.

Treatment by injecting the serum of immune animal subcutem has given encouraging results. Helman, Semmer and Itzkovitch,

Pilavios, Bonome and Vivaldi, Johne, Schindelka, Prieur and others record recoveries in recent cases and there need be no doubt of its value in subjects that are naturally somewhat refractory to the germ. Babes introduced the blood serum of the ox, which is naturally immune, and Prieur speakes with confidence of the treatment of cutaneous glanders in man and certain cases of pulmonary glanders in the horse.

TREATMENT IN MAN.

The surgical treatment of glanders in man does not differ materially from that given for animals. It is more frequently possible to deal with the local lesion in its early stages and then a thorough cauterization of the infected sore, and a little later the complete excision of the primary nodule, or the limited regional infiltration, the curetting of the adjacent parts and the maintenance of thorough disinfection by irrigation, sponging and compress will often be followed by success. Where such radical measures are inadmissible the infected parts should be excised and curretted as far as safe and the adjacent parts subjected to carbolic injections (1 : 200). Abscesses should be evacuated, fistulæ slit open, and thorough disinfection applied For the affected air-passages, iodoform insufflations and antiseptic gases, sprays, and solutions should be freely used.

Internally, antiseptics such as sulphocarbolates and tincture of muriate of iron, iodides, and carbolic acid have been largely employed. Whitla recommends 5 grains of quinia in 15 grains of tincture of muriate of iron every four hours. Tonics (arsenate of strychnia), and stimulants, ammoniacal or alcoholic, may be called for. Special symptoms such as pain, rigors, nausea, vomiting, diarrhœa and profuse perspirations must be met by suitable remedies. Finally blood serum from healthy cattle or from immunized animals may be employed subcutem. Pure air and nourishing, easily digestible food are very essential. Every effort should be made to check the disease at its outset, as generalized acute glanders is speedily fatal, and recoveries in chronic cases usually leave a broken down constitution.

MYCOTIC LYMPHANGITIS : EPIZOÖTIC LYMPHANGITIS.

Geographical distribution and nature : Japan, China, India, Philippines, Africa, England, Ireland, etc., Northern and Southern Europe ; caseating nodules in skin, subcutem, in mucosæ and internal organs. In *horse*, skin nodules, and glandular abscesses, with thick ovoid refrangent bacterium ; saccharomyces (Hitt) ; swellings first local, then multiple, and general, extending along lymph vessels to glands (Hitt) ; along air passages to lungs, from prepuce or scrotum to peritoneum (Hitt), or from conjunctiva to other parts in the orbit (Caparini and Ferner). In *cattle*, multiple, subcutaneous nodules—hazlenut to walnut, isolated, hard, painless. Suppurate slowly. Same saccharomyces (Tokishige, Nocard) May invade lungs with fatal result in a year. Mallein test gives no reaction. Treatment : actively antiseptic ; open, curette, pack with antiseptic gauze, sublimate bandages ; internally, tonics, antiseptics.

Synonyms : Lymphangitis Saccharomycotica (Farcinoides : Epizoötica : Ulcerosa) : African Farcy : Farcy in Cattle, Japanese Farcy, River Farcy, Algerian Farcy.

Horses (and cattle) in certain countries (Japan, China, Sweden, Finland, France, Algiers, Philippines, S. Africa, England, Ireland) are subject to a chronic lymphangitis resembling cutaneous glanders, but associated with the development of fungi in the nodular, caseating swellings in the skin, subcutem, in mucosæ and in internal organs.

Nocard describes the affection in the horse as characterized by red exuberant skin nodules (buttons, boils), which burst and discharge a thick, creamy, yellowish or bloody pus. The surrounding lymph plexus swells up into corded lines, with, at intervals nodules or abscesses. The infection extends to and implicates the lymph glands and general pyæmia may follow. The thick ovoid refrangent, pathogenic, bacterium stains in Gram's solution.

In Northern Europe and Asia, Southern Europe and especially France and Italy an analagous affection is described by Rivolta, Claudio-Fermi, Aruch, Tokishige and Nocard and Leclainche. Tokishige and Hitt attributes this to saccharomyces, farciminosus (cryptococcus) Fermi and Auroch say it does not ferment sugar, so the older name of *Cryptococcus of Rivolta* is more appropriate. The slightly ovoid cells are grouped in zoöglœa form, and are easily recognized by the refringent appearance of the

envelope of each (Nocard). It stains easily by the Gram-Nicolle method : Take on a cover-glass, a smear of pus from the wound, ulcer or pustule, dry, pass three times through the flame, stain five minutes in Nicolle's violet (saturated solution of gentian violet in 90% alcohol 10 cc ; 1% aqueous solution of carbolic acid 100cc), run it off and wave for a moment or two in water, cover for 3 or 4 minutes with Gram's solution (I. 1 part, KI. 2 parts, distilled water 200 parts), run this off, treat with alcohol for 1 or 2 seconds, then apply counterstain of vesuvine saturated solution for 3 minutes, run it off, dry, and examine, or mount in Canada balsam, (Pallin).

Cultures have been made in peptonized bouillon, on agar, nutrient gelatine, on potato, or on horse serum containing 2 per cent. of agar.

The infection attacks horses and according to Tokishige, cattle, entering by sores and abrasions of the skin of the limbs, or under the harness (back, breast, rump, head). Confined at first to the point of infection it becomes multiple, being conveyed from place to place, through the soil, floor, stall, harness, blankets, brushes, combs, straw, etc., and many extend on the mucosæ. The wounds fester, forming pus and crusts over a more or less considerable swelling. In a variable time, (one to twenty weeks) the surrounding lymphatics become swollen and tender (corded), and nodular swellings appear on their course which fluctuate, burst and discharge a whitish or yellowish pus, sometimes oily or slimy. The disease extends along the lymphatics, invading the lymph glands and even the internal organs.

Tokishige describes cases in which it extended along the respiratory mucosa, causing constriction of the nasal passages and larger bronchi, with dyspnœa, and finally, invaded the lung. In other cases it spread from the prepuce, or scrotum, upward along the tunica vaginalis, spermatic cord and testicle. Caparini and Ferner describe it in the orbit, affecting the lids, nictitans, conjunctiva and adjacent parts, with nodules and abscesses. Mazzanti describes the case of a filly which died with pea-like nodules on the colon, and ulceration, with dirty, black, purulent centres, and indurated borders. Tokishige describes the affected cattle as showing a vast number of subcutaneous, hard, painless nodules, varying in size from a hazel nut to a walnut and covered by light colored skin. The nodules were isolated and not con-

nected by swollen, beaded lymph vessels. They encreased and suppurated much more slowly than in the horse. In three cows microscopically examined he found apparently the same branching fungus (saccharomyces), as in the horse cases. The mortality is about 10 per cent. Nocard, Pallin and Mettam failed to infect cattle by inoculation from the horse.

In the *farcy of the ox* of Guadaloupe there form subcutaneous nodular abscesses, with cordiform swelling of the lymph plexus and trunks, often proving fatal in a year through extension to the lungs. The abscess breaks, discharging whitish, creamy or caseous, or grumous contents, containing a bacillus (Nocard) streptothrix (Metchnikoff). The abscesses usually appear under the sternum or belly and later extend to the thighs and legs. In all such cases the mallein test is inoperative.

Diagnosis. It is most likely to be confounded with farcy or, if the upper air passages are affected, with glanders. It differs in the absence of general ill-health, emaciation or unthrift, unless, in very advanced and generalized cases; in the habitual absence of fever; in the hearty appetite; in the redness and exuberance of the granulations, and their tendency to heal under antiseptics; in the opaque, thick creamy character of the pus; in the absence of reaction to mallein; in the presence of the cryptococcus in abundance in the pus; in the immunity under inoculation of sheep, goats, dogs and above all Guinea pigs. Tokishige claims the infection of cattle and the presence of the cryptococcus in the lesions, but all inoculations of cattle elsewhere have failed.

Relapses and recurrences of the disease in the same animal have been repeatedly noticed.

The *treatment* of such cases should be actively antiseptic to destroy the germ while still local. After opening and evacuating the abscesses, excise or curette the diseased tissue, or destroy with the actual cautery, pack the cavities with pledgets soaked in tincture of iodine, iodized phenol, creolin, lysol, mercuric chloride, or biniodide, zinc chloride, or copper sulphate, or dusted with aristol, iodoform, or iodized starch. The surrounding swelling, if any, may be painted with tincture of iodine, or covered with cloths wet with a sublimate lotion or other antiseptic. Pustules and corded lymphatics may be laid open, curetted and

dressed antiseptically. Protect against flies. Internally, tonics and antiseptics may be given : arsenite of strychnia, quinia, iron or copper sulphate, sulphites or hyposulphites of soda or potash.

Prevention. As incubation in the horse may extend from 1 to 3 months (Pallin), importation of solipeds from an infected country can only be safely made subject to a three months quarantine, under the most critical veterinary supervision, every sore being examined microscopically by one who is expert in recognizing the cryptococcus. Then before release they should be thoroughly sponged with a mercuric chloride solution. Close supervision for six months more is important. Harness, covers, poles, shafts, and stable implements and appliances should be similarly treated or destroyed. In case of the appearance of the disease in an imported animal, it should be at once destroyed, cremated, rendered, or deeply buried, and the grave fenced in. This is imperative in warm weather when insects abound. Any return of army horses or mules from the Philippines without such exhaustive precautions would place the United States in the most imminent peril of a visitation such as overtook S. Africa, Gt. Britain and Ireland from a similar neglect. In quarantined animals any suspicious subcutaneous nodule should be extirpated ; it may be a focus of chronic infection.

To suppress the disease in an infected country the stud or herd must be put under the control of an expert on the microbe and disease, who must make an exhaustive microscopic examination of every sore, ulcer, pustule, nodule, corded lymphatic, or muco-purulent discharge from mouth, nose, eyes, ears, sheath, vulva or elsewhere.

Daily examination of each animal should be made, special attention being given to parts which are specially exposed to mechanical injuries (galls, contusions, bites, kicks, interference, cuts, treads, insect, leech or snake bites, stings, surgical wounds, etc.), but also to the whole surface, the mucosæ, the superficial lymph glands (submaxillary especially) and vessels, the coronet, etc.

No common use of halters, covers, brushes, combs, rubbers, scrapers or sponges should be allowed.

The suspected herd should be picketed, each animal apart, or stabled so that they cannot bite nor kick each other, and a

screened stable excluding insects is important. Manure should be often removed and burned, or put in deep, dark covered pits to cut off the breeding of flies.

Wounds must be dressed under careful antisepsis, discarding sponges and cloths, and washing under a jet of sterile (boiled) water, or use a fresh swab of sterilized cotton for each wound, burning the swab after use. As an antiseptic dressing mercuric chloride (1:250) is an excellent application and is to be thoroughly applied. Where flies cannot be excluded from the stalls the wound may be covered by antiseptic (sublimate, iodoform, boric acid) gauze.

The stable should be cleaned and whitewashed, and the floor covered with freshly burned quicklime, with or without chloride of lime. Pallin flames the surface of the woodwork and then applies sublimate solution. Mud floors have litter burned over them and are then removed to a depth of six inches, and filled in with fresh earth.

The horses of an infected stable or stud are suspected for six months after the last case and cannot be taken to mingle with other horses. By keeping them divided in small isolated groups (50–100), animals may be taken from a group after six months complete immunity, but should be kept under careful scrutiny for six months longer.

Infected pastures may be burned over in the dry season, pastured for one year with sheep or hogs, or subjected to a rotation of cultivated crops.

RABIES AND HYDROPHOBIA.

Synonyms. Definition: Acute, infectious, cerebral disease, of domestic and wild carnivora, shown by intellectual, emotional, and aggressive nervous disorder, and extreme nervous excitability. Animals susceptible: canine and feline animals, biting animals and those bitten, of all warm blooded kinds; receptivity greatest in carnivora. Geographical distribution: where population is most dense, trade and movement most active—north temperate zone. Australia, Tasmania, New Zealand, Azores, St. Helena immune—protected by quarantine. Islands in Elbe, places with enforced muzzling. Causes: contagion: inoculation; rutting; skunk bites; absence of flowing robes (men, children). Virulent matters: saliva, bronchial mucus, flesh, blood (not in sheep?), milk, (in advanced stages), nerve tissue. Morbific agent particulate; filtered liquid noninfecting: blastomyces, bacillus, found but not uniformly: claims of brain symptoms from each. Viability; saliva virulent for 11 days; brain 3 weeks at 32° to 63° F., for months aseptic and in contact with carbon dioxide; water 20 to 38 days; body in grave 44 days; virulence lost by drying in thin layer yet kennels remain long infecting; glycerine preserves; robbed of virulence by iodine, citric acid, bromine, chlorine, sulphurous and mineral acid and cupric sulphate. Bites of men effective to 50 per cent., protection by clothes, wool, flow of blood, cleaning of teeth by succession of bites. Infection by licking, sneezing, coughing, inhaling, feeding. Incubation 15 to 60 days in dog, 20 to 45 days in horse, 14 to 60 days in sheep, swine and cattle, 14 to 64 days in man. Prolonged cases doubtful. Symptoms in dogs: change in disposition, more amiable or morose, dull, apathetic, excitable, unwonted silence or howling, restless, depraved appetite, swallowing small objects, searching, tearing sticks or clothes, licking cold stone or genital organs, hiding in dark corner, dull expression, irritation where bitten, hoarse voice, rabid howl, eyes follow unreal objects, snaps at them, listens for unreal sounds, irritability, fury when threatened with stick or shown another dog, bites without growl, bears master's blow in silence, eyes red, fixed, pupils dilated, squinting, lonely wandering, biting, swimming rivers; restless excitement alternates with torpor and seclusion, slouching gait when worn out; in *paralytic form*; weakness, paresis, palsy of lower jaw, hind limbs, body, dull, prostrate, apathetic; in lethargic form; no fury nor paralysis, but profound prostration and apathy, no coaxing, threat, punishment not even another dog will rouse. Paralytic and apathetic forms wind up the furious; cases with intermittent attacks. Diagnosis: by marked change of disposition or habit, voice, appetite, any hyperæsthesia, strabismus, watching or snapping at unreal objects, taciturnity, fury in presence of other dog or stranger, snapping, biting without giving voice, fury when threatened with stick, open mouth without disposition to paw it, lack of muscular coördination, paresis, paralysis. Distinction from lactation anæmia, bone in palate or throat,

dislocation of maxilla, stomato pharyngitis, cutaneous hyperæsthesia, pharyngeal anthrax, epilepsy, pentastoma, cysticercus, filaria immitis, nematodes, and tæniæ, auricular acariasis. Symptoms in *cats*: changed voice, depraved appetite, hiding, restlessness, irritability, bites, scratches, man or dog. Symptoms in solipeds: trembling, hyperæsthesia, easily startled, ears and eyes alert, dilated flashing pupils, roused by dog or stranger, attacks with teeth and heels, mischievous, pawing, kicking, rolling, rising, straining to urinate or defecate, neighing, sniffling, snorting, everting upper lip, grinding teeth, biting, stiffness or impaired control of limbs, generative excitement, spasms, paralysis, hyperthermia, perspiration. Symptoms in cattle: irritability, restlessness, alert head, ears, eyes, squinting, dilated pupils, loud bellowing, lashing tail, or docility, yet pursues a dog, using horns, heels, (exceptionally teeth), stamps, paws, has genital excitement, anorexia, spasms, weakness, paraplegia. Symptoms in sheep and goat: hyperæsthesia, irritability, genital excitement, fury in presence of dog, stamps, butts, paws, snorts, bleats, grinds teeth, becomes paretic, and paraplegic. Symptoms in camels and deer: camels show furious and paralytic forms, biting and snorting; deer have hyperæsthesia merging into paralysis. Symptoms in swine: extreme irritability, restlessness, start at slightest cause or none, tremors, squealing, jerking, grinding teeth, clenching jaws, bite especially a dog, or not, weakness, lethargy, paralysis. Symptoms in rabbits and Guinea pigs; may be furious, more often paralytic. Symptoms in birds: restlessness, ruffling of feathers, pecking or striking man or dog, or at phantom, palsy. Symptoms in wild carnivora: lose fear of man, approach and attack him, are furious and later paralytic. Skunks bite slyly. Symptoms in man: irritable cicatrix, anxiety, sighing, tremor, insomnia, bad dreams, fever, thirst, spasms of throat when seeing, hearing or taking liquids, dyspr œa, retching, vomiting, spasms, roused by noise, squinting, dilated pupils, mental delusions, reticence, taciturnity, suspicion, mania, exhaustion, paresis, paralysis. Lyssophobia, its detection and cure.

Synonyms. Canine madness; Rabidus Canis; Lyssa; Lytta; Cynolyssa.

Definition. Rabies is an acute, infectious disease, affecting the cerebral and medullary nervous centres and characterized by intellectual, emotional, aggressive and other nervous disorders and by extreme reflex excitability.

Animals susceptible. While the disease is seen most frequently in the canine races (dogs, wolves, foxes and jackals), and in the cat family (cat, lion), it is liable to spread widely among animals that use the teeth as weapons of offense, and such as they can readily attack. When inoculated, all warm blooded animals contract the disease. Thus the carnivora and omnivora (dog, wolf,

fox, hyena, jackal, cat, lion, badger, and skunk and, to a less extent, the pig and horse) may become active propagators of the infection, which may spread widely among their herbivorous victims (cattle, sheep, goats, deer, rats, mice, chickens and pigeons) through their bites. Man suffers mainly through the attacks of dogs, cats and, in certain localities, wolves or skunks, but he is also liable to become infected from handling rabid domestic herbivora. Different genera differ in susceptibility, the receptivity being apparently greatest in the carnivora.

Geographical Distribution. Rabies is confined to no country nor climate but it attains its greatest prevalence in the north temperate zone, where there is the densest population and where activity of travel favors the propagation of infection. The facility for inoculation is the one determining cause, thus the islands of Australia, Tasmania, New Zealand, the Azores and St. Helena have never suffered and the first three exclude the disease by rigid inspection and quarantine. During the Hamburg epizoötic of 1852–53, the islands in the Elbe escaped though both banks were ravaged. Again, where the muzzling of all dogs has been rigidly enforced, as in many German cities and districts the disease has been practically extirpated.

Etiology. Long before the days of pathological bacteriology rabies was recognized by veterinarians as a disease due to infection alone. Its absence from the various countries above named, and from South and West Africa, its rapid propagation in other countries (La Plata, Malta, Hong Kong) into which it had been introduced for the first time, and its restriction and disappearance wherever muzzling had been strictly carried out, had practically settled this question. The development of the disease in animals which had been experimentally inoculated was no less significant of this truth.

Other alleged causes are based on secondary factors that favor inoculation. The alleged evil influence of the hot season (dog days) is accounted for by the period of *rutting* of the dog which occurs in spring, and brings together troops of jealous dogs following individual bitches and fighting for their favors. This gives a great impetus to the propagation of the disease which accordingly becomes more prevalent during succeeding months. The statistics of Bouley show, however, that the season of its

greatest prevalence in France is March, April and May, that it subsides slowly up to midwinter and increases again in spring. Boudin's statistics show the greatest number of cases in man toward midsummer which is explained by the long period of incubation after bites sustained in the spring, but also because clothing is lighter in summer and infection more likely to reach the skin, and because the heat of the weather tends to cause hyperthermia and encrease susceptibility.

The large preponderance of male dogs among the victims of rabies (7 : 1) has led to the theory that sex predisposes, but the explanation is rather that the male bite each other in their jealousy while they respect the female, the object of their sexual passion. In inoculated cases, males, females, and castrated contract the affection indifferently and with equal readiness.

The skunk is found to be infected in certain localities, and it has been claimed that the affection is native to this animal, and that the form differs from that which prevails in the dog, but the restriction of the disease to sharply circumscribed areas in Michigan and Kansas, while skunks elsewhere show no such malignant quality, demonstrates that this is but an accidental infection of the family of the Mephitis in a given locality.

Men and children suffer in far greater numbers than women, the difference being mainly due to the protection furnished the female by the flowing skirt, though also in part because of the more frequent contact of men with dogs.

Virulent Matters. In 1813 Gruner and Count Salm demonstrated the virulent properties of the saliva of the rabid dog. This has been many times repeated, though as shown by Pasteur, Raynaud, and others, inoculations with saliva often kill in one or two days by reason of the presence of other infected germs, or suppuration ensues and destroys the rabific virus so that oftentimes not more than one in four develops rabies. Galtier succeeded in inoculating 3 rabbits, 1 dog and 1 sheep with the juice expressed from the salivary glands, and Nocard, Leclainche and Peuch have found this to be very virulent. Paul Bert found the bronchial mucus virulent. Rabies has been frequently conveyed by consuming the bodies of rabid animals, yet Peuch and Galtier have failed to convey infection by the flesh. Eckel and Lafosse successfully inoculated the blood of goat to sheep, of man to dog

and of dog to dog. Yet many of the older observers agree with Peuch and Galtier in pronouncing the blood non-virulent. Pasteur, Roux, Bujwid, and Helman failed to obtain virulent cultures from blood. Peuch and Nocard have found the milk virulent, and Bardach found the same to be the case in a woman for two days before her death of rabies. In some partially refractory animals like the sheep, the blood seems to destroy the virus, as intravenous inoculations have been made with impunity. The probability is that the blood is habitually non-virulent in the early stages and in mild cases, but becomes virulent in violent and advanced cases. Pasteur first showed that the virus has a special election for the brain and nervous matter and that the central nervous organs are constantly infecting. Rossi inoculated with a piece of nerve trunk, and Doubouc and Babes advanced the hypothesis that the virus advances along the nerve to reach the brain.

It was formerly supposed that the saliva of omnivora and herbivora was nonvirulent but repeated experiment has proved it to be infecting. The blunder was due to the fact that these animals did not often transmit the disease by biting. Some writers even deny that rabies exists at all as an infectious disease. One alleges that the danger of rabies is "*less than the gallows.*" Experiment shows most conclusively that it is much more certain when the conditions are fulfilled. One may live a long lifetime without seeing a genuine case of rabies, but so he may without seeing leprosy or plague, and he is no more entitled to doubt their existence than he is the existence of Manila, or of the other side of the moon.

Morbific Agent. The actual factor which produces rabies is not certainly known so that we cannot speak of its bacteriology. That it is due to a specific particulate germ is now indisputable. Paul Bert and Nocard filtered the virulent fluid through plaster and found the clear fluid that has passed through nonvirulent. Rivolta, Babes, Bouchard and Peuch severally passed a solution of the rabific brain matter through a Chamberland filter and found the clear filtrate harmless. The virulent agent is therefore not a body in solution but a solid (organism) which is held back by the filter. Hallier, Klebs, Galtier, Gibier, Pasteur, Fol, Babes and Dowdeswell have respectively attempted the cultivation and in-

oculation of organisms found in rabific liquids but none has stood the test of further experiment. Memmo found a blastomyces in the brains of six rabid rabbits and one hydrophobic child. It stains with the aniline dyes but not by Gram's method : when thrown into the peritoneum of Guinea pigs it produced clonic spasms and death in 24 hours, and in the dog, after an incubation of 8 days caused emaciation, salivation, a disposition to bite, paralysis of the hind limbs and death in 48 hours. Bruschettini using agar or bouillon, containing lecithin or cerebrin and defibrinated dog's blood, inoculated with pieces of the brain of an inoculated rabbit, obtained in 24 to 36 hours a group of small transparent drop-like colonies at first microscopic, but becoming larger with each new culture on fresh media. These colonies contained a very small, short, thick bacillus, which stains readily in Ziehl's carbol-fuchsin, and then presents a central clear band giving the appearance of a diplococcus. In fluid media spherical forms are produced, but in fresh cultures the diplococcus aspect reappears. Injected subdurally in rabbits it gave rise to what appeared to be paralytic rabies and could be inoculated from animal to animal, with similar results. It failed to grow in the usual culture media from which the brain products are absent, thus fulfilling the conditions, of a microbe the point of election of which was the nerve cells. Marx who has sought for Bruschettini's organism in 60 cases of rabies has only once found anything resembling it and concludes that it was merely a contaminating organism, which caused a paralysis (Centr. f. Bacter. 1896.) Negri claims protozoa in the ganglion nerve cells as the active factor.

Viability of the poison. Galtier found that the virulent saliva remained potent for 11 days if preserved from drying. It persists for 3 weeks in the brain and medulla kept at 0° to 12° C., for a month in sealed tubes, or for several months if protected against septic microbes and in contact with carbon dioxide.

In water it is preserved for 20 to 38 days so that water soiled with saliva may easily become a means of infection. In graves it has been found virulent as long as 44 days after burial so that in medico-legal cases results may be obtained by exhuming a suspected animal (Galtier).

Haubner found that drying the saliva in thin layers rendered it nonvirulent and Pasteur has shown that the rabbit's medulla loses its virulence in 14 or 15 days when dried in contact with air, and

apart from putrefaction. Such laboratory results must however be qualified by the facts recorded by Blaine, Youatt and others in which hound after hound died of rabies from living in a dry kennel in which a rabid animal had preceded them. Until we know the living germ itself of rabies, it is unwise to infer too positive results from experiments on that microbe, in what may be but one stage of its existence.

The virus is very resistant to cold, having survived a temperature of—60° C. (—76° F.) for several hours (Roux), —10° C. (—14° F.) for ten months (Jobert). Virulence is destroyed however by a temperature of 48° C. (118.4° F.) in five to twenty minutes (Galtier). Light destroys the virulence in 14 hours at 30° to 35° C. (Celli), but encreased pressure has little effect on it (16 atmospheres, Nocard and Roux).

Glycerine at room temperature preserves the medulla in its full potency for four weeks, but destruction ensues if it is heated (Protopopoff). An aqueous solution of iodine (6:100) destroys the virulence (Galtier), as does also citric acid, bromine, chlorine, sulphurous acid, the mineral acids and cupric sulphate.

Ratio of successful inoculation to bites of rabid animals. As rabies is usually transmitted by the bite, it is well to note that not all the bites of rabid animals are effective. Of 183 dogs confined with and bitten by a rabid dog 91 contracted rabies; of 73 cattle bitten 45 became rabid; out of 121 sheep bitten 51 succumbed and of 890 persons bitten, 428 took hydrophobia. Of 440 persons bitten by rabid wolves 291 contracted the disease. The escape of such a large proportion is variously accounted for. Wolves naturally attack the face, throat or hands where there is no protection by clothing, and inoculation is therefore much more certain. Dogs, especially in cold weather usually bite man through the clothes which wipe off the virus from the smooth conical teeth before they reach the skin. Long haired animals are often protected in the same way. In other cases the bite is sustained on a very vascular part and the free flow of blood washes out the poison. In still other cases the rabid animal making a number of snaps in rapid succession comes to the last with the teeth wiped clean and harmless. Again the prompt washing or cauterizing of the wound tends often to protect against infection.

Under favorable circumstances, however, every bite infects, and the writer has seen six animals, bitten in the same stable, all contract rabies, while a man bitten through the coat sleeve by the same dog, and cauterized an hour later entirely escaped. If the bites are multiple, deep and irregular, the danger is greater.

The licking of wounds is an occasional mode of infection, the rabid dog in the early stages of the disease sometimes showing an unusual disposition to fawn upon its owner.

Again particles of saliva may be projected by sneezing or otherwise and lodge on sores, or on the mucosa of the nose, eye or lip so as to cause infection. Galtier has conveyed the disease experimentally to rabbits in 11 cases out of 75 by making them breathe the atomized infecting liquid, or by dropping it into the nose. In the same way he infected Guinea pigs and sheep.

Galtier and Bujwid have conveyed the disease, exceptionally, to rabbits and rats by feeding infecting emulsions. Galtier has also produced rabies in $\frac{1}{10}$th to $\frac{1}{3}$d of the cases operated on by instilling the virus into the eye.

Incubation. The duration of incubation varies with the species, individual, the seat and character of the bite, the amount of virus instilled, the potency of the virus, the age, size and weight of the subject, the excitement of *rutting*, climatic or weather vicissitudes, fatigue, and nervous or febrile disorders.

In the *dog* incubation rages from 15 to 60 days, and perhaps 4 to 6 months. It is claimed to have lasted a year but this is somewhat doubtful. In *cats* it has varied from 15 to 60 days.

In *solipeds* it ranges usually from 20 to 45 days. The extremes stated are 15 days and 20 months.

In *cattle* it ranges from 14 to 50 days (exceptionally 70 days), in *sheep* and *swine* from 14 to 60 days. It has been claimed to have lasted 4 months in swine and 5 months in sheep.

In *rabbits* subdural inoculation has an incubation of 15 days, shortening in 25 removes to 8 days, and finally to 6.

In *man* incubation is alleged to be even more varied. The rule is from 14 to 64 days, but it is claimed to have been as short as three days and as long as 1 to 12 years (Chabert). In the human being, however, there is always the danger of the disease caused by simple dread (lyssophobia) and until these protracted cases can be verified by successful inoculation on the lower

animals, they must be held as extremely doubtful. The Montpellier cadet has been often quoted who left, a few days after he had been bitten, spent ten years in Holland, then returning to the school and learning for the first time that his fellow cadet, who had been bitten by the same dog had gone mad, he too became rabid without loss of time. Such cases have often been cured by moral suasion and have been seized upon to corroborate the heresy that there is no such thing as genuine rabies in man.

Any incubation, in man or beast which has exceeded 40 days should be considered as doubtful, until certified by the successful inoculation of rabbits or other small animal. For casual inoculations the incubation rarely varies much from the time embraced between 16 and 30 days. It is abridged by a special receptivity; by an overdose of the poison; by the inoculation of a virus of unusual potency; by the youth of the animal inoculated; by great heat of the weather; by all forms of violent excitement; by *rutting*; and by the inoculation of the virus on the head and above all on the cerebral meninges. By this last method incubation may be reduced to 6 days.

Symptoms in Dogs. In dogs as in other animals rabies is manifested in two great types: the *furious* and the *dumb* or *paralytic*, which, however, usually succeed each other in fully developed cases. Yet the *furious* phenomena may be entirely omitted, and again the victim may die in the early *furious* stage so that the paralytic does not appear. The prominence of one form over the other is to some extent determined by the germ derived from a previous case of the same kind or by the family, temperament and habits, bull dogs and hounds being specially subject to the furious type, and house and pet dogs having rather the paralytic form.

The *premonitory symptoms* are in the main the same for both types, and as these may enable us to recognize the disease before the period of extreme danger, it is especially important that they should be well understood.

Some marked change in the disposition or habits of the animal is the first obvious variation from health, and in a district or country where rabies exists any such change should be the warrant for instant seclusion of the dog before there is any disposi-

tion to bite. The unwonted habit may be of almost any kind. The lively, amiable dog may become suddenly dull, apathetic or taciturn ; the quiet, unexcitable dog may become unusually affectionate, fawning and demonstrative, licking the owner's hands and face and perhaps infecting him before any suspicion is aroused. Sudden capricious changes from fawning to apathy or sullenness, or the opposite, should be dreaded. The noisy dog may become suddenly silent ; while the silent dog may take to howling without apparent cause. A great restlessness, watchfulness or nervousness, a tendency to start at the slightest sound, and a disposition to move at frequent intervals in search of an easier position or place to lie in, are most dangerous symptoms. A morbid appetite, with a disposition to pick up and swallow all sorts of non-alimentary objects (straw, thread, cord, paper, pins, nails, coal, marbles, pebbles, cloth, earth, dung or urine), in a mature dog is most suggestive. Searching around, scraping, tearing sticks, clothes and other objects to pieces, licking of smooth cold stone or metal, of his penis, or of the generative organs of a bitch, are often early phenomena. The dog may hide in a dark corner, going to sleep and grumbling or growling when disturbed. He may make night hideous with his howls, *baying at the moon*. He may stand with a dull, melancholy, hopeless expression of countenance, as if beseeching his master for relief from his nameless suffering. But as yet there is no disposition to bite. The dog still responds to the call of the master, but with dulness and apathy, in marked contrast with his usual prompt, alert and loving response. There may be congestion, itching and irritation in the seat of the bite, and it may be licked, scratched or gnawed until raw, tender or bleeding. An early change in the voice may be noticeable. There is at first a certain hoarseness, which gradually develops into the pathognomonic rabid howl which is quite recognizable at a distance. The dog turns his nose upward, and with open mouth, emits a howl which, at first hoarse and low, rises into a shriller and higher note before completion, and which may be repeated several times without closing the mouth. It is not an ordinary howl but rather a cry of distress, and, to the educated ear a grave note of warning. Sometimes the dog is dumb from the start.

Close observation will often detect evidence of mental delusions and hallucinations even at this early stage. The apparently sleeping dog suddenly starts up with an air of suspicion and excitement. His eyes may turn after phantom flies or other objects at which he will presently snap. He moves hither and thither with a curious, inquisitive air, searching in dark corners, or under curtains or articles of furniture for some imaginary object. He may stand in attentive attitude listening to fancied sounds, and then bound in that direction in spite of an obstructing wall or utensil. If recalled to reality by his master's voice his healthy attitude and affection may be completely though temporarily restored. He may compose himself to rest or sleep, and soon again start a victim to further delusions.

The *furious stage* is ushered in by a more pronounced manifestation of the above symptoms. The rabid howl is more frequent and characteristic. The insomnia and the restless movements, change of place, searching, scratching or tearing are likely to be more prominent. The delusions and the watching or snapping at phantoms are more marked. The causeless sniffing and scraping may imply disorder of the sense of smell. The exalted hyperæsthesia and reflex excitability is usually a most marked feature, frequently modified by a sinister, inimical or malevolent disposition. Some subjects show evidence of intense itching, most marked perhaps in the seat of the wound; others are abnormally sensitive to currents of cold air; they start at the slightest noise, a flash of light, a touch, or the approach of a stranger. A paroxysm of fury may be caused at once by shaking a stick at the patient, but, above all, by the test of presenting a dog before his eyes. He at once rouses himself and, with flashing eyes, dashes himself on his supposed enemy and bites viciously, but without bark or growl. The sudden and mute attack is pathognomonic. But there may be symptoms suggestive of some hypoæsthesia. A blow from the dog's master is usually borne in silence. The animal shrinks from the blow but does not yelp nor howl. During a paroxysm he will lacerate his gums or loosen his teeth by biting on a stick or iron bar; he will even seize a red hot bar without shrinking. He will often gnaw his limbs or body so as to expose and even lacerate the muscles, driven by the pruritus, or the insatiable disposition to bite. Yet

he has still an instinct of self preservation, retiring from a light, or blazing brand, and seeking to evade the tongs used to seize him.

The eyes assume a remarkable expression. They are often fixed, and at times the pupils are widely dilated allowing the reflection of light from the brilliant tapetum lucidum, resembling flashes of fire, and adding greatly to his ferocious appearance, and the sense of terror that he inspires. The conjunctiva is congested, of a dark red and the general expression of the eye is very striking. The soft, trusting, affectionate eyes may still for a moment meet those of the loved master, but there is in them a dull, hopeless, anxious and suffering expression, which appeals for pity and should be viewed with dread. To others there is likely to be less of the affectionate appeal and more of the sinister, suspicious, resentful and malicious expression. To the stranger, therefore, the eye may be a better guide than even to the owner. Squinting of the eyes and closure of the lids for a few seconds are not uncommon. As the disease advances the dilatation of the pupils is more marked, indicating the paralysis of the optic nerve.

In this stage the disposition to wander is characteristic. The rabid dog leaves his home and wanders off a long distance, say ten or twenty miles, snapping in his travels at man or beast that may irritate him, returns dirty and exhausted and seeks anew his dark place of seclusion, or he may snap at and bite even his master. To those who come in contact with him at this time the dog is especially dangerous from his extreme irritability though weak and exhausted.

If the wandering rabid dog meets a strange dog he attacks him and bites without growling or barking. If the bitten dog does not yelp nor retaliate, but simply flees, the rabid animal moves on, but if he bites back, or howls, he worries him, rolling him over and biting again but always in silence, in marked contrast with an enraged but healthy dog. If he comes up with a herd of cattle or pigs or a flock of sheep or fowls, the result is similar. If they remain perfectly still they may possibly entirely escape, but if they scamper off with noise as usually happens, he rushes at them and bites one after another, so that in a confined yard or park all may suffer. A man meeting the dog increases his danger by making an outcry, whereas if he remains perfectly quiet he

may possibly escape. Bouley says that a canine attendant is, in a sense, a measure of protection to a man, as the rabid dog attacks first the animal of his own species, giving the man a chance to escape.

In his wanderings the rabid dog will swim rivers, having *no dread of water*, just as at home he will plunge his nose in water though unable to swallow. When abroad in this way he exhausts himself by his paroxysms and may perish in one of them, or he may meet his death from man or animal. He may in his exhausted state seek a dark secluded place where he may remain for a time and renew his travel later, or he may pass into the paralytic condition and gradually sink.

When shut up, and his vagrant disposition curbed, the paroxysms are liable to appear intermittently, a period of torpor and quiet alternating with one of restless movement, searching, scraping, howling, biting of any animals within reach and later of men, beginning with strangers. A paroxysm can usually be roused by shaking a stick at him and always by presenting another dog.

There is sometimes a difficulty in deglutition, the dog acting as if he had a bone in his throat which he was trying to dislodge, and fatal bites have been sustained during well meant attempts to remove the hypothetical bone. This bears a resemblance to the pharyngeal spasms which are such a marked feature in the hydrophobic man. But it is not roused, as in man, by the sight or sound of water. On the contrary, water is sought and often swallowed at first and even, exceptionally, throughout the disease. He may even take his usual food for a time. The bowels are usually torpid, and any fæces passed are black and fœtid. Diarrhœa may set in later.

In the early stages the rabid dog walks or trots like any other dog. It is only when exhausted by wandering, or violent paroxysms, or both, that he droops his head and ears, hangs the tail between the legs, and slouches along with arched back, and unsteady, swaying limbs. The appearance of these last symptoms implies advancing debility and paresis, and the near approach of paraplegia. The symptoms may, however, be temporarily relieved by a period of seclusion and quiet.

In *dumb* or *paralytic rabies* the striking peculiarity is the omission of the preliminary furious stage, and the disease merges at

once into paralysis after the premonitory symptoms. In these cases the early nervous symptoms tend to prostration, weakness and dulness or even stupor, there is no disposition to escape, but rather to seek seclusion and quiet, there is rarely howling and then only at first, and soon there is paralysis of the masseters and dropping of the lower jaw, and there is neither ability nor desire to bite. From this the paralysis extends to the hind limbs and then to the fore limbs and trunk. In other cases one limb is the first to suffer, followed by the face, limbs and body. The most prominent feature is the widely opened mouth, the flaccid, hanging tongue and drivelling saliva. The buccal mucosa, at first red and moist, becomes bluish, dry and powdery. The eyes are dull, mournful, suffering or altogether without expression and the pupils are usually widely dilated. The hind limbs are usually utterly helpless and often the fore ones as well, the prostration is extreme and the patient lies quiet and helpless until released by death in two or three days.

A third form, known as the *lethargic*, is a modification of the paralytic. There may be neither delirium nor marked paralysis, there is no drooping maxilla, pendant tongue nor stringy, hanging saliva, but only a profound, nervous prostration and complete apathy. The patient curls himself up in some dark, quiet corner and cannot be roused by coaxing or punishment, by hunger or thirst. In this, as in the dumb rabies, the common test of presenting another dog fails to rouse excitement or paroxysm. If left undisturbed, these patients may live to the tenth or fifteenth day.

Beside the typical forms there are all intermediate grades, inclining more or less toward the furious, or the paralytic or lethargic. Galtier mentions cases that showed aphasia, scarcely any disposition to bite, swaying movements of the body and limbs, muscular incoördination, tucked up, tender abdomen, rolling the body like a barrel, and marked dyspnœa. Others show at first a slight disposition to bite which is quickly checked by a very early paralysis of the masseters. In still others the attempts to bite are yet seen after dropping of the jaw, but though moved, it cannot be completely closed.

In exceptional cases rabid dogs have shown *intermissions* of apparent soundness extending over eight days (Youatt), two

months (Pasteur), or even six months (Perrin). Rare as these are they must apparently be accepted, and must qualify to some extent the trust reposed in immunization.

Diagnosis. The early diagnosis of rabies in the dog is of supreme importance as enabling the owner to destroy or seclude the dangerous animal before he has developed the disposition to bite and to propagate the disease. If the dog himself is known to have been bitten, every premonitory symptom should be carefully looked for and critically studied. Any change in the general habit, unusual liveliness or dulness, restlessness, or somnolence, unwonted affection or taciturnity, special watchfulness with vision, or hearing, change in the character or tones of the voice or the disposition to use it, propensity to swallow foreign nonalimentary bodies, retching, vomiting or costiveness, propensity to hide away in a dark corner or to leave home and disappear for a day or more, disposition to gnaw and tear up wood or clothing, or hyperæsthesia affecting any part of the senses should be carefully noted. Note especially any exaggeration of the generative instinct, any strabismus, or redness of the eyes, any turning of the eyes or head after imaginary objects or snapping at them, any disposition to anger or attack when another dog or cat is presented and any disposition to growl at or to bite a stranger. This tendency to anger and resentment though not always present is usually a symptom of the very greatest value. If a dog is known to have bitten or attempted to bite any person or animal he should be tied or shut up in a safe place for four days at least, under veterinary supervision, and set free only on condition that no symptoms have developed at the end of this period. A hasty decision that a suspected dog is not rabid must lay a veterinarian open to the most serious charges, in case rabies occurs in man or beast from the bites.

Diagnosis is less difficult after the paroxysmal stage has set in. Then there is the extreme irritability and hyperæsthesia, the appearance of reflex spasms of the pharyngeal muscles and chest under the influence of any peripheral excitement, the derangement of the senses, the dilated pulpil, the flashing eye, the squinting, the taciturnity and the propensity to bite. Later still the lack of coördination of movement or the paralysis, especially of the hind limbs and of the jaws.

The symptoms may be simulated by those of some other diseases. Thus the bitch which has been exhausted by lactation may show delirium with taciturnity and a disposition to snap. The dog, which is habitually struck or threatened by passers by, may acquire a sinister look and a disposition to bite on every occasion. The presence of a bone or other foreign body fixed between the upper molars, and various injuries of the jaws, teeth or throat may cause inability to swallow, change of the voice and a morose disposition and expression. In such cases there may be vomiting, rubbing the jaws with the paws as if to disengage something, and salivation, but there is no delirium, fury, muscular weakness nor paralysis. In paralytic rabies on the other hand, along with the open mouth and drivelling saliva, there is no disposition to paw the mouth nor face, the buccal mucosa is not smply red but of a deep violet, and there is attendant weakness or paralysis of the hind parts.

Galtier has seen inability to swallow and dropping of the lower jaw from violent stomato-pharyngitis, and from dislocation of the maxilla.

Great tenderness of the skin from inflammation due to blistering or caustic agents or from rheumatism may cause such alert apprehension and disposition to bite in self defense that it may simulate hyperæsthesia of rabies.

From pharyngeal anthrax and violent angina, rabies is distinguished by the extreme exaltation of the special senses, the marked hyperæsthesia and reflex excitability, and, as in the other diseases mentioned, by the perfectly lucid intermissions. Epilepsy is not to be roused by sudden noise, movement nor attempts to swallow, it is not associated with hyperæsthesia and in the *haut mal* the spasms affect the muscular system more generally. A disposition to bite, and spasms and other nervous symptoms, resembling to some extent those of rabies, have been seen in cases of pentastoma in the nasal sinuses, cysticercus in the brain, filaria immitis in the blood, nematodes, and tænia in the bowels, and auricular acariasis, but there is no such hallucination nor visual delusion, no alteration of the voice, no cutaneous anæsthesia, no exalted reflex excitability. Cadeac finds rabiform symptoms with disorders of the special senses in animals dosed with various essential oils, but the odor of these essences about the mouth and in the breath would serve to distinguish.

Symptoms in Cats. The disease makes a rapid progress, and often ends in death in three or four days. There is a marked change of voice, the calls being hoarse and bass, having been compared to that of a cat in heat. As in the dog, there is a disposition to pick up and swallow objects that are in no sense food, perhaps even its own urine or fæces. There is a great tendency to hide away under furniture or in secluded corners so that the malady may be far advanced before anything is suspected. If seen the patient shows restlessness and frequent movement, with a marked excitability under noise or other disturbing influence. Any disturbance may arouse a paroxysm, accompanied by occasional wide dilatation of the pupils, with flashes from the bright carpet in the vitreous chamber, and the patient may spring at human beings and bite or scratch the hands, face or other uncovered portion of the body. It shows the same disposition to bite small animals, and especially dogs. A glairy often frothy saliva is found in the mouth and often around the lips. As the disease advances and paresis sets in, the cat usually crawls into some dark secluded corner and there dies.

Symptoms in Solipeds. These may vary much in different cases but the leading characteristics, as seen in the dog, are prominent also in the soliped. There is marked restlessness, trembling, and extraordinary hyperæsthesia, as seen in starting at sounds, sudden flashes of light or other causes of disturbance. The ears are held erect and watchful for sounds ; the eyes are red and mostly vigilant with at times pupillary dilatation and flashing. The sight of a dog rouses him instantly to attack it with teeth and heels. A stranger is liable to be similarly treated though the horse is still docile and kind to his keeper. There may be itching of the skin and above all of the seat of the bite. During a paroxysm the movements are usually violent, dangerous and even mischievous. There is constant restless movement, pawing, kicking the ground, the stall or attendants, lying down, rolling, rising, straining to urinate, or defecate, eversion of the rectum, shaking the head, muffled neighing, sniffing, snorting, everting of the upper lip, grinding of the teeth, or biting. In the absence of such paroxysms deglutition may be difficult, and the appetite depraved, the patient preferring earth or manure to the natural food. With spasms or paralysis of the pharynx, the food

may be returned through the nose. In some cases a stiffness or impaired control of the limbs has suggested inflammation of the feet. In the uncastrated male and female, generative excitement is the rule, the protrusion and erection of the penis, the swelling and rigidity of the clitoris, with frequent straining to pass water, the whinneying of the animal for its mate ; attempts at copulation and even ejection of semen may be observed. The seat of the bite may be red, angry and itchy, so that the horse rubs, nibbles or gnaws it, often breaking it open anew. There is usually ardent thirst, and no dread of water, even when swallowiug is difficult or impossible.

During a violent paroxysm the horse often bites the halter, blanket, manger, rack or stall, seizes the adjoining horse with his teeth, or gnaws, or tears strips of skin from his own shoulder, breast or limbs. In the same way he uses his feet with the most evident purpose of injuring man or beast that may approach him, or he breaks down his stall.

Even at an early stage there may be spasmodic movements of the eyes, face or body, and later there appear signs of paresis, often commencing in a hind limb and extending to paraplegia and general paralysis. Sometimes paralysis begins in the muscles adjoining the seat of the bite. The temperature, at first normal, may rise to 104° F. in the advanced stages, breathing and pulse are greatly accelerated, and the skin may be bathed in perspiration. Spasms are not entirely superseded by paralysis, and death often takes place during a convulsion, from the fourth to the sixth day. In apoplexy death may ensue in one day.

Cases in which paralysis is not preceded by a furious stage, are not uncommon in horses.

Symptoms in Cattle. Cattle are frequent victims of rabies, which assumes mostly the furious type, yet in certain outbreaks the paralytic or lethargic form predominates. There is first a strange irritability and restlessness, very unlike the habitual quiet disposition of the animal. The head is raised, the ears alert, the eyes prominent, red, fixed, with occasionally widely dilated pupils and brilliant flashes from the tapetum lucidum. Sometimes they roll or squint. They may appear wild and ferocious or dull and hopeless. Loud and terrified bellowing is not uncommon, switching of the tail, drivelling of saliva, and ex-

ceptionally there are attempts to bite. More commonly the natural weapons of offense are employed, the animal kicks, stamps, paws and, above all, tries to gore man or beast, but especially any dog which may appear. They even make such attacks on purely imaginary beings and without any real, tangible enemy present. The bull, cow or heifer present the usual signs of genital orgasm. Appetite is lost or depraved and rumination arrested. Pharyngeal spasm or paralysis is not uncommon, and signs of colic with frequent defecation may be seen. Violent paroxysms are easily roused by the sight of a small animal and especially of a dog. In these attacks the animal may break his horns or teeth or otherwise injure himself. Sometimes the infection wound becomes irritable, itchy and red or even abraded and raw by licking.

The animal becomes rapidly exhausted and even emaciated by the violence of the paroxysms, and paresis sets in with dragging movements of the hind limbs, which advance to paraplegia and general paralysis. Rolling of the eyes, squinting and pupillar dilatation may be present. Death usually takes place from the fourth to the sixth day.

The purely *paralytic rabies* is not uncommon in cattle. There may be persistent yawning or other sign of nervous exhaustion or depression, halting on one or more limbs, usually behind, which advances to complete paralysis. In other cases the symptoms are those of mental dulness, and profound lethargy with gradually advancing emaciation. In paretic and paralytic cases the characteristic paroxysms on presentation of a dog may be absent.

Labague claims to have seen intermittent cases with an interval of 27 and even 36 days.

Symptoms in Sheep and Goat. In these there is the same regular succession of symptoms through intense hyperæsthesia and excitability, fury and genital excitement to the terminal paralysis, or the palsy may set in early without premonitory violence. Among the marked features have been noticed, a change of expression, the pupils dilate, eyes flash, they lick or mount their fellows, they lick or rub the bitten part, snort, stamp or scrape with the fore feet, setting themselves in the attitude of attack, they may butt other sheep, fowls, and other animals,

above all dogs, or they may deliver the attack in the air only, at some phantom enemy. They may bleat hoarsely or brokenly, grind the teeth, or work the jaws with the formation of froth about the lips. Galtier says they may even attempt to bite. Sooner or later weakness of the limbs, muscular incoördination, swaying and staggering bespeak enervation, the sheep lies constantly, and if raised stands with unsteady semi-flexed limbs, has trembling or convulsions and dies paralytic in from two to five days. Death has been delayed till the twelfth day (goat) or thirteenth (sheep). In some cases paralysis has been present from the start, the subject lying prostrate from the first.

Rabies in the Camel and Deer. In Algiers camels are often times bitten by rabid dogs and contract the disease showing both furious and paralytic symptoms. They are especially dangerous because of their propensity to bite, and to scatter the virulent product by sneezing.

In England rabies has prevailed extensively in parks of deer, which shut inside high walls have been bitten remorselessly by rabid dogs that had gained admittance. The symptoms of hyperæsthesia and paralysis followed the same general course as in other animals.

Symptoms in Swine. Rabid pigs are usually very restless, excitable and sensitive to all causes of disturbance. They will hide under the litter in the darkest corner but soon start without apparent cause, turn around and lie down again, or they bound up with grunt or scream and rush off as if pursued, push or leap against the wall, stand with ears pricked as if listening, and start violently at any noise or a flash from a lantern, trembling, squealing, or having muscular jerking. The eyes are at times fixed, or may roll, squint or flash from pupillary dilatation. The voice is hoarse, deglutition difficult, there is frequent clenching of the jaws, or grinding of the teeth, and frothing around the mouth. The victim may tear with his teeth the boards of his pen, or gnaw pieces of wood, he may swallow wood, pebbles or earth, and plunge his face in water or other liquid without fear or apparent dread or dislike. The disposition to bite may be viciously shown, and he may strike dangerously with his tusks, while in other cases it may be entirely absent. Peuch quotes cases in which no paroxysm was aroused by the sight of a dog, and others

in which the pig would rise and grunt without showing any desire to bite.

Sows will sometimes bite or devour their offspring during a paroxysm, yet nurse and care for the survivors during the intervals.

Emaciation, weakness, exhaustion, lethargy and paralysis appear early, the victim remains down, or, if raised, moves weakly and unsteadily and no longer pays attention to noises nor blows. Death may ensue from the first to the sixth day.

Symptoms in Rabbits. Experimental cases in rabbits are now very common and usually assume the paralytic or lethargic type, there is weakness in the hind parts, advancing in a few hours to paraplegia, the fore limbs may be used for a time while the hind are flaccid and dragged, or the animal lies on the sternum and belly with the head sunken and resting on the feet, or he lies extended on his side, in a state of insensibility. In the early stages he may still masticate, with froth collecting on the lips, but there is difficulty in swallowing. If a foot is pinched it is drawn up often with a cry. The bowels are torpid though a few small, round, hard pellets are sometimes passed or a little urine. Incubation is shorter than in the dog, and the virus retains this habit when inoculated on other animals.

In the experimental cases in the Anti-rabic Institute in St. Petersburg, Helman found that the rabbits inoculated with virus from dogs having furious rabies, contracted furious rabies, while those inoculated from the less furious type of street rabies had the disease in the dumb form.

Symptoms in the Guineapig. In this animal as in the rabbit the disease has been mainly seen in the experimental form, and has assumed the paralytic type. It trembles, moves stiffly or weakly, lies most of its time and passes rapidly into paralysis. There is usually no tendency to bite, yet Peuch in a case of intra-ocular inoculation from a rabid cat observed the most violent excitement, loud hoarse screaming, bounding in different directions, biting of the wires of the cage, and other manifestations of violent rabies. In both forms there are liable to be convulsive movements of the jaws, accummulation of frothy saliva, and a free discharge of urine.

Symptoms in Birds. Chickens bitten by mad dogs have been

seen to prove restless, erecting the feathers and moving aggressively toward man or dog or phantoms of their imagination and attacking with bill or spurs or both. This, however, rapidly advances to paralysis and death. In cases due to intracranial inoculation on the other hand, somnolence, lethargy, or coma appeared early, and was quickly followed by paralysis. This affects especially the legs and neck. Recoveries are common and the animal is thereafter immune. Subsidence, with complete intermission of symptoms, is more common than in rabbits, the disease reappearing later, and perhaps finally ending in recovery.

Symptoms in Wild Carnivora. The rabid wolf, fox, jackal, hyena, coyote, ferret, polecat and skunk lose their fear of man, and approach and attack him in field, village or city. The disease has its furious and paralytic stages as in the dog, and the animals attack according to their nature, wolves being dangerous from flying at the face and throat, and skunks from stealing up and biting without warning.

Symptoms in Man. After the period of incubation prodromata may be present, or there may be suddenly and without any premonition, violent spasms of the pharynx and inability to swallow. The premonitory symptoms when present consist in irritability of the cicatrix which becomes red or blue, swollen, itchy or pricking, and an aura or shooting pain may extend from this toward the heart. There is anxiety, sighing, tremor, restlessness impelling to frequent change of place, insomnia, disagreeable or painful dreams, weariness, and gloomy forebodings. The face is pale and drawn, and the eyes wander or have a look of apprehension. There is some fever and often marked thirst, and the attempt to swallow rouses slight spasm or a sensation of tightness in the throat. The fauces, pharynx and eyes are congested, reddened and it may be swollen. Pulse and respiration are both quickened, the inspiration being often prolonged and sighing, and the expiration sobbing. At first the intellect is unimpaired; there is no illusion nor hallucination.

In cases in which premonitory symptoms are lacking, violent spasms of the throat and chest are commonly roused by an attempt to drink and this is so painful that the patient cannot again be induced to try. After this any suggestion of drinking, the offer of drink, the noise of trickling water, the sight of water, the sight of a vessel in which the water was contained, or even of a clear

shining surface of glass or metal is likely to bring on a paroxysm. This hydrophobia is peculiar to man, being rarely seen in rabid animals. During a paroxysm dyspnœa is extreme, respiration is gasping or sighing, and in the attempt to dislodge the tenacious mucus which is present in the throat, hoarse or shrill inarticulate sounds are emitted which have been supposed to resemble the bark of the dog. There is a sense of closure of the throat and of rising of the stomach, and retching or even vomiting may ensue. Hyperæsthesia, reflex irritability, and exaltation of the special senses, now become extreme, so that a paroxysm may be brought on by the slightest disturbance, a current of air, a bright light, the rustling of a dress, the noise of a footfall, the noise even of talking, or a slight touch. The "tendon reflex" and skin reflex are often much encreased. During a paroxysm there are muscular trembling and clonic spasms,—sometimes opisthotonos. The intervals of complete relaxation, however, serve to distinguish from tetanus. The face is red and drawn, the eyes congested and some times squinting, the pupils dilated and the expression one of suffering, apprehension and horror. Mental disorder appears sooner or later, the speech is disconnected, with indication of delusions from which the patient may at first be recalled by the attendant. There is, however, a constant disposition to be reticent, morose and above all, suspicious, as shown by the absence of a direct look, and the frequency of side glances as though in expectation of a hidden danger. This may even rise to mania, the patient charges those about him with having caused his sufferings, or with conspiracy to injure him, and he may seek to defend himself with hands, feet, teeth or any available object. The necessary restraint aggravates and prolongs the attack. On its subsidence the patient collects his scattered senses, regrets, apologizes, and warns against future occurrences of the same kind which he realizes to be beyond his control. Sometimes the delusions continue during the remission as well. Sexual excitement is common in man as in animals.

The convulsive paroxysms may last ½ to 1 hour, and they tend to encrease in duration and force. A violent paroxysm may cause sudden death from asphyxia or apoplexy.

Sooner or later exhaustion and paresis appear. The convulsions become gradually weaker, the reflex irritability and hyper-

æsthesia abate, and the patient may become once more able to swallow, but an ascending paralysis, beginning in the limbs spreads over the body and death occurs in from one to eighteen hours. In the paralytic stage there may still be slight jerking of the muscles, or tremors, but violent convulsion no longer occurs, and there is extreme prostration, with hurried, rattling breathing, small, weak, irregular pulse and finally, stupor and coma.

Diagnosis in man. The only additional point, to those already stated for animals, is in regard to *lyssophobia*. This false form of hydrophobia is usually fortified by the fact of a bite, but as a rule it lacks the exaltation of common sensation and of the special senses which characterizes genuine hydrophobia. Very often also there is a flaw in the history, the dog that inflicted the bite is unknown and may still be alive, in which case no medicine is so good as to bring the healthy dog into the presence of the patient. The dog may have been killed by an excited community without any identification of his symptoms as those of rabies or any post mortem examination to throw light on his case. The attack may have come on after a conversation on the subject of the bite, or of rabies, and perhaps, as in the case of the Montpellier cadet, long after and when the patient had for the first time heard that the dog that bit him had been mad. It may be that rabies does not exist in the district and that no other victim in man nor beast can be adduced. It may be that the patient has a nervous organization or is subject to hysteria, and therefore specially predisposed to any disease of the imagination. Such cases cannot be accepted as rabies until a successful inoculation has been made on one or more animals.

RABIES AND HYDROPHOBIA. LESIONS. TREATMENT AND PREVENTION.

Lesions : blood fluid or clot diffluent. Fauces, pharynx and larynx congested, exceptionally ulcerated. In dog, mouth cyanotic, with tenacious mucus, sublingual petechiæ and erosions, stomach contains many foreign bodies, but no food, small intestines and cæcum empty, and like stomach congested : petechiæ on skin and elsewhere, cutaneous and cardiac veins gorged, hyperæmic liver, kidneys and bladder : brain congested, capillaries dilated or blocked, hæmorrhagic, leucocytic collections in lymph spaces, nerve cells swell up with hyaline and Negri bodies near nuclei, and neuroglia

has hyperplasia, especially near the respiratory centre. Congestion of nerves. Leucocytosis. Therapeutic treatment : Orrotherapy : of little avail. Nerve edatives ; darkness, quiet, nutritive enemata, chloroform, chloral, etc. Prevention : eradicate the virus ; muzzle all dogs absolutely, under heavy penalty, for one year ; Gower's view ; examples of muzzling ; collar with name and owner, shoot all unmuzzled dogs, cage for six months bitten dogs and cats, also all imported dogs, shut up in cage for 10 days all dogs that have bitten ; treatment of bites, tourniquet, cup, suck through tube, wring wound, cauterize—hot skewer, cautery, mineral solid caustic, mineral acids on pledget or through a tube ; Pasteur method : emulsion of spinal cord (of rabid rabbit) after aseptic æration in vitro for 3 to 14 days, injected in graduated doses for 21 days, table of doses, table of mortality ; Orriotherapy : by blood serum of immune animal : advantages, disadvantages—technique ; Use of sterilized brain matter from rabid animal : experiment ; protection by snake venom.

Pathological Anatomy. The blood is fluid or the clot diffluent. Congestion of the fauces, pharynx and larynx is patent during life but may have disappeared after death. Yet I have seen extensive ulceration of the vocal cords in a rabid cow. The congestion may extend to the trachea, bronchia and lungs. In *dogs* the buccal mucosa is often cyanotic, covered with a thick mucus, and may present sublingual ecchymoses and erosions, and wounds of various kinds made by objects bitten or swallowed. The stomach in the same animal is usually almost pathognomonic, being filled with foreign bodies of all kinds—straw, hay, hair, wood, coal, pebbles, pieces of metal, cord, leather, cloth, earth, sand, etc.—the result of the depraved appetite. There is an absence of normal food principles in stomach and small intestines and the cæcum and colon are usually empty. The gastric mucosa is congested, and may be wounded and its contents mixed with blood. Such a condition of the stomach in a dog, that has been bitten, and which after a customary incubation period has shown symptoms like those of rabies, is virtually diagnostic.

The following lesions are common to man and animals : Marked emaciation, cyanosis or petechiæ in skins that are naturally white, early sepsis, cutaneous veins and heart gorged with dark inspissated blood, hyperæmic liver and kidneys, and slightly congested, petechiated empty bladder.

The most important lesions, however are those of the central nervous system. In seven cases out of nine, Gowers found these very distinct. There were vascular disturbance, capillary dilata-

tion, capillary clots, minute hæmorrhages, and accumulations around the affected capillaries of leucocytes occupying the lymph spaces. Benedikt and Babes attach much importance to the formation of hyaline patches in the thickened walls of the vessels and around them, compressing the vessels in some cases to virtual obliteration. The nerve cells swell up, and show small hyaline bodies in the vicinity of the nuclei, and these latter finally disappear. Germano and Capobianco found in addition marked hyperplasia of the neuroglia. Babes, who looks on these changes as pathognomomic, takes a small portion of spinal cord, hardens it in alcohol for 24 hours, stains it with aniline red and examines for the characteristic hyaline nodes.

These brain lesions have been found mainly in the medulla near the floor of the fourth ventricle and the respiratory center, but they are also to be found in other parts of the encephalon and spinal cord. The greater constancy of the medullary lesions serves to explain the characteristic symptoms.

Histological changes in the Gasserian ganglion of the fifth nerve, or the flexiform ganglion of the tenth just below the lacerated foramen give conclusive results in advanced cases. The ganglion is placed for a few hours in a saturated aqueous solution of mercuric chloride, then washed in water, then in alcohol, then mounted in paraffin and sectioned. The sections are stained in Delafield's hæmatoxylon solution, and placed under the microscope. Normally each nerve cell is enclosed in a capsule, which contains also a single investing layer of endothelial cells. In dogs dead of rabies, the single layer of endothelial cells is replaced by a great aggregation of cells evidently the result of the proliferation of the single layer. The ganglion cell under their encrease has atrophied or even completely disappeared. This is met with throughout the whole ganglion. These lesions are constant in dogs that have died of the disease, but may be altogether absent in subjects killed in the early stages. They are best shown in the dog, and less so in man or ox.

Other lesions are the Negri bodies, cosinophile granules found in the Purkinje cells of the cerebellum and the large ganglion cells of the Cornu Ammonis. These cell-inclusion bodies may be round .5 to 2.3μ, or oblong .5 x 1.5μ or 2.4μ. When fixed in Zenker's fluid, embedded in paraffin, sectioned, stained 10 to 15

minutes in a saturated alkaline solution of cosin, then 3 to 5 minutes in alkaline methyline blue, they form bright red objects in the blue nerve cell. They are most easily discovered in advanced cases, and were shown in 344 out of 550 rabid dogs (Poor). Similar bodies were found in one case of experimental tetanus, and this, together with their absence in many cases, especially in the early stages, detracts from its value as a reliable pathognomonic lesion. Negri and others, however, recognize in these the etiological factors of the disease, and would predict rabies or no rabies on their presence or absence.

When a practitioner has skill, leisure and laboratory facilities to examine the plexiform ganglion or cerebellum as above, he may gain valuable corroborative evidence, but standing alone, neither is trustworthy in the early stages, so that the old well attested means of diagnosis, by anamnesis, history, symptoms, and gross lesions, remains as the generally applicable and reliable diagnostic phenomena.

Congestions of the peripheral nerves have also been found. Lüttkemüller found in rabies a moderate increase of the white blood corpuscles and a great number of microcytes.

Therapeutic Treatment. It was long thought that rabies was necessarily fatal, as indeed nearly all developed cases are to the present day. For this reason and much more on account of the risk of preservation and propagation of the deadly germ, the attempts at curative treatment in the lower animals have been looked on as utterly unwarranted or absolutely criminal. Yet it is now known that very exceptionally a recovery takes place, and in that case immunity for the future may be counted on. Yet the frightful danger attendant on the preservation and treatment of a rabid animal, may well forbid the keeping of any of the lower animals affected by rabies unless it be in the safest seclusion and for the production of immunizing or curative products.

Orrotherapy with the blood serum of an immunized animal is of little value, and attended by risk from the rabid animal, but will be noticed below as a prophylactic.

In man when the disease is manifested, palliation has been obtained and very exceptionally recovery, under darkness, quiet, nutritious enemata and antispasmodics or soporifics. Among such antispasmodics and nerve sedatives may be named chloroform,

chloral, curare (3 alleged recoveries), eserine (1 recovery), pilocarpin (1 recovery), morphia, datura, atropia, and bromide of potassium. Others have recovered without any medicinal treatment, so that the mildness of the attack must be duly considered in every case.

Prophylaxis. The most effective way of preventing rabies is to eradicate the virus from the country. All immunizing measures resorted to after the infecting bite has been sustained, are of little value as compared with this, they may save the bitten individual, but they do nothing to prevent others from being bitten in the future, and indirectly they contribute to the maintenance of the disease by drawing attention from such radical measures as would rid the country forever of the scourge. In his great work on Diseases of the Nervous System, Gowers puts this not a whit too strongly when he says: "The enforced muzzling of dogs for a period of one year would almost certainly stamp out the disease. That such a measure is not adopted is a national disgrace, which is accentuated by the fact that the Government derives part of its revenue from a tax upon dogs. The opposition to the use of the muzzle is one of the strangest developments of morbid sentiment. There are apparently thousands of well-meaning people who would prefer that hundreds of dogs should perish every year of a painful disease, that many human lives should be annually lost, and scores of persons should be subjected for months to acute mental agony—rather than that dogs should be made to wear an apparatus which causes them a trifling annoyance. This perverted sentiment ought to be met with universal abhorrence as a disgrace to humanity." Such a statute, backed by a penalty in some degree commensurate to the homicidal criminality of the person who would leave his dog free to inflict this horrible disease on humanity, would doubtless be effectual, but some nations have such laws on their statute books, and yet allow them to become dead letters. Others have enforced them to good purpose. Berlin in 1853 had many cases of rabies and muzzling was enforced. In three years the disease was completely eradicated and the city enjoyed nine years of immunity or so long as the law was enforced. Similar successes were met with in Holland, new cases occurring only on the borders or in imported dogs. London in 1889 had 123 cases and

muzzling was enjoined. In 1892 the cases were reduced to 3, and the muzzling law was suspended, and a steady yearly encrease resulted, until the 1st three months of 1896 furnished as many as 72 cases.

In the absence of this radical measure muzzling should be enforced for a year in any locality where a case of rabies has occurred, and every dog should wear a collar with the name and residence of his owner inscribed on it. All stray dogs and all unmuzzled ones should be summarily shot. Dogs and cats that have been bitten by rabid animals should be destroyed or shut up in cages for six months under veterinary supervision. Imported dogs should be similarly secluded. Dogs that have bitten animals or men should be shut up for ten days under supervision, when, if rabid, the animal will develop unequivocal symptoms.

Treatment of bites. Absorption from a wound in a limb may be prevented by applying a tourniquet. Wounds on the body may be cupped, or sucked through a tube. Or the wound may be wrung to encrease the flow of blood. As soon as possible it should be thoroughly cauterized. A hot skewer, a Paquelin cautery, a stick of silver nitrate or zinc chloride or caustic potash or a crystal of cupric sulphate will meet this end. If liquid caustics are to be employed they can be applied to all parts of the wound by means of a pipette, a glass tube, or swab.

With thorough cauterization shortly after the bite there is practically nothing to fear, and even if it has not been applied for hours after, it is still valuable in destroying the poison left in the wound from which a continuous infection of the brain, by the transmission of the unknown germ and its toxins, would otherwise take place. It has besides, in the human being, a good moral effect against lyssophobia by giving the bitten person a certain sense of protection.

The Pasteur Method. This is based on the fact that the spinal cord of the rabid rabbit when removed aseptically, and kept *in vitro* in a dry atmosphere, loses in virulence day by day, until on the fourteenth day it is harmless. To render the air more drying, caustic potash is introduced into the flask. The culture of the poison in rabbits intensifies its virulence, until the virus becomes the strongest known and when inoculated subdurally, reduces the incubation to six or seven days.

In Pasteur's early experiments he began injecting the emulsion of the cord desiccated for 14 days, following with that of the 13th day, and so on to that of the 5th. It was soon found that this was comparatively ineffective when inoculation had been made with a strong virus or in a large dose, and the treatment for such cases was modified to what is now known as the intensive method. The weaker forms of the virus are given at shorter intervals on the first days of treatment, and the stronger forms repeated again and again, and, in place of a 15 days, course of treatment, this is extended to 21 days. The following table illustrates the course :

Day of Treatment	*Number of Days that cord had been desiccated.*	*Dose Injected.*
1st day { morning.	14 days / 13 days.	3cc.
1st day { evening	12 " / 11 "	
2d " { morning	10 " / 9 "	3cc.
2d " { evening.	8 " / 7 "	
3d " { morning.	6 "	2cc.
3d " { evening.	6 "	
4th "	5 "	2cc.
5th "	5 "	2cc.
6th "	4 "	2cc.
7th "	3 "	1cc.
8th "	4 "	2cc.
9th "	3 "	1½cc.
10th "	5 "	2cc.
11th "	5 "	2cc.
12th "	4 "	2cc.
13th "	4 "	2cc.
14th "	3 "	2cc.
15th "	3 "	2cc.
16th "	5 "	2cc.
17th "	4 "	2cc.
18th "	3 "	2cc.
19th "	5 "	2cc.
20th "	4 "	2cc.
21st "	3 "	2cc.

Under this treatment the system becomes educated in the production of antitoxins, and perhaps also in phago-cytosis so that when subjected to the lethal doses of three, four, five and six days preservation, it successfully resists them. The most conclusive argument in favor of its efficacy is this undeniable fact that the individual escapes death under injected doses which in any unprotected system would prove fatal.

The results as given by the report of the Pasteur Institute are furnished in the following table, from which are excluded such cases only as developed the disease during the course of treatment, which therefore remained incomplete.

Years.	*Persons Treated.*	*Deaths.*	*Mortality per cent*
1886	2671	25	0 94
1887	1770	14	0 79
1888	1622	9	0 55
1889	1830	7	0.38
1890	1540	5	0.32
1891	1553	4	0 25
1892	1790	4	0 22
1893	1648	6	0 36
1894	1387	7	0.50
1895	1520	5	0.33
1896	1308	4	0.30
1897	1521	6	0.39

The following table gives the number of individuals treated who had been bitten by animals which had been proved rabid by successful inoculation of other animals, and of those bitten by reputedly rabid animals, and their respective mortality.

		Died.	*Mort. per ct.*
Bitten by animals proved rabid by inoculation	2,872	20	0.69
Bitten by animals pronounced rabid by veterinarian	12,547	61	0.48
Bitten by animals suspected of rabies	4,747	15	0.31
Average mortality			0.46

The Pasteur treatment by its great success in persons who have already been bitten has in a great measure robbed hydrophobia of its terrors, only it must be resorted to as early as possible in the period of incubation. It has also been advocated as a means of immunizing subjects that have not been bitten but are more or less liable to be so, and on this basis a large number of dogs have been passed through it. This is not likely to be adopted in the case of the human being, the more so that a few, although on the whole a very limited number of persons, have developed rabies long after the taking of the Pasteur treatment. This has been attributed to the retention of latent germs in the system, and argues besides, a remaining susceptibility to the poison.

In spite of its brilliant success and the great boon it has been to humanity, the Pasteur treatment is not an ideal one. Its suc-

cess does not consist in an entire extinction of rabies, but merely in the reducing of its evil results; its success is indeed based on a preservation and propagation of the germ and a continuous danger of infection of new subjects; finally, the proposition to end the disease by passing the whole canine race through the treatment, is open to the objections that this would require a fabulous outlay, and that even then some rare cases are not found to be fully protected. To continue the disease, when it may be exterminated, and to palliate its results by the treatment of generation after generation of dogs, must be promptly condemned by the political economist, to say nothing of the consideration of probable human infection.

Orrotherapy. It is not surprising that essays were made in the line of serum treatment. Babes and Lepp in 1889 had some encouraging results in transferring the blood of an immune animal into a healthy one. But Tizzoni, Schwarz and Centanni have especially worked out this method. These have shown that the blood serum of immunized animals destroyed the virulence of the rabic poison, whether mixed with it before injection, or injected with it, or injected within twenty-four hours afterward. A very small amount of the serum is required and though delayed until the end of the first half of the incubation period, it is only necessary to multiply the amount by six or eight times. In this it has a great advantage over the antitoxin of diphtheria or tetanus, the former of which has to be multiplied 20 to 100 times, and the latter 1000 to 2000 times in the later stages of incubation. Further, it is possible by drying to secure the serum in a permanent form which will remain active for a length of time if secluded from air and light.

This has the decided advantage over the Pasteur treatment, (1) that it employs the antitoxin already formed instead of waiting for its formation in the body of the subject injected with the attenuated virus: and (2) that it does not introduce into the system a virulent germ capable of propagating in a favorable medium, but only an agent which is antidotal to that germ.

It has the disadvantage as compared with Pasteur's method that its action is purely therapeutic in the sense of acting as an antidote, while it produces no permanent immunity. It does not, like the toxins, educate the cells to produce an encrease of anti-

tixin, and it can only protect so long as it remains in the system. Whenever it is eliminated or destroyed, the susceptibility to rabies returns. Hence it is important to continue its administration as long as the microbe remains in the system. As in tetanus and diphtheria antitoxin treatment, it is also important to destroy the microbe and its toxins in the infection wound.

The animal which is to furnish the antitoxin is immunized as in the Pasteur method by a succession of graduated doses of rabic virus. After a treatment of 20 days the rabbit or sheep furnishes a serum which is protective when injected in the proportion of 1 of serum to 25,000 of body weight, even though its use be delayed until 24 hours after the introduction of the virus. The sheep can be immunized in 12 days by doses of 0.25 gramme of emulsion of the infected cord to every kilogramme of body weight. To maintain the serum at its highest standard the treatment must be repeated at intervals of 2 to 5 months, as the animal may be able to bear it without loss of condition.

Treatment with Sterilized Brain Matter from a Rabid Animal. In 1886 I sterilized with heat an emulsion of the spinal cord of a man who died of hydrophobia and injected two rabbits with 3 one drachm doses each, and a third with 4 one drachm doses on as many successive days. These rabbits were afterwards inoculated with virulent spinal cord and remained well for nine months, while three control rabbits injected with virulent cord, but which had received no previous treatment died rabid.

Puscarin and Vesesco have shown that the virus is rapidly weakened by heat up to 60° C. at which point the virulence is destroyed. It becomes easy therefore to secure in this way a toxin uncomplicated by any living microbe.

Theoretically the sterilization and use of infecting nervous substance, should share with the Pasteur method the advantage of the selective action of the medullary matter in uniting with the toxins and robbing them to a greater or less extent of their toxicity. It has the additional recommendation that it introduces no living germ, and thus obviates any possible danger of the propagation of disease through the animal operated on.

Fernandez claims an immunity from rabies for dogs that have survived the bite of a viper. Many facts and experiments are adduced in support of this.

Synonyms. Definition : infectious disease, due to bacillus, and shown by tonic spasms of groups of voluntary muscles. Animals susceptible : warm blooded animals—dog and chickens least : ⅔ ds. solipeds, ¼th cattle. Pathology and causes : Bacillus tetani : 4 to 5μ by 0.2 to 0.3μ, often enlarged by spore at one end ; anærobic, liquefying, tardily motile, until spore forms, grows in ordinary, alkaline media under hydrogen, death point 60° to 65° C. (140° to 149° F.), for spores 80° C. (176° F.), for an hour, dried it lives for years, in putrid matter 2½ months, stains easily, saprophytic in garden mould, in ingesta of man and horse, abundant in tropics ; infection local, killed by oxygen in blood, toxins tetanize : tetanin, spasmotoxin, toxalbumen, diastase ; spasms first local near wound, then abruptly general, intravenously causes general spasms first, theory of fermentation in blood ; changes in nerve cells, neuroglia, ependyma, peripheral nerves : muscles soft, pallid, red, ruptured, fibres, ecchymosis ; rigor mortis early, marked : sarco lactic acid. Accessory causes : traumas and their causes, parturition, umbilical infection, alimentary. General symptoms : incubation 3 to 15 days, minimum 6 hours, tonic contraction of muscle groups of locomotor system beginning near infectiou wound,—trismus, orthrotonos, opisthotonos, emprosthotonos, pleurosthotonos, ocular muscles ; costive ,difficult urination, hyperæsthesia, irritability, perspiration, hyperthermia, mastication, deglutition, sucking : Symptoms in *horse* : neck raised concave above, nose elevated, nostrils wide, eyes sunken, haw protuded, ears rigid, pricked, facial muscles rigid, prominent, mouth drawn back, muscles of back hard, tail elevated, trembles, limbs extended outward, stiff, stilty, jaws clenched or open slightly, stands : Symptoms in *cattle ; sheep* and *goat ; swine ; dogs ; birds :* Course : violent cases with short incubation are rapid and fatal ; mild ones with prolonged incubation hopeful ; cattle slow, sheep, goats and dogs acute. Mortality : sheep and pigs 100 per cent. ; horses 75 to 85 ; cows 70 to 80 ; lambs very fatal. Death from asphyxia, hyperpyrexia, or exhaustion. Lesions : trauma, often healed ; congested nerves, gray horns of myelon, increase of cells and granules in nervous matter of cord, corpus striatum, cerebellum ; blood extravasations at torn muscle fibres, intestinal and cystic congestion : Diagnosis : from *strychnia poisoning* by slow advance, and persistence of spasm ; from *rabies* by absence of bite, the continuous masseteric spasms, by absence of resentment, mischief, hallucinations or depraved appetite ; from *rheumatism* by the persistent trismus, hyperæsthesia and excitability ; from *meningitis* by the trismus, perfect mentality, absence of clonic spasm ; from *tetany* by shorter and less perfect remissions, failure to develop under nerve pressure, or improve under thyroid extract ; from *laminitis* by the absence of high early hyperthermia, heat and tenderness of the feet, and advance of hind legs under the body. Treatment : best in slight cases, after long incubation, with slow progress ; antispasmodics ; rest, darkness, absolute quiet, no litter, nor visitors, slings, sloppy

food, gruels, milk, green food, at level of manger; clothing to favor perspiration; excision or antisepsis of wound, carbolic acid, bleeding, opium, prussic acid, potassium cyanide, bromides, physostigma, eserine, chloroform, sulphonal, trional, tartar emetic, tobacco, apomorphia, lobelia, phenacetin, acetanilid, cocaine, chloral, phenic acid, iodine terchloride, iodide of potassium, orrotherapy, antitoxin; best as a preventive, value decreases with development of disease; cerebral injections; brain emulsion; use up toxins in blood; no use if nerve centres are already in combination with toxins, only to ward off fresh toxin. Toxins produce leucocytosis. Prevention: disinfection of all dirty wounds, injections of phenic acid, or iodine; remove foreign bodies, use muriatic and carbolic acids; antisepsis of navel; disinfection of stables, feet, careful shoeing; immunization.

Synonyms. Lockjaw. Trismus.

Definition. An infectious disease of animals and man, characterized by tonic spasms of the voluntary muscles in a given region or more generally, with exacerbations, and dependent on the bacillus tetani.

Animals susceptible. Immunity cannot be claimed for any class of warm blooded animal. Experimentally the dog and chicken prove among the most refractory, in keeping with the comparative insusceptibility of the last named animal to strychnia, but neither can be held to be in any sense immune. Inoculated frogs become tetanic if the temperature is maintained above the normal standard. In 208 cases in domestic animals recorded by Cadiot and Hoffman, 140 were in horses, 10 in mules, 5 in asses, (solipeds, 155), 28 in cattle, 9 in sheep, 5 in goats, 5 in pigs, and 6 in dogs. Such statistics are liable to prove misleading when we have no means of comparing them with the members of the different genera from which the cases were drawn and the relative exposure of each genus to traumatic lesions (infection atria). Solipeds lead with practically ⅔ds of the entire number of cases, but these were presumably the most numerous of the domestic animals, and preëminently the work animals and therefore the most liable to traumatism. Cattle follow with ⅐th of all cases but here again the large numbers to be drawn upon, and the proportion of work oxen and wounds, are to be considered. The omnivora and carnivora are comparatively little susceptible and among these the chicken may be included. The omnivorous rat is quite susceptible.

Tetanus occurs in 1 per 1000 sick horses in the Prussian army (Friedberger and Fröhner), and in 1 per 3000 sick in that of

Wurtenburg (Hering). It is so prevalent in San Domingo that a gelding costs twice as much as a stallion (Wagenfeld). Heat and filth favor its preservation.

In man tetanus is most frequent as the result of wounds (in feet and hands) which are most likely to come in contact with the soil, and it has visibly decreased in connection with the general adoption of antiseptic surgery.

Pathology and Etiology. Sir James Simpson suggested in 1854 that puerperal and surgical tetanus was due to the absorption of a poison produced in the wound (Woodhead). Spinola charged it on infection in wounds in horses. Carle and Rattone in 1884 successfully inoculated 11 out of 12 rabbits with the products from the wound of a man suffering from tetanus. A year later Nicolaier produced tetanus in animals by inoculating them subcutem with garden mould or street dust, and found in the suppurating wounds in connection with various other microbes a minute bacillus longer but thinner than that of mouse septicæmia to which he attributed the tetanizing action. In 1886 Rosenbach inoculated two Guinea-pigs with the pus of a tetanic man, and found in the sores of the tetanic pigs the bacillus of Nicolaier in company with another larger spore-forming bacillus. In 1889 Kitasato succeeded in making pure cultures of the bacillus tetani, and successfully inoculated the disease on mice, rabbits, and Guinea-pigs producing typical tetanic symptoms and death. This was promptly corroborated by Tizzoni and Cattani and later by a great variety of observers.

Bacillus Tetani. This organism is a minute rod 4 to 5 μ in length by 0.2 to 0.3 μ in thickness, with slightly rounded ends. In many mature forms the one end is enlarged by the formation of a spherical, refrangent spore which gives the bacillus the appearance of a pin or a "drum-stick."

The bacillus is anærobic, liquefying, tardily motile, and sporogenous. When spores form the bacillus loses its motility. It grows at room temperatures, in ordinary culture media which have a feebly alkaline reaction, and in an atmosphere of hydrogen, but more actively at a temperature of 36° to 38° C. Below 14° C. growth ceases and the bacillus is killed at 60° to 65° C. The spores, however, can resist a temperature of 80° C., in water for an hour, and 100° C. for four minutes. It was this unusual

resistance of the spore to heat that enabled Kitasato to kill off the contaminating organisms and obtain pure cultures from the surviving spores. The spores will survive desiccation for years, retaining their virulence, and may live 2½ months in putrefying material. The addition to the culture medium of 1½ to 2 per cent. of glucose makes the growth much more rapid and abundant, and causes opacity in the medium. The upper portion clears up in 6 or 7 days by the precipitation of the bacilli as a grayish mass. In a glucose culture medium growth is not prevented by the presence of oxygen at the surface. The colonies formed in gelatine plate cultures show an opaque centre with fine divergent rays, and a similar radiating growth is shown in deep stick-cultures. At the end of the second week the gelatine begins to liquefy and form a little gas, and finally the whole mass becomes soft and sticky. The bacillus does not liquefy blood serum. Cultures have a disagreeable aromatic odor.

The bacilli stain readily in aniline colors and by Gram's method. The spores may be stained by Ziehl's method. To 10 parts of a 10 per cent. alcoholic solution of basic fuchsin, add 100 parts of a watery solution of carbolic acid. Float the cover glass upon this, heating gently for three to five minutes until steam begins to rise, wash well in water, and decolorize in nitric or sulphuric acid, 25 per cent. solution, then in 60 per cent. alcohol to remove color from albuminous background. Wash in water and mount. By placing the specimen for two minutes in a watery solution of methylene blue a contrast is obtained, the bacillus blue and the spore red.

Outside the animal body the bacillus has a saprophytic life in rich garden mould, street dust, stables, yards and drains, and the cracks of floors. Nicolaier failed to obtain it in soil from forests and from the deeper layers of garden earth. Marchesi found it to a depth of two metres but no more. Again it is much more abundant in tropical countries than in temperate and cold ones, and appears to be to a great extent limited to particular localities. It has been found in the intestinal contents of man and horse (Babes, Sormani), and in horse manure, and this mingling with the surface soil and generating an abundance of ammonia determines the anærobic conditions which favor the growth of the microbe. This serves to explain the remarkable prevalence of

the disease among those living or working about stables, gardeners, agricultural laborers, soldiers on campaign, and children and others walking with bare feet. The contact with rich infected soil greatly favors inoculation in any accidental wound.

An important feature in the pathology of tetanus is that the bacillus is confined to the seat of the inoculation wound. The many attempts to transmit the infection by blood, nervous matter, and by one or other of the tissues have uniformly failed, though the pus of the infected wound has proved virulent. Similarly, the attempts of Kitasato and others to obtain cultures from the animal liquids or tissues apart from the wound have been futile.

By inoculating the toxins remaining in the pus of the infection wound, however, or in virulent cultures from which the bacilli have been removed by filtration or in which they have been destroyed by heat, all the symptoms of tetanus can be produced (Kitasato, Kund Faber, etc.) In such cases too, the symptoms appear at once, as soon as the toxin is absorbed, and not after a definite period of incubation as in inoculation of the unaltered virus. Kitasato, Vaillard and Vincent reached this conclusion by another channel. They inoculated mice at the root of the tail with virulent tetanus cultures, and at definite intervals after, namely, half an hour, one hour, and one and a half hour, they made a circular incision round the wound and thoroughly cauterized the whole, thus destroying all the inoculated bacilli. They found that tetanus was prevented in those animals only which were operated on in the first half hour. Again, Kitasato injected mice with 0.2 to 0.3 cc of the blood from the heart of a fresh tetanus cadaver, and thereby produced typical tetanic symptoms and death in 1 to 3 days.

Various poisons have been separated from cultures of bacillus tetani. Brieger isolated three substances—tetanin, tetano-toxin and spasmo-toxin—which in large doses caused tetanic symptoms and even death. Brieger and Fränkel later isolated a toxalbumen which proved of incomparably greater potency. Again, Brieger, Kitasato and Wehl separated what appeared to be an enzyme or diastase which proved 500 times more potent than atropia. This was in form of yellow, transparent flakes, soluble in water, but which was not destroyed by drying, nor in the dry state by absolute alcohol, chloroform nor anhydrous ether, but which, like

the virulent cultures of tetanus, was easily destroyed by acids, alkalies, hydrogen sulphide, or heat. Like the natural virulent product this may be kept unchanged for months on ice, apart from the light, or with the addition of 0.5 per cent. of carbolic acid, or its own bulk of glycerine. It kills the Guinea-pig in a dose of 0.000025 gramme, and the mouse in a dose of 0.00000025 gramme.

While the propagation of the bacillus in the animal body appears to be local, and the general tetanic symptoms are caused by the absorption of the poison, it remains to be seen on what organ this directly operates, and what accessory conditions favor its efficiency.

In *cases due to inoculation* the spasms are at first local in the vicinity of the inoculation wound and later become general. Kund Faber shows that there is no gradual transition from the local manifestations to the general, but the latter appear abruptly and in force as a new and independent phenomenon. When we consider further that in inoculation with pure cultures (uncontaminated by pus or saprophytic microbes) the wound often heals promptly, without any sign of remaining local irritation, we may conclude that simple nervous irritation in the sore cannot be invoked as a cause of the early local spasm. It is more likely due to the local diffusion of the poison into the peripheral nerves while the little that has been absorbed is as yet too much diluted in lymph and blood to seriously derange the nerve centres.

When general spasms set in it must be assumed that the poison has reached the nerve centres in toxic quantities, either through the circulation or as is alleged by Babes and others through the nerve trunks. When the poison is injected intravenously the general spasms are the first to appear. Again the section of the nerves of a limb, before inoculation, prevents spasms in its peripheral muscles when all the body beside has become tetanic (Tizzoni and Vaillard). The removal of the brain from a tetanized frog had no effect, while the removal of a portion of the spinal cord abolished the spasms in the muscles corresponding to that part. Moreover Gumbrecht cut the whole of the sensory nerves of a limb but the spasms occurred in its muscles notwithstanding. It must be admitted, therefore, that the general tetanic spasms are induced by disorder caused by the poison in the spinal nervous centres.

Gumprecht and Goldscheider claim that the poison reaches the spinal centres by way of the nerve trunks, basing the conclusion on the observation that the spasms sometimes remain for a time more marked on that side of the body on which the wound or inoculation was made. Absorption through the circulation also is conceded.

Courmont and Doyon claim that the product of the bacillus tetani only operates as a ferment, which produces in the blood the real tetanizing agent, basing the conclusion on an apparent delay in its action, in man, as compared with strychnia, and on the prompt action of the injected blood of a tetanic animal in which this poison is presumably preformed. It should be noted, however, that the disease in man is only seen after accidental inoculation of the bacillus, and that time must be allowed for the increase of the microbe.

Vaillard and Vincent have shown that the promptitude and certainty of the result depend on the age of the culture employed. A culture of 5 days in bouillon at 20° to 22° C. will not harm a Guinea pig in a hypodermic dose of 0.25cc. to 0.5cc. A culture of 20 days old is deadly.

The action on the nerve cell of the spinal cord has been investigated by Goldscheider and Flatau, who found degeneration of the chromatin granules within a short time after inoculation. (Centr. für Allg. Path. Anat. 1897). W. K. Hunter found that the ganglion cell stained more diffusely than normal cells. There were also some capillary dilatation and punctiform hæmorrhages in certain cases (Brit. Med. Jour. 1897).

Péchoutre examined the lumbar enlargement of tetanic rabbits by Nissl's method and found the following lesions in the motor cells of the anterior horns: 1st. A partial or total disappearance of the distinct outer marginal line; enlargement of the cell and pericellular space; diffuse coloration of the acromatic substance; a disappearance of the regular concentric disposition of the granules of Nissl which were in part reduced to a fine powder; 2d. Encrease of nucleus and nucleolus.

Others have observed encrease of the cerebro-spinal fluid, thickening of the ependyma, nuclear proliferation in the neuroglia, and softening of the cord, but in many cases no appreciable lesions in the nerve centres have been found, and none can be

affirmed as constant. Neuritis in the region of the wound is sometimes found, especially if the lesion is a contused or painful one.

The muscles often show lesions, the result of the violent contractions. There may be points of ecchymosis and partial rupture of individual fibres, they may be of a deep red, or again pale, soft, and as if parboiled. There may be hyperæmia or œdema of the lungs, congestion of the larynx, ecchymosis on the pericardium and other serous and mucous membranes, and congestion of the liver, spleen and kidneys. Rigor mortis sets in rapidly and is usually very persistent. The muscles contain an excess of lactic acid.

Accessory Causes. Whatever contributes to traumas must be classed in this list. Solipeds, work oxen and dogs are especially exposed in this sense. In all animals, castration wounds; in horses and lambs, amputation of the tail; in solipeds, pricks, bruises and fistulæ of the feet; all kinds of surgical wounds; in females, the parturient condition; and in the new born, the umbilical sore form infection atria. The tendency to infection in wounds of the feet in animals, and of the hands and feet in man, is easily explained by contact with the virulent earth or dust. Children running barefoot, or injuring their bare knees and soldiers sleeping on the ground are similarly exposed. The contamination of the clothes is the main condition. It has been held that infection never takes place from the gastro-intestinal canal, but the facts that the bacillus is frequently present in the prima viæ and that the mucosa is often perforated by blood-sucking parasites, suggest that some cases (idiopathic) are probably due to intestinal infection. The gland ducts also and the follicles of Peyers' patches and of the solitary glands offer available fields for the colonization of the bacillus and for infection.

General Symptoms in Animals. In experimental cases, in which there has been a large intravenous injection of the blood of a victim of tetanus, the symptoms may set in speedily and violently. In casual cases, however, there is usually an *incubation* period varying on an average from three to fifteen days in the *horse* while it may be as short as two days in *cow* and *sheep*. Hoffmann quotes one incubation in the horse as but six hours after a wound in the neck, and another as twenty-five days, fol-

lowing a castration. The last is rather unreliable as infection may have taken place long after the operation. He quotes cases in the pig and goat, after castration, as eight to fourteen days, and one in the dog almost immediately after a bite on the loins.

Following the incubation the marked phenomena are tonic contractions of groups of muscles beginning usually with those near to the seat of the infected wound and extending with varying rapidity to the locomotor muscles generally (limbs, croup, back, loins, neck, tail, abdomen) and those of mastication (jaw) and the eye. The muscles of respiration are only involved at a late date, causing stertorous breathing and it may be asphyxia. Peristalsis is impaired so that there is some costiveness and tardiness in digestion. Urination becomes difficult and infrequent on account of the difficulty of assuming the normal position for the act, and spasm of the sphincter vesicæ and dangerous distention of the bladder may follow. The urine is often albuminous and has a high density and color. Priapism is not infrequent in the male.

The predominance of the spasm in special groups of muscles has been the occasion of giving different names to individual cases.

Trismus or *lockjaw* is that condition in which the muscles of the jaws are violently contracted so that the incisors can only be separated slightly or not at all.

Orthrotonos is that condition in which the muscles in tonic spasms keep the neck, back and loins rigid, straight and unbending, in one horizontal line.

Opisthotonos is when the muscles of the spine are in rigid spasm, so that the back and loins are slightly depressed, the tail elevated and trembling, the neck drawn upward with a concave superior border ("ewed"), the head extended and the nose elevated.

Emprosthotonos is caused by spasms of the muscles of the ventral aspect of the body, with arching of the back and tucking up of the abdomen.

Pleurosthotonos is when the trunk is spasmodically bent to one side, right or left.

Spasms of the muscles of the eyeball, cause sinking, and apparent diminution of that organ, with the protrusion of the membrana nictitans over one-third, one-half or even more of its front surface. This is often referred to by owners as "hooks".

More striking and pathognomonic than the above is the extreme hyperæsthesia and irritability which rouse into activity or aggravate the symptoms under the slightest cause of disturbance. The effort required to feed from the ground, the stretching of himself to urinate or defecate, the rustling of straw litter under his feet, loud talking, or other sudden noise, banging of doors and windows, hammering in the vicinity, a current of cold air, a flash of light, moving the patient in the stall, attempts at mastication, simple handling, administering medicine, or sudden jerking of the head upward promptly brings on a paroxysm of spasm. Pushing the head suddenly upward or jerking on the halter, is often resorted to as a means of diagnosis, the sudden resulting rigidity of the muscles generally, the rolling of the eye, its retraction toward the depth of the orbit and the protrusion of the membrana nictitans over one-half, one-third or more of the cornea, bringing out the diagnostic symptoms in a striking manner. In very severe cases the head may be drawn upward and backward almost to above the loins (Henry), or one or more of the dorsal or lumbar vertebræ may be broken or crushed (Zundel). A sudden loud noise will sometimes cause the exhausted animal to drop to the ground.

Perspiration is not uncommon. Breathing is usually accelerated, the encrease being in ratio with the violence of the spasms. Pulse and temperature are usually normal at first and may be in slight cases throughout. Even in severe cases the pulse does not rise so much as the respiration. In violent and fatal cases the temperature often rises excessively before death (104° to 110° F.).

When the jaws are not absolutely closed, the tongue is often wounded by the teeth, and, in any case is covered by a tenacious mucus, which may hang in strings from the lips. If the jaws are still movable, mastication is still carried on, but slowly, painfully and imperfectly, and deglutition is more or less difficult. Young animals are unable to suck.

Symptoms in the horse. When the symptoms are fully developed, they are very characteristic. The neck is raised, often concave along its upper border, the nose raised and protruded more or less, the nostrils widely expanded, the eyes sunken, fixed and anxious, with dilated pupils and protrusion of the haw outward and upward from the inner canthus, the ears are pricked, rigid,

and drawn toward each other at their tips, the facial muscles may stand out visibly and are firm, the angle of the mouth is drawn back, the veins of the head are full and prominent, saliva froths or drivels from the lips, the tail is elevated and during paroxysms will tremble, and the muscles of the back and limbs are projecting and hard. The limbs are extended outward to give a wider base of support. If moved, the general stiffness is at once seen. The patient cannot be turned round in his stall, he may not even be able to turn the neck to one side, and if backed he resists, or accomplishes the movement only with the greatest difficulty. In walking, the limbs are used as stilts with little or no bending of the joints, and if turned, the body is not bent but moved around with difficulty as if one rigid mass. The pulse is small and hard, the breathing slightly hastened, and the mucosæ congested and reddened. The jaws may be firmly closed, or they may still part for a time half to one inch. In all severe cases the patient obstinately stands, and if he should drop, or lie down, the breathing and spasms are usually encreased and, in the efforts to rise, the respiratory muscles may become spastic with promptly fatal results.

In *cattle* the same general symptoms prevail. The stiffness of neck and back, the habitual elevation of nose and tail, the stiffness of the legs, propped outward for support, and moved like unbending posts when made to walk, the hardness of the muscles, standing out under the skin, the rigidity of the lips, firmly closed or slightly opened, the general fixity of the erect, retracted ears, and the sunken appearance of the eyes with marked protrusion of the haw, are largely as in the horse. The muzzle is usually dry and hot, the jaws clenched, the tongue firmly compressed against the palate and covered with thick, tenacious mucus, and the flanks are often flattened by the contraction of the oblique muscles, so that they descend almost vertically from the lumbar transverse processes. There is great difficulty in turning and the trunk moves in rather a rigid mass without bending laterally, and the limbs are stiff and stilted. Tympany of the rumen sets in early with oppressed breathing and arrest of defecation and urination, which had been already difficult. The reflex excitability to noises, or other causes of disturbance is often less than in the horse. In cases following metritis, this may be due in part to the depressing poisons absorbed.

Sheep and Goat. Show the same general rigidity of trunk and limbs, the drawing of the head and neck upward and backward, the elevation of nose and tail and the firm closure of the jaws. As the disease advances they may lie on the side with legs straitened and rigid and head and tail raised toward the back. The occlusion of the eye by the haw is the same as in the horse.

In *swine* the spasms begin with the jaws and face, and extend to the neck, back and limbs with the same general symptoms as in other animals. Champing of the jaws and profuse frothing at the mouth have been noted and the protrusion of the haw is characteristic. As in sheep, the animal may lie on its side with head and limbs rigid and an early death may be looked for. Convulsions are easily distinguished by their transient character.

In dogs tetanus is rare. Möller had two cases in 50,000 sick dogs, Friedberger and Fröhner but one out of 70,000. Cadiot saw two cases in ten years of the Alfort clinic. Labat had several cases in sheep dogs. A slight transient trismus has been noticed as common in puppies. When generalized there is stiffness of the trunk and limbs, abduction of the members. The spine may be straight and rigid or drawn upward and backward, and the loins depressed. The haw covers the eye more or less, the lips are rigid, the jaws clenched, and the skin of the forehead wrinkled. The ears are stiff and drawn toward each other, or backward. The reflex excitability is as great as in the horse, the slightest touch or sudden noise producing violent paroxysms. Inability to bark is a marked feature. Temperature may be normal or up to 107° F.

In birds it is very rare, in keeping with the insusceptibility to strychnia, ergot and other tetanizing agents. Dreymann gives one case in a *turkey*, and Babes' experimental cases in pigeons and chickens from a specially virulent bacillus obtained from the horse. The pigeons suffered much more certainly and severely than the chickens, in which there was a marked power of resistance. Dreymann's turkey moved with stiff limbs and body, had the wings clinging firmly to the body, the head and neck extended and the bill firmly closed. The haw protruded over the eye, and there was hurried and oppressed breathing.

Course. Duration. The course of tetanus varies with the genus affected, with the individual susceptibility and above all

with the length of the incubation, and severity of the attack. Cases that set in with great violence, after an incubation of two or three days or less are likely to advance to an early death. An early generalization of the spasms, with high temperature (104° F), hurried breathing, congestion of the mucosæ, and extreme excitability may end fatally in twenty-four hours or within a week.

If on the other hand the incubation appears to have extended over one, two or three weeks; if the disease is at first equivocal, with some stiffness and firmness of the muscles, but with little or no trismus; if the patient can open the jaws an inch or more and masticate even slowly for a number of days after the onset of the first symptoms; if the haw projects only slightly over the eyeball and the excitability is not extreme the prospect for recovery is much better. Such tardy cases may seem to stand still for a week and then have a slight aggravation and this may be repeated, or a slow improvement may set in and go on gradually to complete convalescence. Improvement may be manifested by a softer or more relaxed condition of the muscles, by a slightly freer movement of the limbs and jaws, by a greater ease in swallowing, by encreasing movements of the ears and eyes, by the lessened projection of the haw, by the freer breathing and circulation and by the permanent lowering of temperature to the normal standard. Convalescence is always slow, but especially slow in severe cases in which time must be allowed for repair not only of the central nervous lesions but also of the ruptures and trophic changes in the muscles.

In *cattle* the disease is usually slow in its progress and improvement may not set in till the close of the third week. In sheep, goats and dogs on the other hand it is habitually acute, and death may supervene from the third to the eighth day. In the horse all forms are met with and the result will vary according to the severity of the attack.

Mortality. Friedberger and Fröhner sets the mortality in sheep and pigs at nearly 100 per cent.: in horses at 75 to 85 per cent.: and in cows at 70 to 80 per cent. In tetanus neonatorum in lambs, the deaths reach about 100 per cent. In this case the disease usually sets in within forty-eight hours after birth, and with a very high temperature difficult deglutition becomes a

marked feature, so that if the patient is not speedily killed by dyspnœa, it soon perishes from starvation and exhaustion.

The *cause of death* is usually *asphyxia*, *hyperpyrexia*, or *exhaustion*.

Lesions. There are no constant or pathognomonic structural changes in tetanus. Those that are found are inconstant and as a rule secondary. A wound (entrance channel) can usually be made out, often in the region of the foot, or in connection with castration. In the new born there is the unhealed navel, and in parturient cows the catarrhal, septic or injured womb. In the seat of such wound may be found the foreign body (nail, splinter, etc.), and some pus or simple congestion or even necrosis. The nerve trunks leading from such infected wound may be hyperæmic. The presence of the bacillus in the wound may be determined by microscopic examination or inoculation on a small animal.

The changes in the nerve centres may be congestion of the horns of gray matter, and there may be slight hæmorrhage, exudation, especially shown in the encrease of the cerebro-spinal fluid, softening, cell proliferation, and granular invasion of the nervous tissue. In separate cases the myelon, the corpus striatum and the cerebellum have shown lesions. The meninges are occasionally hyperæmic. Spinal lesions have been noted especially in the bulbo-cervical and lumbar regions. Exceptionally in the horse there are blood extravasations from fractures or dislocations of the vertebræ.

In subjects dying of asphyxia, the lungs and right heart are congested, and the blood may be black, only loosely coagulable and with free hæmoglobin. There is congestion of the intestinal as of the respiratory mucosa, and also of the liver, spleen and kidneys. The congested bladder usually contains urine contrary to what is the case in rabies.

Diagnosis. From *strychnia poisoning* tetanus is distinguished by the gradual and progressive approach of the spasms and by the absence of the intervals of complete relaxation which separate the rapidly recurring and violent spasms of strychnia. In tetanus the spasms may be modified but never completely intermitted, and more or less stiffness, trismus and protrusion of the haw constantly persist. In strychnia too, the paroxysm is far more intense than

in the early stages of tetanus. The spasms of strychnia are general, while those of tetanus are often most intense in particular groups of muscles, especially at first in the vicinity of the inoculation wound.

From *rabies*, tetanus is easily distinguished by the absence of any history of a bite; by the persistence of the tonic spasms especially of the masseters and abdominal muscles during the intervals between the more violent paroxysms (in rabies there is temporary complete relaxation); by the absence of clonic spasms which alone occur in rabies; by the absence of the paralysis which characterizes advanced rabies; by the mental clearness and the absence of hallucinations or mischievous disposition which are marked features of rabies; by the absence of the depraved appetite of rabies; and by the fact that the brain does not contain the infecting germ as is the case in rabies.

From *rheumatism of the neck* (torticollis) tetanus is easily distinguished by the permanent trismus which is not shown in rheumatism, and by the fact that spasms are easily roused by any artificial excitement, indicating an extraordinary hyperæsthesia and excitability which are nearly absent in rheumatism. The steady unmistakable progress of tetanus is in itself diagnostic.

From *meningitis* tetanus is to be diagnosed by the presence of trismus without impairment of the mental faculties or fever. In meningitis the spasms are usually confined to particular groups of muscles and do not become generalized under active excitement as in tetanus. Even if the spasms of meningitis affect the jaws and pharynx they are rarely paroxysmal or roused by excitement as in tetanus. They may even be clonic.

Tetany is more commonly localized in particular groups of muscles, and show longer and more irregular intervals between paroxysms than does tetanus. It is improved by thyroid extract, and may be roused at will to contraction by pressing on the nerve going to the affected muscles.

Laminitis in its most violent form and early stages, may be confounded at first glance with tetanus, but the high fever, the standing on the heels, the advance of the hind legs under the body, the great heat and tenderness of the feet, the impossibility of standing on one fore foot when the other is lifted and the strong pulsation of the digital arteries, are sufficiently distinctive.

Treatment. For fully developed tetanus no known resort of theraputrics can be relied on. In slight cases that have shown a long incubation and a slow increase and extension of spasm and in those having trismus only, a recovery may be expected. Treatment has been conducted largely on theoretic lines and may be divided into antispasmodic, eliminating and antidotal or antiseptic.

Rest, darkness and absolute quiet are the first and main considerations. A dark stall, with no straw litter, the rustling of which often excites the patient, but rather a little chaff, saw dust or even earth to prevent noise from the feet, and the exclusion of all visitors are essential. As a rule slings should be put under the patient so that he cannot lie, nor drop down, and this becomes more imperative as the disease advances. The aggravation of the spasms when down, and the danger of their extension to the respiratory muscles are far more to be dreaded than the temporary excitement caused by the application of slings. Food should be sloppy mashes, of bran, middlings, oat meal, linseed meal, gruels or milk, or green food may be allowed in moderate amount if the jaws are still movable. It must not be forgotten that digestion is impaired and food that is indigestible, especially fermentescible, or in excess, may arouse fatal colics and bloating, yet in a protracted exhausting disease like tetanus, the strength must be sustained by all means in our power. Pure water should always be accessible. Food and water should be furnished in buckets at a level which will not necessitate either raising or dropping the head to get to them (about 4 feet). The food must be given often, in small quantity to avoid fermentation and spoiling. If noise cannot be wholly excluded it may be an advantage to put cotton wool in the ears. I have seen a mare recover when completely covered with cotton wool under blanket and hoods.

Local antiseptic treatment. Theoretically this is of great value since the microbe is confined to the inoculation wound and by the time the first symptoms appear, the spores have developed into bacilli and are in a condition to be easily destroyed by disinfectants. We can, therefore, by caustics or active disinfectants destroy the infecting microbian colony, and prevent the further entrance of any toxins into the circulation and nerve centres. The principal has been shown experimentally successful in cases of inoculation in the tails of cats and Guinea-pigs, and the ampu-

tation of these members as soon as tetanic symptoms appear (Kitasato, Babes). Unfortunately in too many cases, when first seen, too much of the marvelously potent toxin has already reached the nerve centres, and these have already undergone such changes, that the disease is likely to go on to a fatal issue in spite of the cutting off of future supplies of toxin. Yet the principle is sound and proves helpful in proportion as it is applied nearer to the time of infection. The most thorough method is the amputation of the infected member, if like the tail or ear it can be excised without ruining the animal. In 1875, Barbillon had success in again amputating the tail, in a case of tetanus after docking. Next to this comes the excision of the wounded tissues, but this can too seldom be effectually and certainly accomplished, and we must fall back on caustics and antiseptics. The actual cautery if thoroughly applied may be trusted to destroy the bacillus along with the tissues, but most of the chemical escharotics unite with the albumen to form an impermeable film, which protects the tissues in the deeper part of the wound against the antiseptic action. Of the different antiseptic applications *carbolic acid* should be especially recommended as being not only antiseptic, but also an antidote to the toxins as shown below. It has the further advantage of acting as a local anæsthetic, and of not coagulating albumen. Creosote, creolin, lysol, or other antiseptic, may be used instead and should be applied thoroughly to all parts of the depth of the wound on a pledget of surgeon's cotton or through a tube. When agents so little destructive are employed they may be continuously applied to the sores for a length of time.

Recoveries have taken place after neurectomy, and after stretching the nerve going to the wounded part, the theory being to check the afferent (sensory) nervous current, and arrest the reflex spasms. The new irritation, however, caused by the surgical wound is to be dealt with, and may itself turn the balance against recovery.

Nervous derivation appears to have been beneficial in some advanced, or partially convalescent cases. One horse after 14 days' illness (Taffanel) and another after 21 days (Prud'homme), were castrated, bled freely and slowly recovered. Tisserand gives another case without mentioning the stage of the disease. A horse with advanced tetanus was taken to the seashore and shot.

He fell into deep water, swam ashore and made a recovery. But whatever virtue may be in elimination of the toxins by bleeding, in nervous derivation or in the shocking of an unbalanced nervous system, these can hardly be recommended as regular methods of treatment. Yet the older veterinary records contain many instances of alleged benefit from bleeding.

Internal treatment. The whole list of antispasmodics have been tried, with no very satisfactory result. Opium has been extensively employed in spite of its tendency to encrease constipation, and morphia given hypodermically has checked spasm and induced sleep. Hydrocyanic acid and potassium cyanide have shown a decided reducing action on the spasms with the same drawback of favoring constipation. Potassium and other bromides are useful in mild and chronic cases, and may be given in full doses in combination with chloral hydrate. Calabar bean and eserine have been given for their physiological action on the nerve centres, and recoveries have followed their use, but they have little effect on the spasms until the system has been saturated to the point of threatening collapse. Chloroform has the advantage that it can be easily given by inhalation, but while it may be pushed to the extent of temporarily checking the spasms, yet these return at once when the action of the drug is exhausted. Chloroform is always dangerous to a weak or exhausted heart and cannot be given for any great length of time continuously. It is, therefore, very unsatisfactory. Sulphonal and trional have similarly checked the spasms. Gelsemium has given good results in certain mild cases, but it must be pushed to the extent of coming just short of poisonous doses, and the fear of an overdose, together with its failure in severe cases, have prevented its general acceptance. The same end has been sought by the use of nauseating antispasmodic agents, as tartar emetic, tobacco, apomorphia, and lobelia, but, though useful in individual cases, these are on the whole no more successful than other agents. Phenacetin, antipyrin, acetanilid and cocaine have respectively received credit for some recoveries.

Chloral hydrate commends itself as being at once a most potent antispasmodic and hypnotic, and an antiseptic. It can, moreover, be conveniently given as a rectal injection, thus avoiding the irritation and excitement of administration by the mouth. Given

in this way too, it tends to relaxation of the bowels, instead of constipation. Carbolic acid which can be conveniently given by enemata has an anæsthetic action.

Antiseptic and Antidotal or *Antitoxin treatment* is more promising, yet it has failed to come up to the full measure of expectation, mainly because the nervous changes have already reached a stage which cannot be undone speedily or at all. Under this heading would come phenic acid (½ oz.) and probably chloral hydrate (1 oz.), already referred to, and the various compounds of iodine which may be here noted.

Iodine Terchloride. Iodine. Behring and Kitasato secured immunity of two months duration, by injecting the animal with a filtrate of a culture of tetanus bacillus, and then injecting at the same point 3cc., daily, for five days, of a 1 per cent solution of iodine terchloride. Roux and Chamberland had similar results by using iodine instead of the iodine terchloride, and maintained the full measure of immunity by repeating the inoculation every fortnight. Here it is evident that the action of the iodine is directly antitoxic or antidotal, when introduced along with the toxins and before they can reach the nerve centres.

Iodine of Potassium. In an experimental case of general tetanus in the dog, Babes had a recovery in ten days, by injecting subcutem 5cc. of Lugol's solution and thereafter for eight days 10 to 30cc. daily. This suggests the use of this agent along with phenic acid, or as an alternate, in any case in which phenol appears to be losing its effect by use. It may be used hypodermically, or in the drinking water or by rectal injection. It has an advantage over phenic acid in being actively diuretic and eliminating, while phenic acid has the recommendation that it tends to lower nervous excitability and moderate the reflex spasm. Theoretically the combination of the two agents, which do not mutually decompose each other, should give the best results.

These experiments have been often repeated showing clearly the antidotal action of the iodine compounds when mixed with the virus before inoculation, or injected with the virus into the seat of the wound. When employed later when the symptoms have developed, everything depends on the changes already accomplished in the nerve centres, and the severity and generalization of the spasms.

Serum Antitoxin Orrotherapy. Babes appears to have been the first (1889) to use the serum of animals (rat) recovered from tetanus in mitigating and curing tetanus in experimental cases. An attempt on a well developed case in man failed. His method of preparing the serum is as follows: A horse of 900 lbs. (461 kgm.), is inoculated with a mixture composed of 0.5cc. toxin (of which 0.001 mgm. kills a mouse) and 0.5cc. iodo-potassium iodide, and then at intervals of four or five days of 2.5cc., 4, 5 and 10cc. of the iodine mixture. Then stronger mixtures are used: first 2 parts of toxin to 1 part of the iodine mixture; dose 10cc.; then 3:1 dose 10cc.; then 4:1 dose 5cc.; then 15:1 dose 10cc.; then 30:1 dose 25cc.; and finally virulent cultures in progressively encreasing doses 10, 20, 30 and 50cc. One week after the last injection 1cc. of the blood serum will antidote 50cc. of toxin. Injection of toxin is however continued and the dose is gradually raised to 200cc. Eight or fourteen days after the last injection the blood serum may be taken for protective purposes. He has prepared antitoxin from cows in the same way, and Brieger and Ehrlich have prepared it from the goat. Chickens being naturally refractory to tetanus toxin can bear large doses and a potent antitoxin is more speedily secured from them. In the case of the cow the milk is rich in antitoxin.

Other methods of preparing an animal for producing the antitoxin have been resorted to, as injecting it with a mixture of toxin and antitoxin in increasing doses, or again injecting it with a mixture of toxin and thyroid extract in increasing doses. The extract of the normal thyroid contains a natural antitoxin.

It may be fairly inferred that the antitoxin is not formed in the nerve cells alone in their resistance to the toxin, but also in the thyroid, the liver (bile having an antitoxic action), and perhaps in other organs or liquids.

The blood of the immunized animal drawn through the sterilized cannula and aspirating syringe is coagulated in vessels set on ice, and the serum when separated is mixed with 0.5 per cent. carbolic acid and 1 per cent chloroform, and kept in the dark in well closed bottles. It will usually keep for years.

Early experiments with antitoxins showed that when mixed with the toxin before injection it could be trusted to neutralize it. Ehrlich, Tizzoni and Cattani even claimed success in all ex-

perimental subjects if employed as soon as the sligtest symptoms of tetanus were shown. They found, however, that it required 1000 to 2000 times the amount of antitoxin in such cases than was required when it was mixed with the toxin prior to injection. They found, moreover, that when the disease is fully developed the dose must be 150 times more than is required when the first symptoms are shown. It should be added that when the disease has developed rapidly, after a short incubation, and is well advanced the antitoxin treatment is usually of little avail. The changes in the nerve centres are already too great to allow hope of recovery. In man the ratio of recoveries are about as follows: After incubation of 10 days or under, 3 to 4.5 per cent. recover. After an incubation of 11 to 15 days 50 per cent. recover (Woodhead). Lambert claims 46 per cent. recoveries in 114 cases, and 38.71 per cent. recoveries in acute cases with an incubation of 8 days and under.

On the whole the ratio of recoveries is greater under the antitoxin treatment than before, though far from sustaining the optimistic views of Behring and other early experimenters. Babes draws attention to the fact that spore laden splinters of wood, in the wound render the antitoxin useless as a continuous succession of fresh spores, bacilli and toxins are thereby supplied. As this is one of the most common forms of casual infection it interferes seriously with the success of antitoxin treatment.

Roux and Borrel found that in animals, intracranial injection of the antitoxin was the most effective method. Recovery also followed its injection into the cerebrum of a tetanic boy. Babes had recoveries in two cases out of three with cerebral or intracranial injections. As the reflex spasms depend on the spinal centres these would seem to be the ideal points of injection.

Babes who has done a large amount of subcutaneous and intraperitoneal injection of antitoxin, employed for man doses of 300 to 500 cc., which would represent 8 to 10 ozs. as the dose for an ordinary horse. As the antitoxin is rapidly eliminated from the body, these should be repeated daily or every other day. Nor should this supersede other curative measures. The leading principles may be thus stated: 1st. Antitoxin should be used at the earliest possible moment. 2nd. The infected wound area must be thoroughly disinfected or destroyed by caustics, and that at

once. 3rd. The antidotal treatment by phenol and iodine must not be omitted. 4th. Palliative treatment by antispasmodics, narcotics or soporifics must go hand in hand with antitoxic treatment. 5th. Measures should be taken to secure elimination of the toxins present in the blood.

Treatment by brain emulsion. Wassermann and Takaki (Berlin Klin. Woch. Jan. 3d, 1898) have in a number of cases, mixed 1 cc. of brain substance of a warm-blooded animal with ten times the lethal dose of tetanus toxin and injected without producing any symptoms of tetanus. They obtained a similar immunity by injecting the brain emulsion 24 hours after the injection of three times, and in other cases of five times the lethal dose of tetanus toxin. Control cases uniformly died of tetanus. The brain matter was obtained from Guinea-pigs, pigeons, rabbits, horses, and men. They accordingly advanced the theory that brain matter is a direct antidote to the tetanus toxin, uniting with it chemically and rendering it innocuous. The liver, spleen, kidney, bone marrow and blood serum gave no such protection.

Marie, in a series of experiments, injected the brain emulsion and tetanus poison at different parts of the body of Guinea-pigs and found that fatal tetanus ensued. It would appear, therefore, that the brain emulsion acts by direct contact, and that it is only by its meeting and combining with the toxin before the latter reaches the spinal cord that tetanus can be prevented.

Roux and Borrel (Ann. de l'Instit. Pasteur, 1898) demonstrated this union between the poison and brain matter, by making an emulsion of the two, and putting in a centrifuge, which will separate the brain substance from the clear liquid. The fluid obtained in this way was shown by injection on the living animal to contain almost no toxin. Knorr and Blumenthal reached the same conclusion as to a chemical union with the brain matter which robbed the toxin of its toxicity.

Knorr, and Tizzoni, and Cattani and Morax showed indeed, that if the tetanus toxin is injected subdurally or into the surface layers of the cerebrum, it produces not tetanus, but a characteristic cerebral disease. A dose of $\frac{1}{20}$th or $\frac{1}{10}$th cc. of tetanus toxin produces in the rabbit, in 10 to 12 hours, restlessness, constant change of place, and signs of great fear like hiding the head, turning rapidly round, attempting to escape, polyuria, grinding

the teeth, epileptoid convulsions. The toxin in this case had manifestly united with the brain substance while the cord suffered little.

Metchnikoff (Ann. de l'Inst. Past., April, 1898) holds that the brain matter is only valuable in holding the toxin until it can be destroyed by the leucocytes. He showed that the injection of the tetanus toxin in chickens or Guinea pigs greatly encreased the production of leucocytes. He injected tetanus toxin into the aqueous humor of the rabbit without producing much effect, but when the same agent mixed with cerebral substance was injected, the result was a great accumulation of leucocytes, and hypopion. If the mixture of brain substance and tetanus toxins were injected on the brain, little encrease of leucocytes occurred, but if thrown into the peritoneum, a most remarkable leucocytosis took place. In twenty minutes after the injection the fluid withdrawn from the abdomen showed large numbers of leucocytes filled with brain substance, but no free cerebral matter.

The present status of the treatment by brain substance is therefore somewhat uncertain. The value of that agent in holding the toxin is allowed, but like the antitoxin it must be employed before the toxin has reached the nerve centres and united with the living ganglion cells. Its use would be called for therefore at the earliest possible moment and it should be continued so long as there is reason to suspect the production of fresh toxin in the wound. Its direct action on the toxin would suggest its injection around an infected wound, or even as a dressing for the wound in connection with antiseptics. When tetanus has already set in it cannot be expected to undo the evil already accomplished by the union of the toxin with the cells of the cord, though it might in part arrest and hold new supplies of this poison coming from the wound to the nerve centres.

Prevention. In a disease so deadly as tetanus and so refractory to treatment even by antitoxin when it is once developed, prophylactic measures are of the greatest importance. With the extensive adoption of antiseptic surgery there has already been a material diminution in the number of cases, yet a greater attention is demanded to the prevention of casual cases which result from ordinary wounds. Dirty, grimy wounds filled with the dust of stable yard or garden soil, and such as contain splinters

of wood, stones, thorns, straw and the like can only be considered quite safe after thorough disinfection. It has been shown that the toxin is easily neutralized at the time of infection, whereas, after the disease is developed it will require 1,000 or 100,000 times as much antitoxin to produce the same effect. In the case of soiled wounds, therefore, in a valuable animal, a harmless injection of antitoxin or of phenic acid or lodine solution before the development of tetanic symptoms is not an unwise precaution. A succession of such injections might be given to ward off the disease until after a lapse of time exceeding the short and dangerous incubation.

Much more important is the disinfection of the wound itself. All foreign bodies must be removed, but especially those that like splinters of wood and straws are likely to harbor the spores of the bacillus. Then the wound may be thoroughly cauterized thermically or chemically, or it may be irrigated with a strong antiseptic solution and then dressed with some agent that will prove destructive to the spores, and antidotal to the toxin. Strong carbolic acid may be applied to the whole raw surface including the uttermost recesses of the wound, and after a few seconds or half a minute this may be neutralized by filling the wound with dilute acetic acid or alcohol, after which a dressing of Lugol's solution may be applied. Lambert advises a combination of hydrochloric and carbolic acids.

Weaker antiseptics, like a 5 per cent. solution of carbolic acid, do more harm than good, as they destroy the pus and saprophytic microbes and even the tetanus bacillus in the wound, without affecting the tetanus spores, which finding no other microbes to contest with them the possession of the field may find themselves in a better position than before to develop into bacilli and cause tetanus.

Tetanus neonatorum may be certainly prevented by the application of a disinfectant plaster on the navel at birth. Over 50 years ago in Scotland this desideratum was met by applying on the navel of the new-born child a soft and immaculately clean piece of cotton cloth which had just been flamed over a light. On the island of St. Kilda the former mortality of 67.2 per cent. of new-born infants was promptly arrested by dressing the navel daily with iodoform, For new-born animals a cheap and

convenient application may be made by incorporating 1 oz. powdered iodine and 2 lbs. wood tar, and smearing this on the navel.

Much may be done by disinfection of stables and yards where the victims of tetanus have been. The anærobic germ soon loses its virulence in free air and sunshine, and one has to dread especially, filthy stables, collections of manure, contaminated litter, wood, combs, brushes and buckets. In unpaved yards remove the infected surface soil and replace by fresh disinfected earth, or still better, well burned brick.

For horses which are necessarily exposed to manure or contaminated soil, it is commendable to wash the hoofs and pasterns on returning from work and then sponge with a weak solution (5 per cent.) of phenic acid. Another resort is to smear the hoofs daily with an ointment of tar and lard, equal parts. This cannot protect from infection by splinters of wood containing the spores, but is to a large extent preventive in the case of bacilli that might have been otherwise lodged on the surface and which could have been carried into the wounds inflicted by nails and other noninfected bodies. Careful shoeing is all important, to avoid the bruises, suppurating corns and *gravelling* which make openings for the ready entrance of the spore.

Roux and Nocard recommend immunization by protective inoculation. This is not only possible, but would be justified economically in the case of valuable animals, or in all animals in a district where the bacillus tetani is universally spread. The method is the same as advised above for the immunization of animals, for the production of antitoxin.

In districts where tetanus is rare, the cost of universal immunization against the disease would very far exceed the losses from casual cases. Under such conditions it would be an economical blunder.

FOOT AND MOUTH DISEASE.

Synonyms. Definition. Susceptible animals: cloven footed, all warm blooded animals. Historic notes; Geographical distribution; English invasions in 18th and 19th centuries; North and South American invasions in 1870, 1884, 1902; In Asia from immemorial times. Causes: infection in liquid of vesicles, saliva on pastures, roads, feeding and drinking places, halters, etc.; from feet on pastures, buildings, yards, roads, cars, boats, etc.; from teats through milk. Microbe not certainly known, micrococci, streptococci and bacilli found. Virus inert when dried 24 hours at 88° F.; survived 9 months at 32° F., attack immunizes for 5 months; injection of 1 lymph and 2 of blood of immune renders refractory; filtered lymph still virulent; microbe probably infinitesimal; accessory causes; movement, mingling of cattle, sheep, swine, etc., war, trade, common pasturage, infected roads, ships, yards, halters, etc. Symptoms: incubation 36 hours to 6 days; slight fever; redness, tenderness of buccal mucosa and teats, grinding teeth, smacking tongue, tender feet, shaking them backward, bullæ on mouth and teats, not nodular, nor chambered as in variola, salivation, bloody, circular or irregular raw sores, vesicles and erosions in interdigital space, shedding hoofs, sheep walk on knees, gangrenous mammitis; intestinal eruption and diarrhœa in sucklings. Mortality. Prognosis; recovery in 15 days, deaths rare if cared for. Losses from destruction of product and emaciation—occasional abortion. Diagnosis: based on infection of all exposed bisulcates, localization on mouth, teats and feet, inoculability on other warm blooded animals, unchambered vesicles, slight fever, and prompt recovery. Notes of affection in man. Symptoms in man. Prevention and treatment in man. Prevention in animals: exclusion of contagion, immediate and mediate; close infected pastures and roads, stop all movement of bisulcates, disinfect all boats, cars, places and things exposed, exclude visitors, guarantees with strange animals, quarantine and disinfect arrivals, exclude fresh animal products, fodder and litter, wash, disinfect soiled cloths. Inoculation undesirable. Treatment in animals: cleanliness, dryness, disinfection, segregate sick and well, gaseous antiseptics, liquid ointments. Gruels, mashes, sliced, boiled, or pulped roots. Local dressings for mouth, teats and feet. Evulsion of hoof. Mammitis.

Synonyms. Aphthous fever: Aphtha Epizoötica, Eczema Epizoötica.

Definition. An acute infectious disease of the lower animals but especially of ruminants, characterized by a slight fever and the eruption of vesicles, or bullæ on the skin and mucosæ, and usually those of the mouth, feet and teats.

Susceptible Animals. The animals that prove the most obnoxious to the disease are the bisulcates—large and small ruminants

and swine. Man however is susceptible as are also horses, hogs, cats and fowls, when they are inoculated or fed upon the infected milk or other products. It is doubtful if any warm-blooded animal enjoys an immunity.

History, Geographical Distribution. Toward the middle of the eighteenth century this disease prevailed in Central Europe and England. The latter country stamped out both this and the Rinderpest, but it continued to prevail on the continent and was re-imported into England in 1839. It reached America through an importation from England to Montreal in 1870, but owing to more or less effective quarantine, to the absence of cattle traffic from east to west, and above all to the prolonged confinement in yards and stables during our northern winter, it burnt itself out in the course of the year. Another outbreak from an unknown source and through an unknown channel, started probably in June, 1902, near the docks, Chelsea, Mass., was discovered by the authorities on Nov. 14th., and extended through shipments of cattle into Vt., N. H., and R. I. Unlike the invasion of 1870, this began at the terminus of cattle traffic, and failed to gain the alarming extension of the earlier plague, which had in succession swept into Quebec, Ontario, Central and Eastern New York, New Jersey, and every State in New England. Like that it broke out in autumn when the seclusion in winter quarters and the absence of birds as infection-bearers counteracted extension. Then in place of trusting to the mere arrest of cattle traffic, etc., as in 1870–1, the invasion of 1902 was met by vigorous slaughter of the affected herds, and indemnification of the owners. In all 4461 animals (cattle, sheep, swine, goats) were killed and the owners reimbersed to the amount of $128,908.57. The outbreak lasted eleven months, the stockmen having seen cases as early as June, 1902, and the last slaughters having been 3 herds of 59 cattle in May, 1903. The two outbreaks were equally virulent, equally contagious, and equally free from fatalaties. Each was a fair sample of the disease as constantly seen in Europe. The first was more extended because it first gained a status in Ontario and New York. Had the trend of cattle traffic been westward rather than eastward, in either case it would have swept the continent with the free headway at first allowed it.

Several importations at different times have been reported, in

which the owners secluded the herds for some months and then disinfected the premises with complete success. One occurred at Portland Me., in 1884, in which cattle, supposedly recovered, traversed the highway to the quarantine station, and a team of oxen which followed them the same day contracted the disease. The seclusion of both put an end to the trouble. In Asia it has prevailed from time immemorial, and it was imported into South America in 1870.

Etiology. This disease has long been known as caused by infection alone. Excluded from England in the middle of the eighteenth century it did not appear again until re-imported in the middle of the nineteenth, and then speedily overran the whole island except the breeding districts into which strange stock were never taken. In South America it was unknown until imported from the Old World into the Argentine Republic and then it made a wide extension and maintained itself where the stock was kept on unfenced ranches. In our fenced North-Eastern states it died out in 1871 and was stamped out in 1903,

The infection is especially resident in the vesicles or aphthæ. From the mouth this is distributed, with the abundant drivelling saliva, on pastures, roads, feeding and drinking troughs, ponds, streams and halters, and readily communicates the disease to healthy stock following in the same places. From the feet and especially the interdigital space, it is left on the vegetation, buildings, yards, cars, boats and all other possible media to infect other stock in turn. From the teats it mingles with the milk so as to infect the young suckling and all animals and men to whom the milk may be given. It may become dried on litter and other light objects and carried by the winds, or it may be carried on the feet of men or animals including birds, but apart from this it is not readily diffused and oftentimes a broad highway may set a limit to its propagation.

The infecting microbe is not definitely known. Nosotti found a micrococcus in the lymph of the vesicle, which stained readily in aniline colors, was easily cultivated and pathogenic. Klein found a steptococcus which, similiarly tested, presented an equal claim to be the causative factor. Bassianus and Siegal found in the blood and tissues of a person who died of foot and mouth disease a small oval bacillus, which they later obtained from the

vesicles of three children who were suffering from the disease, and from animals attacked in two successive epizoötics. With this they first successfully inoculated a calf and from the pure cultures obtained from its blood, inoculated three calves and a young pig.

Löffler and Frosch, the recent commission on foot and mouth disease in Germany, report that no organisms could be seen nor cultivated from the lymph found in recent bullæ of the buccal mucosa, though this lymph later passing through a Berkefield filter still proved virulent when inoculated on calves. A Pasteur filter arrests it.

They found that the lymph became inert when dried for 24 hours at 31° C. (88° F.), while it retained its vitality and virulence after exposure for 9 months to a temperature of 0° C. They concluded that it could not penetrate through the unbroken skin nor mucosa, and that it was most effective when injected into the blood or peritoneal cavity. One attack conferred immunity for 5 months. Blood from immune animals, injected into susceptible ones does not confer immunity, but 75 per cent. could be rendered immune if injected with a mixture of the lymph from the vesicle and double the same amount of the blood from the immune animal. Animals so treated become immune to 100 times the infecting dose. Filtered lymph was still virulent and the commission suggests that the microbe may be so small as to pass through the filters and escape discovery by the most powerful lenses. An object one-fifth the size of the smallest known bacillus—that of influenza—would be invisible under our best microscope.

By actual experiment the virus has been found in the nose, larynx, bronchia, stomach and intestines, but into all these the virulent lymph of the bullæ can find its way. In the intestines, indeed, in cases caused by feeding, bullæ have been found on the mucosa.

A most important question would be that of the virulence of the milk, but inasmuch as the vesicles appear on the teats and even on the openings of the milk ducts, and in bursting discharge their contents with the milk into the pail, the milk becomes per force infecting. The experience of Hertwig and his students who infected themselves by drinking the warm milk by way of experi-

ment, has been often repeated unwittingly by unwilling victims, and the many cases of calves, pigs and chickens contracting the disease by consuming the otherwise discarded milk leaves no room for doubt that this product is often infecting.

Among conditions contributing to a spread of infection, nothing is more potent than a free movement of ruminants and swine, whether determined by war, trade, or the intermingling of different herds on commons or unfenced ranges. In infected countries, in which cattle are distributed through large central markets there is always a wide extension after one of these fairs, the infection being narrowly circumscribed to herds receiving cattle from the fair, or those that have travelled on the same roads or fields after the market cattle. It has repeatedly happened that cattle shipped from the United States, where this disease has long been unknown, have been found diseased on their arrival at a British port, simply because they have been tied upon the passage with halters formerly used on infected Irish or Continental stock.

Symptoms in animals. There is first a period of incubation shorter in hot than in cold weather and varying from 36 hours to 6 days (exceptionally 15 days). It is altogether probable that prolonged incubation is really delayed infection, the virus having been attached to the feet for some time before it entered the tissues. Cattle usually show the disease two days after exposure in a public market, building, or conveyance.

There is first moderate hyperthermia (102° to 103° F.), indicated by the clinical thermometer before there is any outward sign of ill health. There may be erection of the hair, tremors or distinct shivering, dryness and heat of the muzzle, redness and even tenderness of the buccal mucosa and teats, saliva drivels from the mouth or may show as a frothy mass at the commissures or margins of the lips, and there may be grinding of the teeth and a peculiar smacking of the tongue and hard palate which may be heard at a considerable distance. There are greatly impaired appetite and rumination. Tenderness of the feet is shown by halting or lameness and by the extension backward and shaking of the hind feet in turn.

With the appearance of the eruption, usually on the second day of illness, the fever as a rule moderates, and on examination of the mouth, bullæ of ⅓d. to 1 inch in diameter may be found on the

inside of the lips, and cheeks, or on the palate and tongue, with, in many cases, a congested areola, but showing no nodule as in variola. These bullæ may extend to the muzzle, pituitary membrane or pharynx. They burst very soon after their formation, exposing a red base of inflamed corium, with a clearly rounded margin, or, at first, with shreds of the torn epithelial covering. The salivation now becomes more profuse, glairy and even bloody, and there is more active movement of the tongue. When the bullæ have been confluent there are formed extensive red patches denuded of epithelium, and the suffering causes a complete but temporary dysphagia. The renewal of the epithelium, however, takes place promptly and may be well advanced in four or five days. Upon the teats the bullæ appear at about the same time but are usually smaller than the buccal, and do not show the thickened base of cow pox. They burst in 36 to 48 hours unless broken earlier by the hands of the milker, forming sores comparable to those of the mouth, which are liable to be kept up by the necessary manipulations in milking.

Upon the feet the eruption shows especially in the interdigital space, at first as vesicles smaller than those of the mouth and teats, leaving erosions and ulcers which extend under the adjacent horn, and upward on the front and back of the pastern. From exposure to mud and filth these are liable to be kept up even longer than those of the mouth and teats, and under neglect the entire hoof is often shed. In sheep and swine the disease may be localized almost exclusively in the feet. Sheep will even walk on the knees.

In young animals and those fed on the milk, the eruption may take place on the intestinal mucosa with violent congestion, diarrhœa and a fatal issue. Aggravated cases may show gangrenous mammitis or abortions.

Mortality and Prognosis. While there are seasons of special pathogenic severity, yet as a rule, the foot and mouth disease is a mild affection and unless neglected, the patients entirely recover in about fifteen days. The pecuniary loss in dairy and feeding cattle has been found to average in Great Britain about $10 per head, and as few animals escape, the consequences are usually very serious. In England the losses from this disease in 1883 reached $5,000,000, in France, those of 1871 were $7,500,000

and in Switzerland, $2,500,000. In Germany, over 7,000,000 animals suffered from 1889–94.

Differential Diagnosis. While a mistake might be made in an isolated case, such a thing should be absolutely impossible where cattle and other animals are collected in herds. The rapid infection of the whole herd, the implication of sheep and swine along with the cattle, and the eruption of the characteristic bullæ on the mouth, feet and udder or on two of these locations to the exclusion of the rest of the body, is not likely to be counterfeited by another disease. An outbreak of gangrenous ergotism in Kansas, Missouri and Illinois in the spring of 1884, was pronounced to be foot and mouth disease by a number of veterinarians, including an expert sent by the Government of Canada. On behalf of the U. S. Treasury I investigated the disease, which caused in many cases sores on the mouths and feet, but it spared all sheep and swine, could not be conveyed to them nor to new born calves by inoculation, and in many cases it caused gangrene of all the tissues, soft and hard, and separation of the limb at a given point, often near the tarsus. The quarantines were raised, the disease made no further extension, and the existing panic subsided.

Infection of Man. The first authentic record of this affection in man we owe to Valentin, who records that during the outbreak in Hesse in 1695 men suffered from inflammation of the gums, tongue and mouth. Michel Sagar says, that in 1764 men who drank the milk were affected with aphtha. In 1828 it was conveyed from animals to men in Bohemia (Nadberny), in Styria (Levitsky) and Wurtemberg (Kolb). In 1834, three veterinarians, Hertwig, Mann and Villain, voluntarily drank a quart each of the warm milk of a cow suffering from this affection. On the second day Hertwig suffered from fever, headache and itching of the hands and fingers. Five days later bullæ formed on the hands and fingers, the tongue, cheeks and lips. In the two others the eruption was confined to the buccal mucosa. Since that time, records of the infection of human beings have been very numerous. During the American epizoötic of 1870 I met with the case of a farmer at South Dover, N. Y., who suffered from sore mouth and blisters along the margin of the tongue from drinking the milk. The danger is greatest in chil-

dren on an exclusive milk diet and who drink it warm. Kolb in 1828, noticed acid vomiting and diarrhœa in such subjects. Hübner observed that beside the buccal eruption such children often suffered from inflammation of the stomach and bowels and that very young children fed on the milk of the diseased cows died. Balfour, Watson and others have noticed similar results in Scotland.

Allbutt saw the buccal eruption in three children in Yorkshire, England, during the local prevalence of the English epizootic in 1883, and secured information of a number of other cases in the same district.

A number of cases were recorded during 1893 in Germany. A shepherd infected himself by holding in his mouth the knife with which he had pared the diseased feet of sheep, and another workman and a veterinarian had extensive eruptions on the hands after dressing the affected feet. A number of milk-maids were infected by milking, the eruption appearing on the hands, and in one case on the breast. A child fed on the milk of diseased cows, had chill and fever with gastric disturbance, and later an eruption of vesicles on the lips and tongue and between the fingers and toes.

Again, in 1895, during the prevalence of foot and mouth disease in the southern part of Berlin, a considerable number of the milk consumers suffered from fever with the eruption of bullæ on the tongue and buccal mucosa generally, which on early bursting left very painful ulcerations. The acute disease did not last more than five days, but left a sense of great weakness for a time. Virchow, who made an investigation, unhesitatingly pronounced it to be foot and mouth disease.

Cases of infection through butter made from infected milk are on record. A Berlin veterinary student suffered from the buccal eruption and erysipelatoid swelling of the ear, and a German clergyman had in addition a period of chilliness, fever, diarrhœa and pruritis. Similarly Schneider quotes cases determined by infected cheese, and Friedberger and Fröhner, cases caused by virulent buttermilk.

Symptoms in Man. In man there is observed the tendency to localization on the same points as in animals. As the hands are naturally exposed to infection by milking or treating the diseased animals, they are especially obnoxious to the eruption, and the

same is true of the mouth when the infected milk or other dairy products are consumed. The bullæ on the buccal mucosa are generally confluent, and often extend to the fauces and pharynx, rendering speech difficult and swallowing painful, and leaving extensive and painful sores which, however, soon heal up. In women the bullæ have been seen around the congested nipples, and in exceptional cases they have been generally diffused over the body.

In cases due to drinking the milk, the early febrile symptoms are liable to be accompanied or followed by nausea, anorexia, abdominal pain and diarrhœa, and still later by the cutaneous and buccal eruption.

The duration of the disease is from 10 to 15 days and as a rule no permanent scars are left on the skin or mucous membranes.

The *diagnosis* is assisted by the knowledge of the prevalence of the disease in herds in the district, and that the patient has handled the diseased animals, or drunk their milk, or eaten their butter or cheese products. The predilection of the eruption for the fingers, the roots of the nails and the mouth is very significant. The disease follows an acute course and convalescence is complete in ten or fifteen days, which serves to differentiate it from most skin eruptions. From variola which pursues an equally rapid course it is distinguished by the absence of the primary nodular swelling, and of the septa or pillars that divide the mature pock into independent chambers.

Prophylactics. The best prevention to man is to exclude the disease from the country and its herds as is now the case in the United States. When the disease does exist in herds the attendants should cauterize any sores on the hands, and wash the hands with an antiseptic, such as a 10 per cent. carbolic acid solution, after handling the diseased. The milk and its manufactured products—butter and cheese—should be withheld from consumption until after the herd has recovered. Infection can be obviated by boiling the milk.

Treatment. The disease follows a rapid course and is self-limiting, and usually benign so that active treatment is not urgently demanded. The local lesions are best met by non-poisonous antiseptics, such as: borax in powder or strong solution; boric acid (4:100); sodium hyposulphite (½ oz. 1 qt.); chlorate

of potash (½ oz. to 1 qt.); salicylic acid (1:100); or salicylate of soda. Pounded ice may be used as a soothing agent. The cutaneous lesions may be wrapped in cloth wet with one or other of the antiseptic lotions. Any disposition to ulcerate may be met by the stick of silver nitrate.

Slightly laxative or diuretic agents may be employed for their febrifuge and eliminating properties and the food should be light, easily digestible and given cold.

Prevention in Animals. When the disease exists in a country or district this includes all measures preventive of immediate or mediate contagion. Arrest of all movement of cloven footed animals in infected districts, disinfection of cars, boats and other conveyances, of markets, yards, highways, seclusion of infected herds and pastures, exclusion of visitors, disinfection of products, certificates of soundness of origin, thorough disinfection after recovery of the herd, such are the leading points to attend to. Inspection or closure of fairs and markets is desirable and any exposure of diseased or infected animals should be visited with heavy penalty, in addition to the cost of detention and supervision. For non-infected country a certificate and guarantee of non-exposure with each cloven-footed animal imported, and of thorough disinfection of the cars, boats, halters, or other objects used upon them, and of the places, fodder and litter supplied, together with quarantine (1 week) and surface disinfection should be required under penalty. Exclusion of fresh hides, bones, guts, hair, bristles, wool, horn, as well as of fodder and litter is essential. Cattle attendants, drivers and others whose clothes are soiled with the products of the barn, should have the same washed and disinfected.

Inoculation has been proposed, and even practiced to pass a whole herd promptly through the malady, but as immunity lasts but three months, and the attendant risks to other herds are greatly encreased it is at once an economic blunder, and a great injury to adjoining owners. Any resulting extension to other herds should be an occasion for a verdict for damages at common law.

Treatment in Animals. Provision is first made against extension of the infection. The floor should be kept clean, dry and covered with sawdust, tan bark, gypsum or litter sprinkled

with these or with phenic acid. The herd should be divided into two lots—the apparently sound, and unsound kept strictly apart under separate attendants, above all separate milkers. As soon as any symptoms are shown by an animal in the sound enclosure it must be instantly transferred to the other and its stall disinfected. Antiseptics such as gaseous iodine (two tablespoonful of tincture of iodine, thrown into a quart of boiling water twice daily), sulphurous acid, salicylic acid, creolin, lysol or other ointment on the feet and teats, may also be used. In this way it may be possible to save a number from an attack, yet most commonly the exposure is common and universal and the malady develops in all simultaneously. For those already attacked, gruels, mashes, and cool pulped, finely sliced or boiled roots may be all that is required, the disease runs its course and recovery ensues in 15 days. As local dressings the following may serve as examples : for the mouth, borax, chlorate of potash, salicylate or sulphite of soda 2 drs. to 1 quart water ; phenic acid, creolin, or lysol, one or two teaspoonfuls to a quart ; for the foot, clean the interdigital space and apply tar and carbolic acid with bandage, or use solutions of creolin, lysol, pyoktanin or blue-stone ; in aggravated cases strong mineral acids with tar ; for the teats, ointments of boric or salicylic acid, creolin, lysol, naphthalin or napthol. Separation of the hoof or mammitis will require treatment according to indications. Mercuric chloride subcutem has been lately adopted to cut short the disease.

MILK SICKNESS. "THE TREMBLES."

Geographical distribution : timbered lands in the United States ; different altitudes, and geological formations ; on hills and wooded bottoms ; known to Indians and pioneers ; now unknown where formerly prevailed. Contagion : through milk ; no specific microbe found in every case. Alleged causes : rhus ; nickel ; spirillum ; bacillus. Prevails in dry seasons ; contracted under night exposure ; confined to given enclosures ; to late summer and autumn. Not conveyed by contagion, indefinitely, as are plagues. Men show very varying susceptibility ; young children may be relatively immune. Fatigue, debility, ill health, predispose. Exertion to fatigue rouses symptoms in animal affected. Cow in full milk eliminates toxins and does not show symptoms ; the milk infects. Steers, bulls and heifers, show marked symptoms Calves suffer through milk ; swine through veal ; dogs through pork ; buzzard through dead dog. Incubation 8 to 12 days. Symptoms : tardy, lazy gait, drooping, anorexia, ardent thirst, inactive bowels and kidneys, milch cows when driven or excited, tremble and may suddenly die. Muscular debility, constant decubitus, complete apathy, neither evades nor resents injury. Bloodshot, fixed, glazed, unwinking eyes, pulse and breathing slow, temperature low, hebetude, torpor, coma ; death 8th to 10th day. Sheep very prostrate. Calves tremble when sucking, vomit and perhaps die suddenly. Pigs and dogs vomit, and show costiveness, remarkable debility and weariness. Man is weary, languorous, weak, apathetic, loathes food, is nauseated, retches. No fever ; but ardent thirst, tremulous tongue, mawkish breath, soft flappy belly, careless of own or family interests, forgetful of decency. Nausea, vomiting of blueish liquid, hebetude, inactive bowels, coma. Lesions : gastro-intestinal congestions ; ingesta like hard balls of saw-dust. Treatment : charcoal, mild laxatives, elm bark, egg-nog, potassium permanganate. Prevention : clear timber land, let in sunshine, cultivate. Insects. Sterilize the milk.

This is an infectious disease which has been found enzootic in certain unimproved, timbered lands of North Carolina, Georgia, Tennessee, Kentucky, W. Pennsylvania, Ohio, Michigan, Indiana and Illinois. Beach says it has never been reported on any of the Western prairies, at any point west of the Mississippi River, in New England, in the Canadas, in any islands, or in any part of the Old World. Altitude appears to have no effect in its production, nor geological formation ; it has been found in the wooded mountains of the Blue Ridge of N. Carolina and Georgia (Kerr, Salmon, Phillips) ; in the hills of Pennsylvania and Kentucky ; (Beach, Phillips) ; on timbered uplands (Phillips) ; and on the wooded bottoms of the Scioto and Miami in Ohio (Phillips, Schmidt) ; in the timbered bottom lands of the

Wabash and White Rivers in Indiana (Phillips); and in the wooded bottoms (Beardsley), and Indian Grove in McLean, Co., Ill. (Beach). The constant conditions are the heavily timbered and virgin condition of the soil.

It was much more prevalent in the time of the early settlers, than it is to-day, many infecting localities having become salubrious in connection with the clearing away of the forests and cultivation of the soil. The disease was well known to the Indians and often proved disastrous to the pioneers, whole communities being swept off as recorded of Pigeon Creek, by Nicolay and Hay in their History of Abraham Lincoln. According to these writers it was "a malignant form of fever,—attributed variously to malaria, and to the eating of poisonous herbs by the cattle—attacking cattle as well as human beings, attended with violent retching and a burning sensation in the stomach, and often terminating fatally on the third day." Even in these early days settlers were loathe to acknowledge the existence of the infection on their lands, doubtless because it depreciated them, and to-day with a better knowledge of the necessary precautionary measures, it has literally disappeared in many places, so that it is now difficult to find a case.

Contagion. That the disease has been transmitted through the milk from animals to man and other animals has been too painfully evident from the first, but no specific microörganism has been found to be constantly present, capable of pure culture in artificial media and of causing the disease when transferred from such media to a new victim. Naturally all sorts of theories have been advanced, no one of which has been demonstrably proved. It has been attributed to eating of poison ivy (Rhus toxicodendron) by the cattle, as this plant was usually found on the infecting lands, but rhus is also common throughout New England and the Eastern States where milk sickness is unknown. It has been claimed that it was due to mineral agents, especially nickel, in the water but the mineral salts in the water are not removed by culture of the surface soil, which puts an end to milk sickness. Phillips (1876) claimed to have found the cause in an actively motile spirillum in the blood, but he had examined the blood of but one patient, and it has not been found in other patients by subsequent observers. Bitting found a bacillus but further research has not determined its constancy.

Beach furnishes a series of observations which should be useful in seeking to estimate the value of any theory propounded. 1st. Milk sickness is a disease of dry seasons. 2d. In unusually dry seasons it is dangerous to leave domestic animals out over night in the localities where the disease is prevalent. 3d. It has never been considered dangerous for animals to pasture on such lands in the day time. 4th. Cattle in one field will habitually escape, and in another with apparently exactly the same conditions and the same flora they are attacked. 5th. The disease is unknown on the open prairies of the Western States, where the domestic animals are not allowed to remain over night in the timber belts. 6th. With occasional exceptions, it is a disease of late summer and autumn. The dangerous lots can, as a rule, be safely depastured in winter and spring. 7th. The pioneers found that they could protect their stock by keeping them coralled on a "tame" piece of land from before nightfall until the fogs and dews became dissipated on the following morning.

For the land to become "tame" it was only considered necessary to cut off the timber and let the sunshine act freely on the surface. Plowing and cultivation did not seem to be requisite in all cases.

A great drawback to research is the difficulty of securing cases to study. Many lots, formerly dangerous, are no longer so, and others still infecting are kept so secluded that casual cases cannot be found, without much expense for experimental animals. Again, owners do not care to depreciate their land by acknowledging that it is infecting. The experiment stations naturally enough look askance, on the proposal to institute expensive experiments on a disease which dies out when the soil is improved. Deadly as the disease is to the individual attacked (man or beast), it is not propagated indefinitely from non-milking subjects, by simple contact or proximity after the manner of plagues. It usually comes to an end by the death or recovery of the subject that has contracted it by consuming meat, milk, butter or cheese, the product of an infected animal. The demand for sanitary police measures is, therefore, less urgent. Different observers claim that cases occur in the large cities, through the consumption of meat, butter or cheese, sent from infected localities, but that the city physician fails to make a correct diagnosis. These must, however, be comparatively rare.

In addition to ingestion as a cause, certain accessory causes ought to be noted. Some men eat the infecting material with impunity, while others succumb to the deadly disease. As the observations have all been made in or near the infecting localities, individuals may be immune through a previous attack and recovery, or there may be a native immunity through unknown conditions. Young children often suffer less than adults, possibly because of the greater activity of their emunctories and consequent elimination of the toxic products and the comparative absence of exhausting or depressing conditions. Their purely animal food (milk) may exercise an influence, and this may assist in explaining the fact that certain adults appear to be refractory.

Fatigue, debility and ill-health are said to predispose the system. Milk sickness attacks most violently those that have been subjected to overwork or severe exertion of any kind, especially in hot weather, those suffering from want of sleep (sitting up with the sick), those having a special cause of mental depression, those suffering from some illness—constipation, indigestion, malaria, etc.

Milch cows are probably more open to the attacks of the germ because their systems are reduced by simultaneous milking and breeding through a number of years. Exertion or fatigue has a potent influence in developing the symptoms, so that it is a common practice in the vicinity of infected localities to subject animals to a good run before purchasing. Paradoxically enough the infected milch cow which is distributing the infecting element freely in her dairy products usually shows, in herself, no distinct symptoms of the disease. If she is dry or farrow she suffers like any other animal, but if in full milk, the toxins, and even the hypothetical microbe, seem to escape in that secretion, which proves highly poisonous to other animals, while the cow retains her spirits, vigor and outward appearance of health. Steers, bulls, and heifers, on the other hand, show violent symptoms.

Calves suffer so long as they suck the milk. The dead calf is eaten by swine, which suffer in their turn, and the dog contracts the disease by eating one of these animals, or by taking infected milk or cheese. The buzzard eats the dead dog and dies as the result.

Incubation is from 8 to 12 days, though it may be reduced to two. (Beach).

Symptoms. In the domestic animals the first indication of illness is a lazy, tardy disposition. The subject stands apart from the herd, with drooping head and ears, listless, indifferent to all around him, and often without appetite; or, in cattle or sheep, rumination. There is usually extreme thirst, but without correspondingly free urination or defecation. Peristalsis is virtually abolished and nothing whatever passes from the bowels. The patient is likely to be found lying down and it is difficult to get him up, and when raised he moves stiffly and with reluctance.

In milking cows there may be no symptoms until the animal is excited or fatigued by violent or continued exercise, as a hard run, or a drive of four or five miles. This develops the tremors alike in milch cows and in the mild cases in dry cattle or sheep. The subject stands still and trembles in a striking way, the action resembling the muscular contractions seen after the removal of the hide in an animal newly killed. The head and ears are drooped, movements are uncertain and stiff, and the animal may even drop dead on the spot.

As the disease advances the muscular debility becomes so great that the animal lies down if possible, and if once down he seldom rises again. The decubitus is extended, the head being stretched on the ground. There is a most complete apathy, the subject showing no fear, no apprehension, no disposition to escape or resent injury. The wildest or most timid steer can be freely handled, and there is no disposition to flight or retaliation. The eyes are bloodshot and become fixed and glazed, winking ceases, the breathing is slow, pulse infrequent, and temperature often sub-normal. The extremities and surface of the body are cool, the muzzle dry, the coat usually stares, the apathy merges into a complete hebetude, torpor and coma, in which condition the animal often dies on the eighth or tenth day. Violent exercise precipitates the death at once. Recoveries are infrequent and attended by no critical discharge from bowels or kidneys, only by a slow, at first almost imperceptible, resumption of natural action.

The milder cases, those that show no appreciable symptoms when at rest, are seized with trembling or rigor when made to undergo the slightest exertion; they appear haggard, stupid and

spiritless, drag their limbs slowly and stiffly and quickly stop from pure weariness and debility.

The prostration is even more marked in *sheep*, which often seem unable to rise, or lack the nervous energy to do so.

Calves tremble while sucking, and will sometimes leave the teat, vomit the contents of the stomach, fall down and perish.

In *Vomiting Animals* (*pig*, *dog*), emesis usually occurs, and torpor of the bowels or obstinate constipation is present. *Pigs* burrow under the litter and are driven out with difficulty, and *dogs* when called on to follow, do so reluctantly, slowly and stiffly and fail to keep pace with their master ("the Slows").

In all animals alike, active or continued exertion rouses or intensifies the symptoms.

In *man* there is at first extreme langour, weariness and weakness, the patient cannot be troubled to move, he loses appetite, loathes food, and soon has nausea and retching—often from the first. There is no chill, rigor nor violent headache as in other fevers; but insatiable thirst; large, flabby, tremulous, moist tongue, coated a dirty white; cold nose, ears and general surface; dry skin; sweet, mawkish or offensive breath; flat, flabby empty belly; without peristalsis or defecation. Respiration becomes very slow, pulse weak and compressible, heart action tumultuous and labored, temperature often below normal, and though sometimes 99° or 100° F., never higher. The patient takes to bed in four or five days, or, after exertion or fatigue, in a few hours becomes profoundly apathetic, expresses no concern for his business, his own future or that of his family, is intolerant of bed clothes or other covering and utterly oblivious of the demands of decency. Nausea continues, but retching becomes weaker, and comparatively ineffective, or brings up a little liquid which has been compared to blueing water of the laundry. The apathy merges into a state of hebetude and this into coma, with fixed, glazed eyes, absence of all winking, and insensibility to irritants and death takes place quietly without a moan or struggle.

Recovery is slow, and improvement for a time is almost imperceptible. In some cases there remains a nervous atony, and in man, a lack of mental and bodily vigor, and a disposition to relapse under exposure to intense heat or fatigue has been noted,

but in many cases recovery is complete and permanent without lasting weakness.

Lesions. Both in man and animals, congestions of the gastric and intestinal mucosæ have been noted, usually with a dark firm condition of the membrane, but in some cases with capillary stasis, and sloughing. Beach never saw indication of tenderness in the abdomen or elsewhere, nor did he ever find blood nor stercoraceous matter in the vomited material. The contents of stomach (paunch in cattle) and bowels formed hard balls like cemented sawdust, firmly adherent to the dry mucosa.

Treatment and Prevention. Treatment by the Indians consisted in giving large doses of powdered charcoal suspended in milk. The early physicians attempted to open the bowels by calomel and jalap, olive oil, magnesia citrate, and even croton oil, but the last generally with fatal results. Milder and hardly less effective treatment consisted in large doses of elm bark. Beach believed he got better results with quinine and egg nog. It might be suggested to try such antiseptics as potassium permanganate, peristaltic stimulants like eserine or pilocarpine, as an eliminating agent pure water or weak diuretics, and nerve stimulants, nitroglycerine or ammoniacal preparations.

Prophylaxis. The time-honored resort of clearing the timber and brush land so as to let the sun act freely on the soil, and the putting in of cultivated crops, is proved reliable and permanent. The other precautions in use are valuable in protecting the herd, but lack the merit of thoroughness and permanence and thus fail to strike at the root of the trouble. They are: 1st the exclusion of domestic animals from the infected woods in late summer and autumn and in very dry seasons; and 2d. the exclusion of stock from such pastures from before nightfall until after the dews have evaporated on the following morning.

The danger which attends on passing the night in the forest, strongly suggests the intervention for the transfer of the poison of some nocturnal animal, perhaps a night-flying insect, like the anopheles, which transmits the plasmodium of malaria. If the germ and its intermediate bearer (if any) were demonstrated, probably other and simpler means of prevention could be adopted.

The fact that the propagation of the disease is not constant and wide spreading, like a genuine plague, lessens the urgency for a rigid sanitary police, yet animals kept on such infected farms,

should be tested by long or vigorous driving before they are killed for food, and all milk devoted to the production of butter and cheese should be Pasteurized or sterilized before use. It might well be questioned whether the clearing and exposure of infecting places should not be undertaken by the state as a sanitary measure.

In view of the fact that a milch cow may not show symptoms of the disease, and yet yield deadly milk, and considering that the owner cannot always tell whether she has been in the infecting woods, or having been in, whether she is infected, it becomes an important public health question whether such a source of deadly disease should be perpetuated, where human food is open to contamination.

VARIOLA : POX.

Definition: Structure of lesion. Nomenclature History, smallpox, sheeppox, cowpox, horsepox Animals susceptible : Man, sheep, cow, goat, horse, pig, dog, buffalo, camel, monkey. Microbiology : A pure contagium, particulate contagium, cocci, sporidium vaccinale. *Horsepox* : Early history, means of infection. Symptoms : Vesicles on lips, on heels, concretions, treatment. *Cowpox :* Relation to horsepox. Causes. Relation to smallpox. Observations of Ceeley, Fletcher, Thiele, Klein, Martin, Reiter, Chauveau, influence of vaccination, of spring parturitions, of infected stables. Symptoms : Incubation, seat and nature of vesicle, inoculations for vaccine. Diagnosis from aphthous fever, rinderpest, eruption of mast-feeding, false cowpox, streptococcus eruption. Duration. Course. Prognosis. Treatment. *Sheeppox :* Synonyms. Definition. Pathogenesis : Sheep, goat, ox, dog, pig, horse Forms : Discrete, confluent, hæmorrhagic. Distribution. Causes. Contagion ; extends on air ; experiments on blood ; wool, hides, litter, buildings, yard, parks, railway cars, boats, clothes, manure, urine, milk, men, dogs, cats, birds, vermin, flies. Receptivity. Overcrowding, filth, starvation, neglect, wars, commerce. Recovered sheep. Disinfectants. Incubation 4 to 8 days ; conditions affecting. Symptoms : Hyperthermia, general disorders, rigors, anorexia, skin blush on parts devoid of wool, red points, papules, vesicles, pustules, desiccation. Successive crops. On eye, nasal mucosa, mouth, pharynx, intestines, lungs. Confluent cases. Lesions. Prognosis. Mortality. Depreciation. Treatment. Prevention : By segregation, slaughter and disinfection ; by ovination. Technique of ovination. Resulting immunity. *Sheep-pox in Goat :* Danger of infection to sheep. *Goatpox. Swinepox :* From man, sheep, goat. Symptoms. Forms : Discrete, confluent. Susceptibility of young. Treatment. Prevention. *Dogpox :* From man ; from sheep. Other eruptions in dog. Symptoms : Fever, flushed skin, red points, papules, vesicles, pustules, Discrete. Confluent. Treatment. Prevention of infection of man and sheep.

By the generic name *Variola* is understood a febrile malady attended by a characteristic eruption on the skin, at first papular, then becoming vesicular and finally pustular. The structure of the vesicle is so characteristic that it may be taken to indicate the variolous eruption as found in man and a variety of the domestic animals. The first indication of the lesion is the appearance on the skin of fine points of congestion like fleabites. This is followed by active diapedesis and proliferation of cells in the papillary layer and rete mucosum, constituting the nodule or pimple stage. In smallpox this is so firm and definitely outlined that it has been compared to the presence of a shot in the skin. As the proliferation of cells increases these form in separate clusters or groups, isolated from each other by septa or walls largely made up of the epidermic cells. In the next stage, therefore, when exudation takes place the lymph accumulates in the spaces occupied by the clusters of growing cells, and is found in a series of chambers more or less perfectly separated from each other, so that to evacuate the whole vesicle, each minute sac must be punctured independently. The vesicles thus differ from others caused by ordinary irritants in that each is chambered, instead of forming one common undivided sac, which may be emptied by a single puncture. In the next stage, when suppuration ensues, the septa usually undergo liquefaction, so that the liquid occupies one undivided cavity in each pustule. For this reason the central depression seen in the larger vesicles (cowpox) in their early stage tends to disappear in the pustule. It may reappear later in the resulting scab. Desiccation, scabbing and desquamation complete the course of the affection, a distinct *pit* being left as a result of the destruction of the superficial layer of the dermis.

Nomenclature. The term *variola* is believed to come from the Latin varius (variegated, spotted) and *pox* from the Saxon pock (pouch). The specific names, drawn from these tongues sustain this view: As, *variola vaccinæ*, *cow-pox*, *kine-pox*; *variola equinæ*, *horse-pox*; *variola ovinæ*, *sheep-pox*; *variola caprinæ*, *goat-pox*; *variola suilla*, *swine-pox*; *variola caninæ*, *dog-pox*. The term *small-pox*, (*petite verole*) is deduced from the small size of the vesicle as compared with that of cow-pox, just as the same has originated the term *smallpox* in sheep.

History. Variola has undoubtedly existed from very ancient times. Moore found it referred to in Chinese records of 1122

years before Christ, but it was only clearly described early in the tenth century by Rhazes an Arabian physician. Gregory, however, found the name *variola* in Latin manuscripts in the British Museum of a much earlier date. The early epidemics of small pox have usually extended from the east, and the disposition has been to refer its origin to the crowded communities of central Asia, but nothing is certainly known as to such origin and the lack of definite recognition and description cannot be taken as implying that the disease did not exist. The extension of small pox to America in 1520 was distinctly traced to a sick negro slave landed in Mexico, and the way in which it swept the continent killing the Indians by tens of thousands, speaks strongly for its prior absence and the extraordinary susceptibility of the hitherto unaffected Indian races.

The variolæ of animals are not recorded until later, the mildness of the forms attacking cattle and horses, and the lack of close observation of the diseases of sheep furnishing a reasonable explanation. We must pass over as uncertain the *lues ovium* of Thomas Wallsingham, (Historia Anglicana), imported in a *rotten* Spanish ewe in 1274, and which prevailed for 25 years destroying nearly all the sheep of the kingdom, also the reference to the "pockes" of sheep in Chaucer's "Pardoners' tale" as highly uncertain. Laurent Joubert in his work on the "*peste*" mentions *sheep pox* as prevailing in 1567, and Rabelais speaks of it as prevailing in France in 1578. It prevailed in Padua in 1649, in Venice in 1664, 1672, and 1674 (Bottani), in Italy in 1690, (Ramazini), in Germany in 1687–8 (Stegmen), in England in 1711, in Hungary in 1712, in France, Italy, etc., in 1714 (Kanold), in Venice and Bohemia in 1719 (Bottani), in Saxony in 1720, in Venice and France in 1723-24 (Bottania, Astruc), in Thuringia in 1725, in Siberia in 1771 (Pallas), and in Persia generally at the beginning of the 19th century (Bruce). Great Britian, long protected by her insular position, was infected by sheep from Germany in 1847 and again in 1862. Under ovination the first invasion prevailed for four years causing wide spread destruction; under strict separation based on thermometry, the second lasted but four months.

Cowpox has existed in England for centuries, but it has only attracted general attention since the introduction of vaccination by Jenner in 1796. *Horsepox* has existed concurrently with cow-

pox, the infection being habitually transferred by the hands of the milkers from horse to cow and vice versa. Jenner found it so common in the Valley of Gloucester, that he considered it as the habitual source of cowpox. Sacco recognized it at the beginning of the 19th century, Hertwig in Berlin in 1830, Röll in Vienna in 1855, and Bouley and others later in different parts of Europe.

Animals Susceptible. Variola in some form affects man, sheep, cattle, horses, pigs, goats, dogs, buffaloes, camels and monkeys.

Microbiology and Infection. It has long been well established that variola is due to contagion alone. The habitual dread of contact with a smallpox patient, shows the general appreciation of the danger of contagion, and the many epidemics, started by the introduction of a smallpox patient and thereafter spreading from that as a centre, together with the long continued immunity of certain insular or trans-oceanic countries illustrate this. One of the most striking examples is the immemorial immunity of the New World until the landing of the variolous slave in Mexico in 1520, and the immediate, rapid and destructive spread of the disease among the native tribes. Sheeppox offers a no less striking example. Prevailing for centuries in Asia and Europe, its extension to a new district was always the manifest result of the movement of infected sheep; England remained immune until her first invasion in 1847, and the second in 1862, in both cases the source was easily traced, and the disease completely extinguished by the destruction of the infection in its circumscribed area; the more distant sheep raising countries, America, North and South, Australia, Tasmania, New Zealand, South Africa, in the absence of importation of infected sheep remain free to the present time. For horsepox and cowpox the demonstration is more difficult, as limited outbreaks have occured at intervals in different localities, traceable more or less clearly to infection from vaccinated persons, yet often mistakenly attributed to spontaneous developments of the disease. Before the days of Jenner however it prevailed habitually in certain dairying districts (Gloucestershire), and I can point to localities in New York, in which the infection is manifestly laid up in the stables, and the disease develops yearly in the heifers coming into milk for the first time and in newly purchased cows, that have not been previously exposed.

The contagion varies greatly in force in the different forms of variola, the milder horsepox or cowpox, requiring actual contact (inoculation) while in smallpox and sheeppox, infection may take place at some distance from the patient (in sheep over 200 yards).

A particulate infecting material was demonstrated by Chauveau, who filtered the virus and inoculated the filtered liquid without effect, while the solids retained on the filter invariably produced the disease.

The identity of the microbe of variola has been much discussed, Guttmann and Grigorjew found in the lymph a coccus (Staphylococcus albus variolæ), Ruete a very motile diplococcus, and others streptococcus, but in cutaneous lesions it is very difficult to exclude such elements. Pfeiffer, Guarnieri, Van der Loeff, Wasielewski and others have drawn attention to small protoplasmic bodies (manifestly protozoa) found in the vaccine lymph, and which appear to be the infecting agents. Wasielewski cultivated these on a rabbit's cornea to the 48th generation, and from the last successfully inoculated a calf and several children.

Funck finds this *sporidium vaccinale* constantly in the vaccine lymph and surrounding tissue, as a refractile, amœboid, spherical organism (spore) 1 to 3μ in diameter, and, less abundantly, a round or ovoid spore cyst 25μ. These cysts are either smooth, or uneven like a raspberry, and have a single or double contoured membrane. They are easily stained with Sudan III. Examined in hanging drops they are seen just under the cover glass, not on the surface of the drops. Larger flattened bodies found in the lymph, with many included spores are manifestly epithelial cells. Copiman cultivated the organism in glycerinated collodion capsules in the peritoneal cavity of rabbits and dogs, producing zoöglœa masses staining peripherically with methylene blue, andwhich caused typical vacciniain calves. He found the same elements in variola of man. The research of Councilman sustains the theory of a protozoon.

HORSE POX. VARIOLA EQUINÆ.

This was recognized toward the end of the 18th century, in the valley of the Severn, England, by Jenner, who believed it to be the origin of cowpox, but failed apparently to distinguish it from ordinary "*grease.*" Today, when we must trace each case to a

preëxistent one in some animal, and ignore the question of primary origin, we must still recognize that it passes readily from horse to cow, and from cow to horse, through the hands of milkers and stable-men, but that it also has the source of vaccinated persons in the families of those handling the horses. Whether it was also often derived from the very prevalent smallpox in past times is doubtful, yet it appears to have been much more common about the year 1800 than it is today. When once started in a stable it passes readily from horse to horse, through the hands of shoeing-smiths and grooms, including, of course, their aprons, brushes, sponges and rubbers, and also through blankets, bandages, litter and other things on which the virus has been received. The susceptibility of the skin in the hollow of the pastern depends mainly on the frequency of chaps and abrasions, and to a certain extent on contact with the road-mud infected by other passing animals. At times the suffering animal licks the affected part, and determines the eruption on the mouth, lips and nose (see contagious pustular stomatitis). Megnin saw cases affecting the external generative organs and transmitted between the sexes by coition. (See vesicular eruption on the genitals). All such cases should be identified by inoculation.

Hertwig, as early as 1830, recorded the existence of horse-pox in Berlin, where it has been seen frequently since, and noted its transmission to man. Bouley gave evidence of its special prevalence at Paris, and of the production of typical vaccine vesicles in man by its inoculation. More recently it has been frequently observed and studied by inoculation and otherwise.

Symptoms. These, whether seen in horse, cow or man, do not differ from those of cow pox, with which it may be held to be identical. The observations of Chauveau, Warlomont, and Pfeiffer show that experimental infection, intravenous, subcutaneous, intratracheal (inhalation), or by feeding almost always failed to give the general eruption which characterizes smallpox and sheep pox. It remains strictly local except when inoculated on other parts of the skin or mucosa. It must be added, however, that small pox or sheep pox, when inoculated successfully on the horse, as a rule remains confined to the seat of inoculation. The constitution of the horse, like that of the cow, tends to resist its virulence.

The affected horse may show preliminary febrile symptoms, but

these are usually too slight to be noticed. They are followed by heat, tenderness and swelling of some part or the skin, usually in the hollow of the pastern and perhaps the back part of the metatarsal region, with more or less (sometimes extreme) lameness. On the swollen skin may be detected nodules, which may pass early into vesicles and pustules. The eruption varies, however, as developed on the comparatively hairless lips or nose, or on the densely pilous pastern.

On the lips, or other part lightly covered with hair, one can easily follow the successive formation of the round nodule, the distinct (sometimes unbilicated) vesicle, with its clear translucent straw-colored contents, and the pustule, which bursts, forming a sore, or dries up forming a dense scab, like that of cowpox.

On the heels, or on any part thickly covered with hair, the vesicle or pustule is rarely recognized, the exudate on the contrary takes place mainly on the surface, which becomes encrusted with an abundant yellowish concretion, matting the hairs together, and sometimes literally covering them. This may be very misleading to the practitioner who expects to see the succession of fully developed vesicle and pustule, and overlooking the true nature of the malady he may allow it to spread widely in a stable.

Describing his inoculation cases, Chauveau gives the following successive phenomena :—

"From the fifth to the eighth day the points of inoculation become distinctly papular. As far as about the tenth day, the papules encrease, and become more prominent, taking the form of an extremely wide cone, with a base of ⅓ to ½ inch. During this period these large conical papules are resistant and painful on pressure, but show no elevation nor change in the epidermis, save a slightly reddish reflection in animals with white skins. Afterward supervenes a new stage which may be called the *period of secretion*. This commences from the ninth to the twelfth day. The epidermis, slightly raised upon all the papule, sweats out numerous drops of a limpid, very slightly yellow serosity.

These drops soon concrete into yellowish, transparent crusts covering the whole surface of the pustule :—a species of characteristic crystallization, very different from the crusts that succeed the vaccine pustules in mare and cow. The secretion, which continues several days, is terminated from the thirteenth to the seventeenth day after inoculation. If then the crust is raised

there is exposed a humid, pink, granular surface not projecting beyond the surrounding skin. This surface is hollowed out by a very deep central cavity, a sort of umbilicus, in which is inserted, like a nail, a projection from the deep surface of the crust."

I would add that after recovery the hair in the seats of the nodules has a lighter color and, on the shanks and higher, remains dappled for the season.

No *treatment* is demanded. The application of a solution of sodium bisulphite once or twice a day, or continuously on a bandage, will greatly modify the intensity of the inflammation, and ward off complex infections. If the skin is left tender or with a disposition to crack, treat it as advised under chapped heels.

COWPOX. VARIOLA VACCINÆ.

This is manifestly the same disease, and due to the same microbe as horse pox. The disease of the one genus is easily transmitted to the other and the lesions and symptoms are the same, as if the virus were derived from an animal of the same species. Differences in the local manifestations appear to be due rather to the varying conditions of the skin and hair follicles, than to any material distinction in the virus.

Causes. Aside from the germ the conditions which favor infection are : the milking of susceptible cows with imperfectly washed hands, after dressing legs, the seat of horse pox eruption ; the milking of healthy cows after those affected with cowpox ; and the milking with hands contaminated with the exudate in cases of vaccination of man. That susceptible cows may also be inoculated successfully from smallpox patients, under given conditions appears to be true, but in Western Europe and America this is very uncommon, and would be much more so if vaccination were universally carried out. Among those who claim the identity of small pox and cow pox may be named Ceely, Reiter, Babcock, Thiele, Voigt and Klein.

Ceely alleges the infection of five cows and one heifer, in 1839, in England, from chewing the flock of a bed on which a small pox patient had died. In 12 or 14 days they had tender congested udders, with hard pimples imbedded in the skin, followed by blisters, and brownish scabs. The milk diminished, saliva drivelled from the mouth, the cheeks were inflated and retracted,

the coat stared, their feet were drawn together and the back was arched. The disease was communicatedto the owner. This was clearly an outbreak of aphthous fever, which invaded England in that year, and was still an unknown disease to medical men. The implication of the heifer which would not have been inoculated with variola through the hands of the milker, and the salivation which is unknown in cow pox, but points directly to the buccal vesicles of foot and mouth disease, are conclusive on this point.

Ceely, later, after many fruitless attempts to convey smallpox to the cow, at last met with results which indicated cowpox, and which he thereafter passed from cow to cow with the characteristic cowpox eruption.

Fletcher further reports the transmission of smallpox through the horse to the cow, and thence to the child in the form of cowpox.

In 1836, Thiele, Kasan, S. Russia, inoculated some cows on the udder with smallpox lymph, and conveyed the lymph of the resulting vesicles back to man, and from man to man for seventy-five generations of the virus without finding any variation from the type of the true vaccine disease. He repeated the experiment with equal success in 1838.

Such experiments, made before the days of careful antiseptic, or aseptic, laboratory methods, by men who were daily engaged in making vaccinations, cannot be very implicitly relied on, yet the success of Thiele in Central Asia, the early home of variola, may indicate the possibility of a transition, under given eastern conditions, which, to say the least, is exceedingly rare in Western Europe or America.

The experiments of Klein, conducted under modern methods, are more conclusive, and seem to imply the possibility of smallpox passing into cowpox, in the bovine system, under some not yet clearly defined conditions. Until such conditions are sufficiently well known, so that they can be controlled at will, no one can be justified in attempting to produce lymph for vaccination by simply passing smallpox virus through the system of the cow.

It seems important to note one or two instances of the evident transmission of smallpox from man to man through the bovine system.

In 1860, Martin inoculated variolous matter, from a man who

had just died of smallpox, on a cow's udder, and subsequently inoculated about fifty persons from the eruption caused in the cow. Most of those so inoculated had unmistakable smallpox and three died. Reiter had a very similar experience.

Chauveau (French Commission) inoculated twelve susceptible cattle with smallpox virus and produced, in all but one, small conical (smallpox) papules and vesicles, and in ten of these, on subsequent inoculation with cowpox, six proved immune, three had rudimentary pustules, and one had a distinct cowpox eruption.

A milch cow and two heifers were inoculated with smallpox and cowpox on two sides of the vulva, with the result that each disease appeared in the seat of its inoculation, with its characteristic vesicles, and the two developed side by side. The smallpox vesicles were by inoculation conveyed from ox to ox, with steadily decreasing activity. Inoculated from the cow on a child, it caused great hyperthermia, vomiting, one large vesicle like vaccinia and a general eruption like varioloid. Inoculation from this child upon another produced a mixed eruption of cowpox and varioloid. Inoculation from the second child on a bull and heifer produced papular eruption only.

Smallpox virus, inoculated on a horse produced a papular eruption, but failed to affect another horse that had been previously vaccinated. Cowpox virus inoculated on the first horse which had had the papular eruption, caused a second papular eruption (not cowpox). The virus from a vesicle in the first horse caused a similar eruption in another horse, on which it was inoculated. The lymph from the papular eruption led to a similar eruption in cattle, on which it was inoculated, but did not protect against cowpox, subsequently inoculated.

The lymph from the papular eruption in the horse, inoculated on two children, produced fever, vomiting, a general papular (smallpox) eruption, in which a few of the pustules only showed a tendency to umbilication. A child and its mother in the same ward contracted varioloid. A child inoculated from one of the first named children, had six large umbilicated vesicles like cowpox and a general papular (smallpox) eruption. Another child inoculated from the last had six large umbilicated vesicles, and a general papular (smallpox) eruption. From the papular eruption of one of these children a horse and seven cattle were inoculated and in all a varioloid eruption resulted.

The rational conclusion is, that while there is every indication of a primary identity of the two diseases, and indeed of all forms of variola, as shown by a disposition of the virus from one genus, when inoculated upon a totally different genus, to show some indication of the characteristic eruption of the latter, yet the generic type, which comes from the long-continued growth in the one class of animal, becomes so fixed, that it cannot be overcome at once, and sometimes apparently not at all, by transferring it to an animal of another class.

If the unfortunate results obtained by Martin, Reiter, and Chauveau, are insufficient to deter from the use of smallpox lymph which has been passed through the cow, the long experience with humanized vaccine, which in its inoculation from man to man for a century has shown no tendency to revert to smallpox virus should be a sufficient warning against such dangerous optimism.

No deduction can be safely drawn from the comparative mildness of most of the cases caused by reinoculation, from cow or horse to man, inasmuch as that all forms of variola can be rendered less severe by resorting to inoculation, which was extensively practiced to limit the ravages of smallpox before the days of vaccination, and is still largely resorted to in the case of sheeppox in Europe. In each of these diseases the mortality can easily be reduced to 2 per cent. instead of the 20 to 50 per cent which are lost when the disease is contracted casually.

As occuring casually, cowpox like horsepox is rare. Yet in Denmark, a dairying country, 1,037 cases were reported in 1877–8, and 878 cases in 1888–9. I have found some outbreaks explainable, through the existence of vaccinations in the families of the milkers, and Bollinger says that in Germany, most outbreaks take place in spring, the time when children are vaccinated. He should have added that this is the usual time of parturition in the cow, the time when primipara are first subjected to the danger from the hands of the milker, and when the cow from the non-infected district is brought into an infected stable for the season's milking. In a dairying district in Tompkins Co., N. Y., the affection appeared every spring, in the same barns, in heifers with their first calf and in newly bought cows. All older cows, bulls, steers and unimpregnated heifers escaped.

Symptoms. The period of incubation is two days, after inoculation, and though it may appear to extend to a week when the disease is contracted accidentally, it is impossible in such cases to state the exact date of infection. The preliminary fever is not always present, or recognized, yet there may be slight encrease of temperature, partial impairment of appetite and rumination, extra firmness of the fæces, a higher color of the urine, and above all a slight diminution of milk, which is a little more watery and coagulates more readily, than the normal.

This is followed by heat and tenderness of the udder and the appearance on the teats of small, pale-red nodules the size of a pea or larger. In one or two days more the nodule, largely encreased in size presents in the centre a depressed or *umbilicated* bluish white portion, with a firm yellowish, reddish or reddish blue margin, and outside this a soft pink areola, shading off into the white skin. The epidermis is raised at points by a viscid, yellowish lymph, enclosed in a series of saccules (multilocular vesicle). The vesicle encreases to 8 or 10 lines in diameter by the eight or tenth day, and exceptionally, the umbilication is effaced by the excessive production of lymph. If left unbroken a brownish shade appears in the centre and gradually extends toward the periphery, the contents becoming purulent, and the pustule gradually drying up to form a crust. The drying and thickening of the crust goes on until the fourteenth day and the crust is usually detached by the twentieth, leaving a pale rose colored, smooth, shallow depression, which forms the permanent pit left after the skin has healed. The primary scabs usually show the central umbilication, and always the conical projection in the center of the deep aspect, and corresponding to the pit.

Vesicles on the mammæ may pass through the above stages, but those on the teats are usually ruptured by the hands of the milker as soon as the liquid is thown out, and this gives rise to troublesome sores, with complex infections, at times implicating the gland tissue so as to cause destructive mammitis with loss of one or more quarters, and in any case abraded and irritated at each milking, so that the animal resists handling, the milk is drawn off imperfectly, and dries up or the cow becomes an inveterate kicker. If the milker has not been vaccinated he is liable to contract the disease.

A succession of vesicles often appear on the same animal, so that they may be found in all different stages of vesicle, pustule and crust on the same bag at one time. The later eruptions may be the result of inoculation from the earlier ones, and tend to prolong the attack materially.

In inoculation of the bovine animal for the production of lymph for vaccination, the skin of the abdomen from the symphysis pubis to the umbilicus is shaved, or in other cases the skin between the thighs, or in still others the skin on each side over the loins, and the virus applied in 50 to 200 points, by preference scraped until liquid oozes, but without any escape of blood. In a warm room the eruption matures in four or five days, its form taking on an appearance approximating that seen on the hairy skin of the horse. The individual lesions are somewhat extended corresponding in form and size to the abrasion on which the lymph was applied, and usually present the appearance of a raised patch, covered by a grayish film of epidermis, on the removal of which there is seen a raw alveolated surface filled with the amber-colored lymph.

Differential Diagnosis. From *aphthous fever*, cowpox is clearly distinguished by (a) the multilocular structure of the vesicle, while that of aphthous fever is a single undivided cavity which can be drained completely by a single needle prick ; (b) by the pitting or umbilication, the aphthous vesicle being uniformly rounded and convex ; (c) by the absence of vesicles or sores on the mouth and feet, which are rarely wanting in the aphthous eruption ; (d) by the comparative absence of hyperthermia and constitutional disturbance, which is better marked though still slight in aphthous fever, and (e) by the absence of the intense and subtle infection of aphthous fever, which quickly attacks a whole herd and extends with equal rapidity over sheep, goats and pigs, attacking all cloven-footed animals virtually without exception. The cowpox patient, on the contrary, does not necessarily infect the cow in the next stall unless milked by the same hands, and spares heifers, bulls, steers, sheep, goats and pigs.

From the *rinderpest* cutaneous eruption it is easily distinguished by the presence of lymph in the lesion, that of rinderpest being a mere epidermic concretion ; by the absence of the intense fever, anorexia and general constitutional disturbance, and of the early

and high mortality which characterize that disease; by the absence of rapid and uniform infection of other cattle irrespective of a common milker; by the immunity of heifers, steers and bulls, which are speedily prostrated by rinderpest, and by the absence of the congestions and epithelial concretions of the mucosæ which characterize rinderpest.

From the *leg irruption* found in animals feeding on distiller's swill and grains, or on the mast of beet sugar factories, by the history of the outbreak, of the dietary, of the seat and nature of the disease, and by the escape of animals living on a different aliment.

From the *false cowpox* (varicella) it is distinguished by the unilocular lesion of the latter, its absence of areola, and its rapid pustulation and drying, in five or six days into a thin papery crust instead of a thick, firm, umbilicated scab, as in cowpox. Varicella is further liable to appear in successive crops and thus last for several weeks.

The *streptococcus eruption* on teats and udder, is marked by the formation of abscesses of various sizes from a simple pustule upward, by the unilocular condition of the pus sac, by its tendency to invade the deeper tissues, and by its rupture and granulation without the formation of the thick umbilicated scab of cowpox.

The *hard warty growths* on the teats which last for weeks or months should never be mistaken for cowpox.

Cowpox usually lasts for some weeks in a herd, the duration depending on the number of susceptible animals and, whether these are habitually milked by the same person.

Course. Prognosis. It is a mild affection, which does not endanger life, yet it causes considerable loss through diminution of the milk secretion and, it may be, altered character of the milk, through the persistent sores and ulcers of the teats, through inflamation of the mammæ, and through an acquired habit of kicking.

Treatment is rarely needed. Any costiveness should be corrected by a cooling saline laxative (½ to 1 lb. Epsom salts) or by soft food, and milking should be done with great care to prevent rupture of the vesicles and the formation of sores. A teat tube may be used if necessary. Sores may be dressed with bland ointment. An ounce each of spermacetti and sweet almond

oil with half a dram of gum myrrh. Or the vesicles or sores may be washed after each milking with a solution of 2 drams hyposulphite of soda in 1 quart water.

SHEEPPOX. VARIOLA OVINA.

Synonyms. Pocks ; Peltrot ; Clavelee, Picotte, (Fr.).

Definition. An acute febrile affection, eminently contagious, prevailing epizoötically in sheep and goats, characterized by early and marked hyperthermia, and general constitutional disorder, followed by the appearance on the bare or merely hairy portions of the skin, of diffuse redness becoming intensified in points, a rounded papular eruption, passing into vesicles, pustules and scabs, which later dry up and drop off in 15 to 20 days.

Pathogenesis. Beside sheep and goats which contract the disease by exposure, the following genera have been successfully inoculated : ox, dog, pig, horse.

Forms. Two typical forms are recognized : (*a*) the *discrete, regular* or *benign*, in which the vesicles remain relatively few, and well isolated from each other, and (*b*) the *confluent, irregular* or *malignant* in which the vesicles are generally diffused over the body, even on the parts covered by wool, and set so close together that they merge into each other forming extensive continuous lesions. Other forms are the *hæmorrhagic, purple* or *black sheeppox*, the volante or intermittent kind, etc.

Geographical Distribution. Formerly common in Central and Western Europe, it still prevails continuously in the Balkans, the Danubian Principalities, Italy, Spain, the South of France and Algiers. Like other forms of variola, its permanent home is in Asia.

Causes. Long before the advent of modern bacteriology, sheeppox was held to be always and everywhere the result of contagion alone. Whenever it entered a new locality it was as the result of the importation of an affected sheep or one of its products : insular places like England maintained a permanent immunity, though the disease prevailed on the other side of the narrow straits or channel ; yet when imported (1847 and 1862) it demonstrated a general susceptibility of the flocks on exposure or inoculation ; more distant lands (America, Australia, Tasmania,

South Africa, etc.) in the absence of imported infection remain clear to the present day. This absence for centuries from countries where cowpox, horsepox and even smallpox prevails speaks volumes for the clinical difference of the germs.

The infection is more intense and diffusible than that of cowpox and horsepox resembling in this the smallpox of man. Absolute contact is not necessary, in either case the infection is carried in the air either on dust or otherwise, and above all in a confined building, a crowded sheep fold or a dusty highway.

In all forms of variola the virulence is concentrated in the lymph of the vesicle, and in horsepox and cowpox it is largely confined to this, whilst in sheeppox in severe cases it must also at times infect the blood, as lambs are occasionally born with sheeppox. On this basis the infection of the secretions generally, has been asserted, but it is to be supposed that in moderate cases these are contaminated after secretion. Nocard and Roux produced immunity by the transfusion of blood from the sick to the healthy, but in no case a variolous eruption. Even the serosity from the swollen lymph glands failed to convey the disease.

In view of the diffusible nature of the germ, however, we must recognize that all secretions may be quickly contaminated as soon as they are exposed, and therefore no product of a sick or suspected sheep can be held to be safe, and all should be treated as presumably infected. The most dangerous products and those most liable to convey the disease are wool, hides, litter, buildings, yards, covers, parks, railway cars, boats, manure, urine and milk. Living creatures like men, dogs, cats, birds, vermin, flies and other predacious insects are occasional bearers of infection.

Receptivity must be considered in every case. In countries and districts habitually immune from sheeppox, all breeds appear to be equally susceptible, the only refractory specimens being sheep that have survived a first attack, and lambs born of ewes that had the disease (naturally or inoculated) during the later stages of gestation. New born lambs, on an exclusively milk diet, are alleged to be somewhat refractory. In a country where sheeppox has prevailed long and extensively, as in Algiers and Brittany, certain breeds of sheep seem to have attained to a large measure of immunity (Nocard). This is doubtless largely due to the survival of the more insusceptible strains of blood, as Algerian sheep carried into France lead to most virulent outbreaks among the native animals (Galtier).

This relative immunity is still more decided when we come to animals of other genera. Though the latest results of research seem to identify the sporidia found in the different forms of variola, yet the long habit of living in an environment found in the ovine race unfits the germ for pathogenic life in various other genera that have their own variolæ. Sacco and Villain reported inoculations from sheep to man, but as the first was experimenting with variola at the same time, and the second did not test his pustules by reinoculation, the results are not convincing. Küchenmeister had a general eruption in sheep after intravenous injection of variola of man, but failed to test it by reinoculation. Voisin and Nocard on the other hand were uniformly unsuccessful in attempts to convey sheeppox to man, and the handling of variolous flocks from time immemorial must have led to many cases in man, had he been appreciably receptive. It is virtually the same with cowpox. Huzard vaccinated 2,000 sheep without producing immunity from sheeppox. Voisin had precisely similar results. He further inoculated infants with sheeppox, and later, successfully, with vaccine lymph.

It does not follow that these genera would be insusceptible under all conditions. Yet the failure to immunize against each other would argue a wider divergence of sheeppox and cowpox, than of smallpox and cowpox, or than of bovine and avian tuberculosis.

Galtier failed to inoculate sheeppox on rabbits or Guineapigs, yet Jourdan, records a destructive outbreak in the Alps, in hares, kept in the same places with variolous sheep.

Accessory External Causes. All unwholesome conditions of life, and especially overcrowding, filth, starvation, and neglect contribute to the extension of the infection. Still more so, the importation of sheep, whether in the parks of armies, or in the channels of trade, by rail, or steamboat, through stockyards or markets.

The virus is possessed of unusual vitality, and in the dried condition, secluded from air and light will remain virulent for an indefinite period. Hence the danger of wool, and dried sheepskins. Even in the moisture of an ordinary shed it has retained its infectiveness for five or six months. The sheep that has recovered from the affection may transmit the disease to others for

a period of six weeks. The virulence is rapidly destroyed by exposure to free air and sunshine, by a high temperature (140° F.), by dilute HCl (2:100), by carbolic acid (2:100), by zinc chloride (5:100), by potassæ permanganas (10:100), by lime chloride (4:100), or indeed by any of the strong antiseptics.

Incubation. This extends from four to seven days on an average, with a minimum of two and a maximum of twenty days. The conditions favoring an early eruption are youth, high condition, and a hot season, or close warm building, with overcrowding. Those favoring a tardy eruption are old age, debility, and above all a cold or wet season with exposure in the open air. Simonds, who inoculated in England in the cold months (October and November) never saw it exceed 13 days. In certain cases, however, when the eruption had already commenced in mild weather, the sudden occurrence of a week of cold and wet, would stop it short to start anew on the return of warm weather a week later. In inoculated cases the incubation is shortened, while it is prolonged when the virus is introduced by the digestive or respiratory passages.

Symptoms. During the last two or three days of a prolonged incubation and especially in old sheep, there may be some impairment of appetite and rumination, dulness, a stiffness of movement of the hind limbs, and a disposition to lag behind the flock. The temperature may even have risen (104.5° F.), yet Simonds has never seen the febrile symptoms precede the eruption.

There follow, trembling, or rigors, accelerated pulse (80 to 90) and breathing, arched back, anorexia, suspension of rumination, costiveness, redness of the eyes, epiphora, a watery discharge from the nose, gradually becoming more viscid, and a marked hyperthermia (105°, 106° or 107° F.) If the skin is white, a blush, with some heat, is shown in the seat of the future eruption, usually on the parts uncovered by wool, the axillæ, sternum, abdomen, udder, inner sides of the thighs, lower surface of the tail, and the face—especially the eyelids, nostrils and lips.

In two days more, deep red points, like flea bites, appear more or less numerously, in the congested parts of the skin, and in twenty-four hours these have increased to firm rounded papules which are felt to extend into the true skin. These vary in size from 3 to 12 mm. or more. In two or three days the papule has

become firmer and less tender, and shows in the centre a paler area where the exudation has resolved it into a vesicle. The characteristic sheeppox vesicle is rounded or flattened on its summit, being rarely pointed as in smallpox, or umbilicated as in cowpox. It is usually surrounded by a pink zone, which is at times infiltrated, firm and resistant. After about the sixth day, the vesicle becomes yellowish from the formation of pus. The pustules, with the surrounding tumefaction, encrease for about three days, when if they have not become confluent nor infected, they begin to dry up, acquiring a grayish crust on the surface which gradually encreases to a thick scab, which in five or six days more is detached leaving a pink, pitted spot covered by forming epidermis. Sometimes no distinct scab forms, but the crust dries, cracks up, and falls off in scales.

All the vesicles do not appear at once, but some earlier and some later, so that the successive stages of the eruption may be often seen together on the same subject, and the case is thereby materially prolonged.

As in other forms of variola the hyperthermia usually subsides with the appearance of the eruption, but as often re-appears in some measure with pustulation (secondary fever of reaction), and becomes more severe in proportion to the extent and confluence of the eruption and the occurence of complex infection.

The eruption may invade the conjunctiva, the cornea (inducing blindness), the nasal mucosa (causing difficult breathing), the mouth (salivation, difficult mastication), the pharynx (difficult deglutition), the stomach and intestines (diarrhœa), the bronchia (cough), or the vulva.

With the occurence of desiccation, in discrete cases, the fever subsides, the swelling of the skin and head dissapears, the discharges from the nose and eyes cease, and the animal is restored to health.

A secondary eruption will sometimes take place, but only papules form, which disappear without reaching maturity.

The duration of discrete sheep pox is usually about three weeks, but it may reach four in cool weather. It is more rapid and more prone to dangerous complications in hot weather. As the members of a flock are usually attacked in succession, it may take three months to pass through a flock.

In *confluent or complicated cases* the fever of invasion is very high, the sheep dull, prostrate, tender to touch along the back and loins particularly, with hurried, labored, panting breathing, and mawkish, fevered breath, the weakness increases rapidly, standing even may become difficult, the wool is loosened and falls off in patches, the exposed skin is red, shading, it may be, into a violet hue. The head droops, the lips and nostrils swell, saliva drivels abundantly, anorexia is complete, though thirst may be ardent, a yellowish and even reddish, fœtid discharge may flow from the nose, respiration is difficult, the eyes are watery and deeply sunken, and the head, limbs, breast and abdomen are extensively infiltrated and œdematous.

Fever does not subside with the occurence of eruption, which may appear thickly over the whole body, the wooly parts as well as the bare or hairy. The vesicles, which have often a dark, unhealthy look, tend to become confluent, and instead of proceeding regularly to maturity, they may remain hard or indolent papules or nodules or blacken and dry up. The secretion, when it takes place, is liable to be a thick, yellowish, fœtid pus. The eruption has much more tendency than with the discrete form to invade mucosæ, and not infrequently the serosæ are involved, especially those covering the lungs, liver and spleen. In the worst cases death may result from the fever before any eruption has taken place, in other cases the extent of the internal lesions tends to hasten a fatal result. With the amount of care that can be given to a flock, confluent cases are usually fatal.

In connection with the itching and scratching of the nose, abrasions and complex infections ensue, resulting in extensive ulceration and gangrene implicating the nasal cartilages and bones. The eruption around the pasterns may lead to shedding of the hoofs, and sloughing of the whole digital structures. Suppurating and gangrenous swellings form subcutaneously and in the lymph glands with fatal results. Blindness is especially liable to happen from the formation of the eruption on the conjunctiva and cornea. Ulcerations and sloughs are common on the internal mucosæ, and even on the serosæ. In implication of the abdominal organs, a fœtid diarrhœa is usually present, and in pregnant ewes abortion is the usual result.

Lesions. The cutaneous lesions are in their multilocular structure, the same as in cowpox, but differ materially in size and

form. The typical vesicle is small (5 to 12 mm.), and neither broad and umbilicated like cowpox, nor conical and pointed as in smallpox, but round with a slight flattening on the summit. On the same animal may often be found all the successive stages at once, red points or hæmorrhagic spots, vesiculation, pustulation, and even desiccation. The early changes are seen in the papillary layer and rete mucosum, in the form of swelling and congestion of the papillæ, exudation, active proliferation of the epidermic and tissue cells, and migration of leucocytes, (embryonic cells, polynuclear), vacuolation of the papule, the spaces being filled with a straw colored exudate, and finally, the replacing of this by pus. If the local inflammation is very acute, the pustule may be resolved into a small abscess. On section of the skin the affected parts are found to be the seat of congestion and gelatinoid exudation, extending into the subcutaneous and intermuscular connective tissue, and even the muscles themselves are blackish and their capillaries engorged. The exudation is especially abundant in the dependent parts (head, neck, sternal and abdominal regions, legs). In proportion to the severity of the attack there are congestion, exudation, swelling, and blood extravasation in the lymph glands.

The nasal mucosa is congested, thickened or even ulcerated with abundant, tenacious, muco-purulent secretion and the nasal chambers narrowed or obstructed, and similar changes may be present in the larynx, trachea and bronchia. Spots of ecchymosis and even distinct variolous vesicles are to be expected. The pleuræ are often the seat of congestion, petechiæ, exudation, and discoloration (red or pale). The lung may show congestion and hepatisation in circumscribed areas, or there may be on the surface of such hepatised portions red or grayish foci, like a lentil, pea or bean, the seat of degeneration or forming minute abscesses. The pericardium and myocardium may be involved to a limited extent.

The buccal and pharyngeal mucosæ may be the seats of vesicles, or erosions or ulcers the result of their destruction, and the gastro-intestinal mucosa presents ecchymoses, vesicles, abrasions, and open sores in the midst of inflamed catarrhal patches. The mesenteric and other lymph glands are congested, swollen, softened and friable. Congestions and petechiæ are found on the

peritoneum, liver, spleen and kidneys, with, at times, nodular foci. In certain cases congestions and effusions have been found on the pia mater and arachnoid, in the cerebral ventricles and the brain substance.

Prognosis. Mortality. Mortality varies much with the severity of the special outbreak and the conditions of life, favorable or otherwise, in which the flock is placed. In the milder forms the mortality may not exceed seven per cent., while in the more violent the whole flock almost may perish. A fatality of 20 to 30 per cent is the general average. Yet the inoculated form kills but 2 per cent.

If at the outset there is great weakness and prostration, complete anorexia and high fever, the prospect is discouraging. If the fever is moderate and strength and appetite retained, the case is very hopeful. On its first advent into a new country it causes a far greater mortality than after it has been long domiciled there and frequent outbreaks have killed off the more susceptible strains of blood. Again very hot weather, or, still more, the prevalence of cold, drenching rains aggravates an outbreak and greatly encreases the fatality.

The loss is not to be measured by the deaths alone. The failure of the crop of lambs, through abortion, the shedding of wool, the loss of sight, hearing, hoofs, digits, flesh, stamina, etc., render recovery far from desirable, in the worst cases, as the animal fails to thrive or pay for its keep. On the contrary it is immune from any future attack, and if left in a thrifty condition it becomes especially valuable in a district where sheeppox prevails.

Treatment. Once established in the system the disease will follow its regular course, through all its stages. Yet we can, by dieting, pure air, cleanliness, shelter and even by medicinal measures, do much to render that course a safe one. Cool sheds, pure air, clean floor, dry clean litter and shelter from rain are above all important. The sheep may be separated in different enclosures in small lots of 5 or 6 to prevent crowding, heating, and excitement, and in any case the infected should be removed from the noninfected, and even from each other to avoid infection and reinfection. This is especially requisite in hot weather.

For the strong and vigorous, a diet of sliced roots and meal

(oat, bran, linseed, barley, wheat middlings) is good, while for the weak, gruels of oat meal, barley meal, linseed meal, or the same agents dry, may be given. Powdered saltpeter may be given in this (1 oz. to 8 or 10 sheep) and common salt allowed to be licked at will. Drinking water may be given pure, or slightly acidulated with sulphuric or hydrochloric acid, or hyposulphite or bisulphite of soda may be used as a substitute for the latter. The bowels are usually costive, and may be relieved at first by 3 ozs. of sodic sulphate, and later, if need be, by soapy injections. Often during the course of the disease a sudden access of fever may be cut short by a milk laxative, if that is not contra-indicated by existing diarrhœa.

Avoid giving heating agents to bring out the eruption. The severity of any case and the danger of complex infection are usually in ratio with the extent of the eruption.

Lotions of hyposulphite of soda may be applied to the affected parts from the first, and even weak lotions of chloride of zinc after the maturation of the pustules. For the eyes, nose and mouth antiseptic lotions may be called for.

In the advanced stages, in weak subjects, tonics and stimulants may prove useful. To the mineral acid, quinine (10 grain doses) or gentian (drachm doses) or other bitter may be added for valuable stock.

Treatment is only permissible in the case of very valuable animals and when they are surrounded with the most perfect antiseptic precautions, to prevent the escape of the infection.

Prevention. As in all dangerous infections this must be the preëminent object, and when a new country has been invaded by the disease, no sentiment nor alleged value of affected or exposed animals should be made the warrant for treatment, nor stand in the way of the extinction of the plague by the most rigorous measures. The recovery of an individual flock is never to be put in the balance with the danger to which other flocks are thereby exposed. To avoid smuggling away of exposed animals, and consequent spread of the disease, the loss should be met by the commonwealth and no folly, misnamed administrative economy, should tempt a stock owner to endanger the flocks of a whole nation.

Sheep and goats from countries where sheeppox exists should be absolutely excluded. If, in exceptional cases they are allowed

to land on our shores, they should be guaranteed by a veterinary certificate as coming from a noninfected district, by a route free of infection, they should be critically examined by an expert on arrival, and if passed, should still with all clothing and utensils, be subjected to thorough disinfection. The clothing of attendants should be similarly dealt with. If importation is merely across a frontier, a quarantine of 21 days followed by a disinfectant bath should be enforced.

If sheeppox has gained a footing in a flock in a new country, the flock should be at once appraised and destroyed, and the place thoroughly disinfected and shut up for three months. All cars, ships, wharfs, landings, chutes, yards, buildings, parks, roads, etc., used by them should be closed and thoroughly disinfected. All flocks exposed to any such place or thing should be placed in strict quarantine for three weeks, under official veterinary supervision and disposed of should they become affected.

As an alternative each infected and suspected flock should be secluded in a well fenced place or shed from which all men except the necessary attendants, all dogs and other mammals, including vermin, all birds and as far as possible all flies are excluded. They should be divided into small lots of 5 or 6 placed in separate pens, their temperatures should be taken 3 or 4 times a day, and any one showing a temperature of 104° or 105° F. should be instantly removed to a separate pen, and destroyed as soon as the disease can be identified. In this way a diseased sheep can usually be removed before it has infected its fellows, and at the worst the infection will rarely spread beyond the 5 or 6 animals enclosed in the pen where the first case appeared. It is well to sprinkle the wool of all the flock with a 5 per cent. solution of carbolic acid. and the floor or ground with chloride of lime. The diseased carcasses should be thoroughly sterilized by burning, boiling, or immersion in strong acids, or they should be deeply buried, the infected pen disinfected on each occasion, and the hands and clothes of the attendants purified.

Careful treatment in this way will usually cut short the disease with the loss of those only that were already infected when the outbreak was taken in hand, but it must be in the hands of men who are at once experts and vigilant and trustworthy. The English invasion of 1862, under the direction of the late Professor

Gamgee, was completely stamped out in four weeks on this plan, whereas the invasion of 1847, met by the expedients of inoculation and quarantine, lasted for four years, with great losses in a number of flocks, and a very heavy bill for continued expert supervision. This should be a wholesome lesson to the American legislators who consider the prompt extinction of infection, by abolishing this source of its increase in the living body, as a wasteful outlay.

Preventive Inoculation, Ovination. If it were possible to give immunity against sheeppox by inoculation with the exudate of cowpox, and without danger to the sheep from fatal cowpox, or from its transformation into the more destructive sheeppox, it would be a most desirable resource. But experiment goes to show that vaccination is useless, in the temperate climates at least. Sacco, Hussan, Buniva and others in Italy vaccinated sheep extensively and claimed to have obtained good results, but this has not been endorsed by subsequent observers. D'Arboval vaccinated 1,523 sheep, of which 1,341 contracted cowpox, and out of 429 of these exposed to sheeppox later, 308 contracted the latter disease. Ceely vaccinated two sheep, both of which afterward had mild sheeppox through inoculation. Simonds and Marson vaccinated 306 sheep, 112 of them successfully, and of these last two-thirds contracted cowpox a second time on re-vaccination. Twenty-nine of the successfully vaccinated sheep were inoculated with sheeppox lymph, and in every case successfully. It is obvious that vaccination, as a protective measure, is absolutely untrustworthy in France or in England. Under the warm skies of Italy, as in Persia, there may be a close relationship between the two diseases, yet in Italy sheeppox was constantly prevalent, and it is to be feared that the immunity in a number of cases was due to a previous attack of that disease rather than to the vaccination. It should be noted that in Persia sheeppox is said to be communicable to man, while in England, Ceely failed to transmit it.

Ceely suggested variolisation with human smallpox as a preventive of sheeppox, but Simonds and Marson failed to convey smallpox to the sheep, though the same animal readily contracted sheeppox. One can hardly contemplate Ceely's proposal with equanimity. Immunity for sheep would be dearly bought at the cost of a general diffusion of smallpox virus.

Ovination or inoculation with the lymph of sheeppox is the only accepted method of immunization. It entails, however, an extraordinary multiplication of the virus in each inoculated animal, and considering the numerous loopholes for its possible diffusion it can only be looked on as a very dangerous and usually, in the long run, a very expensive resort. The experience of England, above referred to, is eloquent in witnessing against ovination, and "in Prussia and Austria the dissemination of sheeppox went hand in hand with inoculation."—Friedberger and Fröhner.

On the other hand *ovination* is not fatal to the flock operated on. The mortality is often below 1 per cent., and virtually never exceeds 4, with a general average of about 2 per cent. It is this comparative impunity of the inoculated flock which closes the eyes of most persons to the great danger to a whole country and the wasteful prodigality of the operation.

Ovination further shortens the duration of the outbreak in a large flock, passing all through the disease in 21 days; whereas as contracted by simple exposure, the duration of the outbreak may last 90 days or more. It further enables the owner to give such protection, shelter and care as will guard the flock against exposure and dangerous complications. But while preferable to the abandoning of the disease to its natural course, it is always to be strongly condemned where it is possible to adopt the method which detects the sick animal in the incipient stage by thermometry, and does away with the infection by its removal, followed by disinfection. The only excuse for ovination is the general prevalence of sheeppox on an island or other secluded district where there is no great added danger from the further diffusion of the virus, and when its simultaneous practice over the whole region can be made the basis for universal disinfection and the definite extinction of the contagium.

Technique of Ovination. The lymph ("ovine") should be taken from a mild case of the disease, and from a vesicle at full maturity (about the 6th day), but containing as yet a clear, translucent exudate, without turbidity or other indication of formation of pus, or other infection. Inoculation is made by preference on the bare lower surface of the tail near its tip or 3 to 4 inches behind the anus. If this is unsuitable, the inner side of

the ear, an inch from the tip is usually selected, though there is here an added element of danger, owing to its proximity to the eye. The insertion is made with an ordinary suture needle, which is introduced obliquely under the epidermis about one line, and pressed upon with the thumb as it is withdrawn. A still better instrument is the inoculation needle or lancet with a groove or spoonlike hollow on one side. Or the skin may be scratched or abraded with the lancet, as in vaccination, until serum oozes, when the virus is rubbed on and the part is covered with a piece of sticking plaster. On the third or fifth day the flock is again examined and those that have failed to *take* are ovinated anew.

The virus is most conveniently taken direct from the affected sheep, but it may be preserved in capillary tubes, or on glass or ivory points, or mixed with glycerine between glass plates, or finally, the first scab well dried may be preserved and utilized, a minute portion being inserted with the lancet in a pocket made under the epidermis.

Attempts have been made to secure a mitigated and safer virus, by diluting the lymph with water or normal salt solution (1 : 50–150) (Peuch), by amputating the seat of inoculation (the tip of the ear) on the 4th or 7th day, when the vesicle is formed) Galtier), by taking a susceptible sheep and inoculating it with sheeppox virus for ten consecutive days, and then selecting for use the lymph from the papule of the sixth inoculation. The inoculations of the seventh day and later give rise to no papule even (Pourquier). By this means it is claimed that the inoculated disease remains strictly local, passes through its successive stages in a shorter time (15 days or less), and is perfectly harmless to the sheep inoculated. Nocard and Mollereau sought the same end by mixing the virus with oxygenated water, and Semmer and Raupach by heating it to 130° F. In view of the facts that it is only under extraordinary conditions that ovination is permissible at all, and that the mortality, resulting from it can be kept down to about 1 per cent., it seems hardly worth while to attempt to obviate this loss, by any method which may come short of the full measure of immunity.

Ovination confers immunity for a year or longer.

The care of the flock during eruption is the same as in sheeppox contracted in the usual way.

SHEEPPOX IN GOAT. VARIOLA CAPRINA.

The goat can be successfully inoculated from the sheep, the resulting vesicles being smaller and less prominent, and the disease assuming a milder type. It is alleged to pass naturally from sheep to goat, and from goat to sheep, as well as from goat to goat but, on the whole, it is a rare disease and goats have been known to live in flocks of sheep attacked with sheeppox, without themselves contracting the disease. The malady seems to be of little account to the goat, yet in sheep countries, the interests of flockmasters would demand that it should be stamped out as vigorously as the same disease in sheep.

GOATPOX ; VARIOLA CAPRINA.

In Persia according to Bruce goats suffer from a form of variola, having larger vesicles, umbilicated and approximating to, if not identical with cowpox. In Algiers there is found a goatpox from which sheep are alleged to be immune (Nocard, Peuch, Bremond, Galtier) so that further observations are wanted to put the different forms of variola of the goat in their proper places. In a country where sheeppox is constantly present, there is of course the possible source of fallacy of experimenting with animals that have already had the disease, therefore it would be expedient to put all forms of goat variola under police control.

SWINEPOX. VARIOLA SUILLA.

Variola appears to be rather more frequent in swine than in goats. Ficanus as quoted by Joubert saw it in 1567, contracted it was supposed from smallpox patients. It was noted by Ramazini in 1690, by Stegman in 1697, and later by Gerlach and others. In 1891, 517 cases were reported in Hungary. It is said to be derived from man by the use of bed straw for litter and from sheep by occupying the same stall. It can be transmitted experimentally from pig to pig, from pigs to goats, and from goats back to pigs (Gerlach). It is also claimed to pass from the pig to man (Freidberger and Fröhner). Young pigs are especially subject to it and one attack confers immunity for the future.

Symptoms. After a febrile condition of variable length, but usually of a high intensity, red spots appear on the head, neck, chest, belly or inside the arms and thighs, at first like flea bites, but passing through the stage of papule to become vesicular on the sixth day. About the ninth or tenth day they become purulent, and in two or three days more a black, concave, circular crust has formed which is soon detached. The eruption may be *local* or *general*, *discrete* or *confluent* and the issue of the case will depend much on this character. In exceptional cases the eruption invades the mouth, eye, throat, stomach, or intestines. Croker notes the accompaniment of a fatal lobular catarrhal pneumonia.

It must be carefully distinguished from urticaria, eczema, and eruptions due to pustulating irritants.

Treatment is in the main the same as for sheep, care being taken to secure perfect cleanliness, pure air, dry clean litter, easily digested food, and protection from crowding, undue heat, cold or wet. Buttermilk and other acidulous and diuretic drinks are recommended, and careful attention to the state of the bowels throughout.

Prevention is still more important, and better than any treatment would be the most rigorous measures for its extinction along the lines laid down for sheep pox. Whether the infection has been derived from man or sheep it must be looked on as eminently dangerous to the class of animals from which it originated, and every available means used for its extinction.

DOG POX : VARIOLA CANINA.

Dog pox is rare, the affection occuring especially in the young. It is said to be derived in certain cases from smallpox patients (Weiskopf), and in others from sheep pox (Röll). The latter claims that the dog has also its own specific form. Dupuis and others claim experimental transmission from man to dog. At the same time eruptions connected with gastric or hepatic disorder, distemper, eczema, or aphthous fever are liable to be mistaken for it.

Symptoms. The young animals suffer from fever for a day or two, followed by heat and redness on the sides and belly, and points of deeper red, like flea bites, which gradually evolve

through papules and vesicles to pustules, and terminate in crusts, that drop off leaving round bare spots. The vesicles may appear locally or generally and may be discrete or confluent. Sucking puppies, when attacked usually perish.

In *treatment* the same hygienic measures are demanded as for other animals, special care being required to keep the bowels and kidneys acting in a healthy manner. Perfect cleanliness must be secured, and nonstimulating, easily digested food. Excessive fever would demand tepid baths, cold sponging, or acetanilid, and an undue warmth, crowding and cold exposure must be alike guarded against.

In view of the alleged sources of the disease in man and sheep, the strictest seclusion of the affected dog, in disinfected surroundings will become absolutely essential and when this cannot be carried out he should be summarily destroyed.

VESICULAR EXANTHEMA OF HORSES.

Definition: **Vesicles usually on external genitals, with fever and infection. Causes: Contagion by coition, clothing or stable utensils; inoculated on man or cow, resembles cowpox, but a first attack does not always immunize a stallion; inoculable on sheep, goat and pig. Symptoms: Incubation one to six days; local redness, heat, swelling, tenderness, papule, vesicle, pustule, scab, desquamation, depigmentation on genitals or in sucklings on mouth, lips, nose; may extend to mammary and inguinal regions, with swelling, stiffness, and constitutional disturbance. Diagnosis from dourine. Treatment: Cleanliness, astringent, antiseptic lotions; open and disinfect abscesses; laxative or diuretics. Prevention: Withhold from breeding.**

Definition. An acute infectious disease, manifested by hyperthermia, hyperæmia, and the formation usually on the external generative organs of a crop of papules rapidly progressing to vesicles, pustules, and open sores, and attended by great local irritation and itching. It runs a mild course and recovery ensues spontaneously in about fifteen days.

Causes. The disease is only known as a contagious affection, propagated from animal to animal by breeding together, or by using the same comb, brush or rubber, or by sucking, and in rare cases by other accidental or experimental inoculations. To make a successful inoculation the virus must be taken from the vesicles

or pustules. When transferred to man or ox it develops an eruption which is indistinguishable from cowpox (Trasbot, Peuch, Galtier). Still a first attack does not always confer immunity, as stallions may have two successive attacks within a few weeks (Steinhoff). This implies that either this is not genuine cowpox, as claimed in France, or that two different exanthematous affections have been classed as one. Inoculation with the virus has produced a characteristic eruption in sheep, goats and pigs.

Symptoms. After a period of incubation of from one to six days there appear heat, swelling and tenderness of the affected part, followed by the formation of papules, gradually passing into vesicles, pustules and scabs which scale off, leaving a white depigmented surface. In stallions and mares the usual seat of the eruption is the external generative organs; in young sucking animals the mouth, lips, nose and quarters, and in inoculated cases wherever the virus has been implanted.

In the *mare* there is swelling of the lips of the vulva and redness of the vaginal and vulvar mucosa with points of a darker red, which become firm, papular, vesicular and finally pustular. A similar eruption shows on the skin of the swollen labiæ, on the lower surface of the tail and on the hips, though on these points the phenomena are obscured somewhat by the abundance of pigment. The contents of the vesicles are at first limpid but gradually change to opaque yellowish-white, brown, or even red from slight blood extravasation. It causes not only marked tenderness but great itching so that the patient rubs the tail and rump, rupturing the vesicles, causing blood extravasations and retarding healing. The sores are primarily two or three lines in diameter but may widen and deepen under the friction. The surface may be red and angry and covered by a yellowish viscid discharge. When they heal they leave on the dark skin, round spots white and devoid of pigment. The itching during the acute stage of the disease, leads to generative excitement, frequent straining, contractions of the erector clitoris and ejection of urine. The animal appears to be constantly in heat, yet the absence of hyperthermia shows that there is no constitutional disorder nor central nervous affection. The disease appears to be purely local.

In the *stallion* after a similar incubation, the penis and usually the sheath become swollen and congested and if free from pigment there are dark points as described in the vulva. These points become firm, prominent, and finally vesicular and pustular. If continued in service the resulting sores may extend and prove protracted. In the absence of service they tend to heal about the fifteenth day. The eruption may extend on the scrotum, the inner side of the thighs, the throat, the lower surface of the neck, and the inside of the forelegs; on any parts indeed that may become soiled on mounting the mare. The meatus urinarius is red, swollen and angry, and shows a mucopurulent discharge. Generative excitement is shown by the more frequent rising and falling of the testicles, protrusion and retraction of the penis, and the frequency of urination.

In bad cases in both sexes the eruption encroaches more widely on the skin, and subcutaneous abscesses may form on the vulva, anus, tail, between the thighs or on the scrotal, mammary or inguinal regions. These are attended by extensive local swellings extending on the croup, or down to the hock, or forward on the hind limbs. In such cases there is more or less stiffness or lameness, with constitutional disturbance, hyperthermia, gastric disorder and emaciation.

Diagnosis. This disease is distinguished from dourine by the known absence of the latter from the locality, by the ready transmission, of the benign exanthema to cattle, by the entire absence of paresis or mental hebetude, by its rapid progress and early recovery, usually in fifteen days.

Treatment consists in thorough cleanliness, and the application of cooling, astringent, antiseptic lotions to the affected parts. Solutions of borax, boric acid, sulphate or chloride of zinc, permanganate of potash, corrosive sublimate, chlorine water, creolin, lysol, carbolic acid, or other such agent with glycerine will act promptly and well. In the severe cases with secondary abscesses the latter must be opened and the cavities treated antiseptically. Cooling laxatives and diuretics with salicylates or hyposulphites may also be desirable.

Prevention. No animal having sores nor discharge from the generative organs should be used for breeding. The owner of the animal infected by breeding has a good claim for damages.

VESICULAR EXANTHEMA IN CATTLE.

Definition : acute infectious, eruptive affection, usually of the genitals, in cattle, transmitted by breeding, and marked by congestion, papules, vesicles and pustules. Cause : infection by coition ; by contact, by gutter, by tail, by licking, in sucklings. Oxen and non-pregnant cows suffer. Symptoms : incubation 1 to 6 days, cows have swelling, red points, papules, vesicles and pustules on vulvar mucosa and adjoining skin, with profuse glairy discharge, and it may be fever : bull has matting of preputial hairs, frequent micturition, swollen sheath, red penis, vesicles, even necrotic ulcers and distortion. Immunization short, uncertain. Treatment as in horse. Prevention : seclusion and disinfection of animals, disinfection of talls, gutters, rubbing posts, trees, complete segregation of sick from healthy,

Definition. An acute eruptive affection, attacking particularly the generative organs of breeding cattle, manifested by congestion, papules, vesicles and pustules of the mucosa and skin and transmitted largely by copulation.

Causes. The one known cause is infection and the most common mode of communicating it is by the generative act. In this it agrees with the mild coital exanthema of horses, and the two affections have been considered as identical with each other and with cowpox (Trasbot). The similarity of the symptoms and the duration of the disease sustain this view, but, if correct it is probable that the affection has undergone a distinct modification which adapts the microbe especially to life in the genital canal, and to the maintenance of its virulence for a longer period, and finally, robs it of the power of producing any permanent immunity. Cowpox, though raised on the lips of the vulva, the inner side of the thighs and the abdomen where it can be easily reached by the tail, does not tend to implicate the genital canal, and it so quickly secures immunity, that neither male nor female would be likely to long convey the disease. Yet in both horses and cattle, experience shows that the acquired immunity of vesicular exanthema is very short lived.

Cattle, much more than horses, are liable to transmission of the disease by other channels than coition. Contact with the same soiled stall and gutter, the direct transference to adjoining animals by the soiled tail, and the greater frequency with which

they lick themselves and others doubtless contribute to this. The frequency of non-coital infection is shown in Numann's cases affecting the anus and sheath in oxen ; also in Fenner's long list of cases in 669 non-pregnant cows, in a number of cows in advanced pregnancy and in calves of a few weeks old (Friedberger and Fröhner).

Symptoms. In Cows. After an incubation of one to six, (or exceptionally ten) days, there occur swelling and redness of the lips of the vulva, and the vaginal mucosa with here and there dark red points, and an abundant clear, glairy discharge. The red points may become firm and papular, and an exudate beneath the epithelium transforms them into vesicles at first translucent but later yellow or brown as pus cells accumulate. The clear mucous discharge meanwhile becomes opaque and, as the pustules open, forming sores, yellowish and purulent. This discharge soils the tails and hips, drains out on the back part of the stall and the gutters and is carried by the tail to the udder and to other stock. Itching may be severe and the constant rubbing and switching of the tail, may irritate the sores and cause them to deepen or extend, beside favoring septic infection and local necrotic processes. In such cases there may be more or less fever, impairment of appetite, rumination and digestion, and of the secretion of milk. Abortion is not unknown though exceptional.

Symptoms. In Bull. The matting together of the long hairs of the prepuce may be the first indication of the disease, attended or followed by frequent micturition, or by standing with the tail raised and the accelerator urinæ pulsating over the ischiatic arch as if passing urine. The sheath appears swollen, the penis red granular, and angry, with red spots, and even the scrotum may be involved in the enlargement. The red points advance as in the female through the different forms of papule, vesicle and pustule to the formation of sores which are more or less red and angry. There may be an indisposition or even inability to protract the penis, and the bull may be tardy to serve, or in case the organ is erected and used, blood may ooze from the respective sores. The disease usually lasts about two weeks, but may recur a few weeks later in connection with a new infection. In aggravated and prolonged cases there may be local septic and

necrotic processes, and general fever and digestive disturbances. Distortions of the penis may render the subject permanently useless as a sire.

Treatment is essentially the same as in the horse. Antiseptic and astringent solutions (sulphate of zinc, iron or copper (2:100), nitrate of silver, carbolic acid, corrosive sublimate, lysol, creolin, etc.,) may be used to advantage. Laxatives and diuretics may be called for.

Prevention. This would require the temporary seclusion and final disinfection of all animals attacked, the thorough cleansing and disinfection of the stalls and gutters, as well as all posts, trees and fences on which the animals rub themselves. The attendants should use no combs, brushes, sponges nor rubbers indiscriminately on the sick and healthy. They should disinfect their hands and, if necessary, their clothes after having handled the sick and before handling or milking sound animals. An obvious precaution is to place the sick and healthy in separate buildings and yards.

INFECTIOUS GENITAL CATARRH IN RABBITS.

Freidberger and Fröhner record a very contagious catarrhal affection of the vulva and prepuce in high bred rabbits, and conveyed from animal to animal by sexual congress. The characteristic symptoms were extensive swelling of the parts and an abundant mucopurulent discharge.

Injection of the genital passages with a sulphate of copper solution (1 to 2 per cent) for several weeks in succession effected a cure.

INFECTIOUS ULCEROUS STOMATITIS IN LAMBS AND KIDS.

Prevails in France Predisposing causes, bad hygiene. Specific cause, streptococcus. Pathogenesis, to lambs and kids through sucking, drinking, feeding; inoculable on Guineapig, not rabbit. Lesions: like thrush, congested patches, with curd like concretion; epithelium, leucocytes, microbe; in bad cases gastro-enteritis hepatitis, or pneumonia. Symptoms: hyperthermia, refusal of teat, froth on lips, dulness, debility, emaciation, death in 15 days; in mouth, concretions and red raw sores; cough, hurried breathing, diarrhœa; subacute cases have little fever, or infection. Mortality 8

per cent. in acute cases, none in subacute. Prevention : separation and disinfection of fold Treatment : antiseptics to mouth; borax, sulphites, chlorate of potash, sodium salicylate, iron chloride, copperas, silver nitrate. Feed through tube or syringe. Internally, sulphites.

This is found in all parts of France, as described by Besnoit.

Causes. The predisposition, as in the case of many other fatal internal inflammations, depends largely on faulty hygiene, foul, ill ventilated, hot, close, damp, overcrowded folds, without sunshine, drainage or free access to the outer air.

The *specific cause* is a large coccus, isolated or in chains of 2, 3, 4 or more (streptococcus), and found abundantly in the pultaceous buccal exudate, and in the internal lesions developed by autoinfection. It grows readily in bouillon in the form of a precipitate in the bottom of the liquid, in gelatine and glucose in very small, thin colonies, and on potato in creamy masses. It is stained by anilin and by Gram's (iodine) preparation.

Pathogenesis. Inoculable on the Guinea pig, it is harmless to the rabbit. It is readily conveyed to lambs and kids by inoculation, or through the teats, by licking, drinking, feeding, etc. Acute cases are very virulent, subacute very little so. Isolated cases of the latter are not uncommon.

Lesions. The buccal lesions are like those of *thrush* (*muguet*). The mucosa is more or less congested and reddened with raised patches, of varying extent, of white, curdy looking concretions, composed of excess of epithelial cells and leucocytes with an abundant exudate. The specific coccus is present in great numbers and often in almost pure cultures. The internal lesions found in the more acute and fatal cases, are mostly in the form of gastro-enteritis, hepatitis and pneumonia, containing the specific cocci, and due to the inhalation or swallowing of the virulent buccal products.

Symptoms. In the *acute cases* an intense hyperthermia is the first symptom, speedily followed by refusal to suck, the accumulation of froth around the lips, great dulness and lassitude, prostration, rapidly advancing debility, and emaciation, and death in 15 days in a condition of marasmus and inanition. When the mouth is opened the curdy white or yellowish white patches of exudation are seen with an abundant glairy mucus, and occasionally red, raw, angry patches from which the morbid growth has

been detached. The symptoms of pneumonia or of muco-enteritis are usually in evidence, with or without diarrhœal or dysenteric discharges. Acute cases are extremely contagious.

In *sub-acute cases* the lesions are confined to the buccal mucosa, as curdy white, raised concretions with ulcerous surfaces, frothy lips and refusal of the teat, but without marked hyperthermia or constitutional disturbance. Contagion is little marked, the disease appearing enzoötically in flocks, or even as but one case in each of several flocks.

Mortality. Eight per cent. of acute cases may perish, but the sub-acute nearly all recover.

Prevention. When the disease has appeared in a flock, or in its vicinity, the mouths of the lambs should be examined daily and affected subjects and their dams carefully separated. A thorough disinfection of the fold and mammæ is imperative.

Treatment. Use antiseptics on the mouth, selecting the non-poisonous articles. Borax may be rubbed freely on the patches. Solutions of sulphite, bi-sulphite or hyposulphite of soda (.1 oz. to 1 qt.) may be used at frequent intervals on the mouth and mammæ; chlorate of potash (5 : 100), sodium salicylate (1 : 100), iron chloride (1 : 100) may be used. Ulcerous patches may be cauterized by iron sulphate, silver nitrate, or the potential cautery. Besnoit advises mercuric chloride (1 : 1,000) for cutaneous lesions, but this would be unsuitable for either mouth or lips. The strength must be sustained by the dam's milk given through a tube or syringe. For gastric lesions the sulphites may be given.

CONTAGIOUS ABORTION.

Definition : Premature expulsion of non-viable fœtus. Synonyms. Susceptible animals : cows, goats, sows, mares. Accessory causes : Ice cold drinks at periods of ovulation, frozen aliments, ice cold bath or rain storm, mechanical injuries, unwholesome fermentescible food, indigestible foods, close stabling, heavy milking, early breeding, inbreeding, stagnant water, ergot, smut, vegetable irritants or ecbolics, constitutional diseases, irritation of generative organs, death of fœtus, urinary calculi, odor of carcass or carrion, contagion, experimental infection between cows, between mare and cow, relation to omphalitis. Nocard's streptococci and bacilli, Bang's bacillus with best growth in two distinct grades of oxygenation, Galtier's observations, bacterium of the Colon group in America, infection of the

calves (pneumonia). Various kinds of infectious abortion. Acquired immunity, variable in different forms. Immunized cow still infecting. Symptoms. Lesions. Abortion in mares. Therapeutics. Prevention: Protection of a sound herd, guarantee with purchase, a government control, precautions with male or unimpregnated female, with pregnant. Extinction in a herd, separation, disinfection, disposal of abortion, manure, urine, disinfection of animal, of genital organs, precautions as to service, new born, fields, ewes. Disinfectants, subcutem or in muscles.

Definition. The premature expulsion of the product of conception, before it is viable out of the womb. Strictly speaking, a parturition in which the offspring is so far matured as to be *viable* is a *premature parturition*, while if it is *non-viable* it is an *abortion*. Vulgarly, however, as applied to herds, the term is used for any early parting with the fœtus. In this wider sense infectious abortion is *the premature expulsion of the fœtus owing to an infectious catarrh of the womb, transmitted from one animal to another by the transference of the microbe.*

Synonyms. Infectious Abortion, Enzoötic Abortion, Epizoötic Abortion.

Animals Affected. Abortion is most common in cows, and less frequent in ewes, goats, sows and mares.

Forms and Accessory Causes of Abortion. It was formerly supposed that abortion in herds was mainly due to mechanical and chemical agencies acting injuriously on the system, and in adopting the explanation of a particulate, transferrable, infectious agent for the habitual widespead forms of the disease, we should not forget these accessory causes, many of which by themselves cause sporadic abortions without the intervention of an infectious element.

The free *consumption of ice cold water* by the healthy dam causes active gastro-intestinal peristalsis and vigorous movements of the advanced fœtus, which can be seen or felt in the right flank, so that this is often resorted to, to determine whether the animal is pregnant or not. If this is frequently repeated or carried to excess in susceptible animals it will at times cause abortions. As in all other cases of mechanical or physiological disturbance, this is most operative at those periods of constitutional changes, which, if the animal had been unimpregnated, would have brought about ovulation and œstrum.

The greedy ingestion of *cold aliments*, like *frozen roots*, or green vegetation covered with *hoar frost*, may have a similar action, the more so that such aliments are extremely fermentescible and liable to cause tympany and undue pressure on the gravid womb.

Even without ingestion, exposure to *cold rain or snow storms* or the *plunging in ice cold water* tends to produce excessive peristalsis and fœtal movements and thereby prove injurious.

Mechanical injuries to the abdomen as *crushing by a gate*, *kicks*, *hooking with horns or tusks*, *hounding with dogs*, *riding of each other when a cow is in heat*, are liable to produce congestions, detachment of fœtal membranes, and even death of the fœtus.

Very *fermentescible foods* like those following a wet season or *bad harvest*, or those made of the leafy albuminous plants like lucerne (alfalfa), sainfoin and clover, act injuriously in the way of causing tympanies and compressions, but it must also be recognized that we are here dealing with fodders abounding in bacterial ferments, and that some of their products may act physiologically as ecbolics, even if the bacteria themselves do not colonize the genital passages as infections.

Insufficient food or very *innutritious forage*, too *close stabling*, *heavy milking*, *early breeding* (dam or sire), *inbreeding*, are liable to lower the general stamina and throw the system more open to the action of other factors.

Stagnant, *corrupt drinking water* has been charged with causing abortion, and the trouble has ceased when it has been withdrawn, but it is difficult to estimate its value in different cases—disturbing digestion, fermentation, poisoning, or introducing of actual infection.

Ergoted grasses and *smut* in maize, wheat or oats, have been often charged with wide spread abortions, and though each has in turn been administered in large and continuous doses without causing abortion, this does not invalidate the many cases in which it manifestly had that effect, nor does it show that obstetricians have been mistaken in their almost immemorial trust in ergot as an ecbolic. It must be recognized, however, that grown under different conditions, of sunshine and shade, and harvested at different stages of its development, ergot varies greatly in its physiological action, as it does also from having been overkept and thus one specimen is effective or dangerous while another is absolutely ineffective.

The specific action of the alkaloids in determining contraction of involuntary muscular fibre, is seen alike when used medicinally and when acting as an accidental toxic agent in causing spasms, nervous torpor or paralysis, delirium ; gangrene of the feet, tail or tip of the ear ; or contraction of the womb and expulsion of its contents. Like other agents mentioned ergot is at times an active ecbolic when coöperating with other similarly operating agencies.

Irritant vegetables of various kinds are to be dreaded. Such as act on the bowels and kidneys, keeping up a constant diarrhœa and diuresis, are quite liable to cause abortion in susceptible subjects. *Savin*, *tansy* and *rue* have obtained a bad reputation in this sense.

Cotton root bark is an active ecbolic, but is not likely to harm animals unless deliberately administered.

Impaction of the rumen or manifolds, and *obstinate constipation* with the resulting straining or compression must be named as an accessory cause.

Severe *constitutional diseases*, especially the infectious fevers, *aphthous fever*, *lung plague*, *rinderpest*, etc., are occasional causes of abortion.

It may be brought about by direct irritation of the womb, as in *ovariotomy* or *dilatation of the cervix*, or again by *death of the fœtus* through *twisting of the umbilical cord.*

Where cattle are subjected to dry winter feeding, in the absence of ensilage or roots, on the magnesian limestone, small *calculi in the kidney* are almost the rule in winter and the irritation caused by these conduces to abortion.

Nor must mental influence be altogether ignored. Cows and even mares are exceedingly sensitive to the smells of *decomposing animal matter*, as seen every day in the vicinity of *abattoirs*, or in case the *carcass* of one of their fellows has been opened, and not infrequently in connection with *carrion*, and the occurrence of abortions after such excitement has been frequently noticed. Cases are on record of specially susceptible cows having had active labor pains, from being with another cow at the time of parturition. Shepherds have noticed that violent thunder storms have been followed by numerous abortions in the flocks.

The Contagious Form. Any of the usual causes of sporadic or accidental abortion may coöperate with the true infectious element and give unusual energy to it, yet it is of the utmost im-

portance to recognize the contagion as the essential cause in all cases in which it is present. This can usually be done by a careful inquiry into the history of the outbreak.

When a herd has been continuously healthy up to the date of the introduction of a cow brought from a herd where abortion has been prevailing, and when following her arrival, one after another of the original members of the herd abort, without any apparent cause in the way of change of feed, water, barn, stalls, or general management, the evidence of the introduction of contagion by the cow in question is very circumstantial and forcible. If pregnant cows standing next to the new cow, or near to her, abort, the argument for contagion is still further corroborated. If the trouble continues in the herd year after year, attacking fresh animals some months after their purchase, the case becomes still stronger.

Or if a cow from a healthy herd is sent to a bull, that has been allowed to serve an aborting cow, and if the resulting pregnancy in the first cow is terminated by abortion, and if this is followed by successive abortions in the previously healthy herd, we may reasonably conclude that we are dealing with the infectious form. If no other appreciable cause for the trouble can be found we may reasonably conclude that this was the channel for infection.

Or a bull is brought from a herd where abortions have taken place, and some time after his arrival, the cows of the previously sound herd begin to abort, the first cases being in those that the new bull has served. The occurrence is manifestly the result of contagion.

Or a newly purchased cow aborts, and is disposed of in consequence, and another cow, placed in the same stall, in due time aborts also, and others follow in succession, especially those that stood next or near to the stall in question. Everything points to an introduced infection.

An indefinite number of other exemplary cases might be adduced, each varying from the last, but all agreeing in this, that the evidence of infection stands out prominently and unmistakably. The infecting material may have been carried by the tail, tongue, soiled stall, gutter, rubbing post, fence or other object, yet the fact of contagion can be demonstrated with reasonable certainty.

These conclusions have been repeatedly affirmed by actual

experimental transmission. The Scottish abortion committee found that healthy pregnant cows often escape, though standing near or even next to an aborting one, but that when a piece of cotton wool was inserted into the vagina of an aborting cow for twenty minutes and then transferred to that of a healthy one, the latter invariably aborted. Galtier found that when the infecting vaginal mucus of the aborting cow was transferred to the same passage of the healthy pregnant one, abortion took place in the latter. He succeeded in conveying the disease in this way from cow to sow, ewe, goat, rabbit and Guinea pig, and found that growth in the body of the rodent intensified its virulence, so that it could then be successfully inoculated on the mare, bitch and cat.

Bang subjected two cows, from healthy herds and three months pregnant, to repeated injections of the products of the culture of his abortion bacillus in serum glycerine bouillon. Three injections were made on April 14, May 23 and June 4, and on June 24 one cow aborted. The other sickened and when killed was found to carry a dead fœtus. Pure cultures of the abortion bacillus were found in the fœtal membranes and liquids of both animals.

Casual Infections. In a case which came under the observation of the present writer, a family cow kept in a barn where no abortion had previously occurred, was taken for service to a bull in a herd where abortion was prevailing, and though she was only present at the latter place for a few minutes, she aborted in the sixth month.

Another cow from the same aborting herd, was taken into another herd at a distance of two miles, where abortion had been unknown up to that time, and some months later the cow standing in the stall next to her aborted. The remainder of this herd was sold soon after, so that the further progress of the disease was not traced.

Tobiassen quotes the case of a cow from an aborting herd, which calved a fortnight before the regular time. The calf was at once sent to another farm where no abortion had occurred, and placed in the same building with the pregnant cows, all of which later aborted. The outbreak thus started lasted for several years.

Jansen as quoted by Sand, reports the case of a cow from an aborting herd having been taken into a herd that had been previously quite free from the disease. Soon after her arrival she

aborted, and later, cows in the same herd aborted. The owner kept the matter secret and sent his cows to a neighbor's bull for service, with the result that for two years abortion prevailed among the cows served by this bull.

J. R. Jansen reports that a cow brought from an infected farm, for fattening purposes, proved to be pregnant and finally aborted, and that 24 of the pregnant cows on the same farm aborted in the same year.

Mörck relates how a cow that had aborted a fortnight previously was taken to a farm where abortions had never been known. She aborted during her next gestation and so did the rest of the herd, 9 in number.

Christensen records the occurrence of a general abortion in a previously healthy herd, members of which had been sent for service to the bull of a neighboring aborting herd.

Nygaard reports that a bull from a healthy herd, but which had been allowed to serve some cows from a neighboring infected herd, was sold to go on a previously healthy farm, where he was put to 14 cows only. Of these 12 aborted, and the complement of the herd, beyond the 14 cows, put to another bull, remained well.

In each of these cases the environment of the animals and the conditions remained the same, the one appreciable cause of the outbreak being the contact with an animal from an aborting herd.

Experimental Infection. Braüer led the way by transferring in 11 instances the vaginal mucus of aborting cows to the vagina of healthy ones. The resulting abortions occurred from the 9th to the 21st day after inoculation. Lehnert repeated this on two cows, the abortions taking place on the twelfth and twentieth days respectively. Trinchera transferred the purulent vaginal mucus of an aborting cow to the vaginæ of healthy ones, determining in the latter, in 9 to 13 days, abortion attended by suppurating vaginal catarrh. He secured similar results by using the material scraped from the surface of the chorion of an immature fœtus.

The Scottish Commission (Woodhead, McFadyean and Aitkin) placed a healthy pregnant cow in an aborting herd, and on two successive days lodged in her vagina for twenty minutes a piece of cotton wool soaked in the vaginal mucus of a recently aborted cow. Within a month abortion threatened, and on the seventieth day a seven-months calf was dropped.

In a second experiment a cow, six months in calf, was taken from a healthy herd and placed in an aborting dairy herd and a quantity of vaginal mucus from a cow that had recently aborted was injected under the skin of the vagina. She calved prematurely at the end of the eighth month of gestation.

Williamsen, while treating a herd for abortion, took a piece of the afterbirth of one of the aborting cows and rubbed it on the vagina of a healthy cow of his own, which had a habit of carrying her calf fourteen days over time. Five days after she had premature parturition.

He took a fragment of the fœtal membrane from the cow just named and rubbed it on the vagina of a pregnant cow condemned for tuberculosis. In seventeen days the cow aborted.

Though not started as an experiment, the writer may name the general extension of abortion from one or two cows in a tuberculous herd after they had been tested with tuberculin. Cases of this kind are doubtless much more common than has been supposed.

Kilborne and Smith developed suppurating vaginal catarrh in cows and mares by inoculating them with the artificial cultures of bacilli obtained from the vaginal secretions of aborting mares.

Turner made cultures from the ovaries of aborting mares, and others from the genital organs of foals suffering from omphalitis, and produced abortions in pregnant mares by injecting with the same.

Pathology. Bacteriology. Franck attributed the disease to *leptothrix vaginalis*, but subsequent observers failed to substantiate this.

The Scottish Abortion Commission isolated five different bacteria from the abortion membranes and vaginal mucus, but failed to identify any one of these as, by itself, capable of causing the disease.

Nocard found in the fibrino-purulent matter between the chorion and womb in aborting animals a micrococcus occurring singly or in chains, and a short, delicate bacillus isolated or in pairs. From the absence of evil effects between pregnancies he opines that the germs grow in the membranes only, and do not affect the womb nor the general system. He recognizes, however, that they survive in the womb from one pregnancy to another in the complete absence of these membranes. In 1894

Nocard found the bacillus coli commune in an aborted calf, which had died *in utero* and precipitated abortion.

Galtier, on the contrary, conveyed the disease variously by the inoculation or feeding of the milk or abortion membranes to sheep, goat, pig, rabbit and Guinea pig, and accordingly claims that the general system of the pregnant animal is infected, and that the germs can be conveyed through the blood to the womb. In deducing this from feeding experiments he appears to make too little account of the ready infection through proximity of the anus and vulva.

Chester, of Delaware Agri. Experiment Station, found in the fœtal membranes of aborting cows a bacillus, which in form and habits of growth closely resembled the bacillus coli commune. In the fermentation test, however, it showed a marked difference.

Inoculated on rabbits it did not prove fatal. Injected into the vagina of a healthy pregnant cow it caused slight catarrhal discharge for four or five days, but the calf was carried to full time six and a half months later.

Bang found in aborting cows, between the womb and fœtal membranes, a considerable odorless, gelatinoid, liquid exudate, and some pus cells. There was active catarrh of the uterine mucosa which often carried the disease over into the next pregnancy. In the exudate he found a number of very minute non-motile bacilli (1 to 3μ), which stained readily with aniline colors, excepting in a vacuole or nucleus which was less deeply pigmented. This bacillus grew well in serum-glycerine bouillon, and more sparingly in serum-gelatine agar. In the latter it showed a remarkable peculiarity which serves to identify it readily from other microbes, in two successive zones of luxuriant growth at two different depths, with an intermediate clear zone, in which little or no growth took place. It seems to prefer a greater or lesser supply of oxygen (21 or 90 : 100) without being able to adapt itself to the intermediate condition. As already stated it produced abortion in the cow in 21 days after injection into the vagina. It also induced uterine catarrh and abortion in ewes, goats, rabbits, Guinea pigs and mares when injected into the vagina. From the vagina it usually reaches the womb, but not always. In several cases in which it was injected subcutem or intravenously it caused hyperthermia, and was later found in abundance in the interior of the womb and fœtal membranes, and

in the bowels of the fœtus. The microbe is, therefore, capable of living in the blood and affecting the womb by whatever channel it may enter the system.

V. A. Moore and the present writer made a series of experiments at the New York State Veterinary College. In the fœtal membranes and uterine mucus of a number of cows, aborting in different parts of the State and therefore long distances apart, we found a bacillus that in form and culture-experiments closely resembles bacillus coli commune. This was nearly always in pure cultures, and in the few cases showing other microbes, these were only such as inhabit the healthy vagina. Our bacillus was never found in the womb nor fœtal membranes of cows that had calved at the full period in healthy herds. It agreed in most respects with the bacillus found by Chester, but differed somewhat in fermentation tests. It differed also in proving fatal to rabbits when inoculated on these animals. Injected in the form of cultures into the vagina in three old pregnant cows it continued to live on the mucosa, producing more or less catarrh and mucopurulent discharge in the different cases, yet all three carried the calf to full time, one having calved on the 123d day after injection, the second on the 167th, and the third on the 190th.

The results obtained at the Delaware College Experiment Station and the New York State Veterinary College, do not differ so seriously, as either one does from those obtained in Europe, by Nocard, Bang, and the Scottish Abortion Committee. The facts that the same germs were found, either in pure cultures, or exceptionally, along with the normal microbes of the healthy vagina, in the womb, and fœtal membranes of every aborting cow, that they were never found in the healthy cow which had calved at full time, and that the generative passages were the seat of a catarrh, alike in the cows that aborted and in those that were inoculated with the abortion discharges, but did not themselves abort, are all but conclusive that this microbe is the essential cause of the abortion.

The single objection to this view, namely that the inoculated cows did not abort, is explained by the fact that in the New York abortions it is the rule that the calf is carried six or seven months from the date of impregnation (the date of the presumptive infection) to that of abortion. It is to be further borne in mind that our experimental cows were old, and may have passed through

the disease before and become in some measure immune, that they were dry during the experiment, and were subjected to no extraneous excitement that would predispose them to abort. The presumption is that had the experiments been started earlier in the gestation, the abortion would have come in due time. The microbe maintained its hold on the mucosa and continued to advance up to and beyond parturition.

Another distinction of the European abortion, is in the presence of the microbe in European form in the digestive organs of the calf, and that the viable calves of infected cows are liable to die of intestinal disorders a few days after birth. Galtier, the Marquis de Poncius and Pry insist strongly on this. On a farm on the estate of the Marquis, where abortion had prevailed for twenty years, the calves of infected cows show at birth, or very shortly after, symptoms of broncho-pneumonia and of a complication of nervous disorders. They are breathless, wheeze, discharge from the nose, cough, scour, have convulsions or other nervous trouble. A large proportion of such calves die; and their lungs are found in part red, consolidated and devoid of air and the bronchia contain a mucopurulent product. Lesions denoting inflammation of the serosæ of the lungs, liver and intestines are common. This coincidence of a fatal disease in many of the surviving calves is exceptional among the aborting herds of New York.

In noting the evidence of a wide difference between the prevalent American and European forms of abortion in cows, one should be prepared to go farther, and accept if need be, still other distinctive forms in each of the two continents. Any catarrhal condition of the uterine mucous membrane, is a recognized hindrance to conception, and cause of abortion, and we must recognize that the forms of invasion of the womb by pus microbes are as numerous as there are irritant germs capable of living in the membrane. The question as to how many of these can produce contagious abortion is to be determined by the susceptibility of the membrane to irritation by each germ, and whether the latter can retain all its power of survival and virulence in passing from one animal to another. The presumption is, therefore, in favor of a variety of forms of contagious abortion, each due to its own specific microbe or microbes, rather than of a single unvarying type

of the disease. In some the affection appears to be a purely local one (American), the microbe being confined to the genital or genito-urinary mucosa, whereas in others the microbes (Bangs', Galtier's, etc.) live also in the blood, producing a general infection.

Two great types at least have been demonstrated in Europe, and one in America. Whether future investigation shall show the presence in one of these continents of the types as yet only known in the other, remains to be seen. If the particular forms should turn out to be limited to different continents we are at once confronted with the necessity of an international sanitary quarantine, and inspection. Matters are bad enough now in our dairying districts, but if we are to be open to the importation of new types of abortion, which do not mutually immunize against each other, but which may be taken one after another in succession through a series of years, they may easily become incomparably worse.

Acquired immunity. The question of persistent abortion year after year, in the same animal, is most important. If a first contagious abortion entails a second, a third, a fourth and a fifth in the same animal, in as many successive years, then manifestly the preservation of such animal is a most wasteful economy, altogether independently of the danger of her transmitting the infection to healthy stock. If, however, she herself becomes immune after a first or second abortion, it may be profitable to retain her for milk production or breeding, provided that she can no longer infect susceptible cows with which she must come in contact.

Acquired immunity of the individual cow is the rule after one or two abortions caused by the microbes with which we are at present most familiar. There are exceptions to this rule due to special nervousness and excitability of given cows, which tend to a indefinite repetition of the abortion, under the stimulation of pregnancy, of the continued presence of the microbe, or of some local disease (tubercle, tumor, parasite, etc.) of the ovary, womb or peritoneum. Yet statistics show that this only applies to a small proportion of cows and these the most nervous and excitable. The tendency toward insusceptibility to the deleterious action of the germ, which may still be present, is in the cow as a rule greater than the disposition toward a nervous encrease of the susceptibility. The difficulty of reaching a conclusion on

this point depends on the fact that stockowners very commonly dispose of aborting cows, and as the freshly bought cows are usually attacked sooner or later, it is too confidently assumed that the old cows would also have continued to abort had they been retained. Many years ago, however, observant New York dairymen had noticed that the same cow rarely aborted over three years in succession, and in the great majority of cases not over two. The owner of a large herd, who has had much experience with the disease, assures me that the rule has been, that a cow did not abort with him a second time. The continuous abortion in a herd was mainly among newly purchased cows and others that had not been previously attacked, including heifers carrying first calf. The same is in a measure true of the European abortions.

Nocard says that after three to five years there is an acquired immunity. Only heifers and the cows that have been recently bought in, abort.

Pemberthy, speaking of England, says that in case of repeated abortion in the same cow, the calf is carried longer each successive year until it comes to its full term.

Sand, in his symposium of the experience of Danish veterinarians, says it is quite exceptional that a cow should continue to abort, but outbreaks of abortion disappear spontaneously if no new cows are brought in.

Bang refers to a herd of 200 head of which 83 aborted in their first pregnacy, and of these only 20 aborted in the second, while 7 failed to breed. Counting the latter as having aborted, this amonnts to less than one-third, while over two-thirds of the cows that aborted in the first gestation carried the calf to full time.

Paulsen quotes the case of a herd of 16, 7 of which aborted after service by a bull in an aborting herd. One of the seven was sold, but the remaining six all went full time in the following year.

Mörck records the case of a herd of 16 cows, the majority of which aborted in the same year. All the aborting cows were sold and fresh ones purchased. Next year the new stock aborted together with some of the cows that had been held over. He continued this course for eight years without any improvement, and then decided to keep the aborting cows as well as the others. In two years the affection disappeared from the herd.

Such small herds, in which all become early infected, and in which there is no further opportunity for the infection of susceptible animals (cows not yet infected, heifers in first gestation, new purchases), furnish a better opportunity than do large herds, to trace the acquirement of tolerance.

In a question of this kind, one must allow for variations in the different types of abortion caused by various microbes, but in the forms with which we are familiar in Europe and America, the acquired tolerance of the individual can be counted on with great confidence. It has indeed been largely traded upon by purveyors of antiabortion nostrums who promise to cure the individual cow, to which alone their drugs are administered. Two evils result: the stock owner's money is paid for that which, unaided, nature would have accomplished for him; and attention is withdrawn from the real necessity of the case, the prevention of the infection of freshly introduced animals. The nostrum vender thus secures for himself a growing market, as the yearly production of fresh cases in the same herds, appears to demand a constant use of the agent which appeared to work so well in the earlier ones.

Immunized animal still infecting. The cow that no longer aborts is not, therefore, a safe member of a herd. As an individual animal she has become resistant to the pathogenic influence of the germ, she is invulnerable to it to the extent that she no longer aborts, but her system and generative passages have acquired no such active bactericidal power over the microbe as to lead to its speedy destruction. The genital passages, once colonized, continue to be a field of growth of the bacillus long after its power to cause abortion in that particular animal has passed. Analogous cases can easily be quoted from the field of pathology. The horse that has apparently recovered from dourine still conveys the disease to others with which it has sexual congress; the recovered syphilitic person is by no means eligible for marriage; the recovered pig continues to carry the infecting swine-plague bacillus in its air passages. In short, it is the rule that the immunized animal can with safety to itself carry a germ that readily infects its non-immunized fellows.

In the case of infectious abortion this is one of the most dangerous elements, as the apparently healthy recovered cow receives no attention in the way of separation and disinfection, but is

allowed to spread the infection through the bull that serves her in common with other cows, and also by being sold into new and healthy herds.

Symptoms. Contagious abortion sometimes takes the form of temporary sterility, the animal taking the male at frequent intervals, but failing to conceive. If conception takes place, the abortion is usually deferred until the fœtus has attained a considerable development—in cows till the third or seventh month ; in mares till the fourth or ninth month ; in ewes or sows till the tenth week.

Often times premonitory symptoms are entirely unobserved. Usually there may be detected some heat and enlargement of the mammæ, with a decrease in the milk-yield, or a serous modification of the milk as in colostrum. Still more striking is a muco-purulent discharge from the vulva—opaque, white or yellow—in marked contrast with the perfectly clear, transparent mucus which appears in œstrum. The discharge may be densely white (in mares) or reddish, and may be accompanied by some swelling of the vulva and redness of its mucosa, which is dull, rough and granular, or even the seat of a papillary eruption. There is rarely hyperthermia or other constitutional disturbance, and in some cases the abortion is only discovered by the finding of the fœtus and its membranes in the gutter or pasture. The membranes are, however, not unfrequently retained, becoming offensively putrid. In other cases a muco-purulent discharge persists for a length of time, insuring sterility so long as it lasts, and causing ill health and emaciation. The fœtus is usually born dead.

The *Lesions* are confined to the generative organs. Bang, found an odorless, dirty yellow, flocculent, slimy and more or less watery exudate between the chorion and uterine mucosa involving the connective tissue between the chorion and arachnoid so as to render it thick and friable. These conditions were well marked in the French and English cases, and perhaps somewhat less so in the American, which are habitually slower in reaching the abortion. Bang found the uterine catarrh with the characteristic bacillus in cases in which the calf had been carried to full term, exemplifying the local presence and culture of the bacillus without the usual abortion outcome. The bacilli were occasionally, though not always, found in the body of the fœtus, and in some

cases the dead and mummified fœtus was found in the womb, which had not been stimulated to its expulsion. He even found the bacillus in the mummified fœtus, and still virulent, after an apparent seclusion of seven months.

ABORTION IN MARES.

This follows the same general course as does the disease in cows. In certain American outbreaks (Kilborne and Th. Smith) it has been traced to a bacillus like the colon bacillus, propagated in the womb and genital passages, and which produced suppurating vaginal catarrh in cows and mares on which it was inoculated. It is liable to occur, without premonitory symptoms having been observed, and to be followed by no marked sequelæ, so that, as in most cases in the cow, it may be looked on as a purely local infection. In England, Penberthy, found it to occur not earlier than the fourth month of gestation, yet he adduces several cases in which the first case in a breeding stud occurred in four weeks after the introduction of an infected mare from an aborting stud. In America on large breeding farms the introduction of the infection has proved ruinous, as many as 70 or 80 per cent. of the mares having aborted in the same season.

The conclusion is inevitable, that as in the case of cattle, the sire may become the means of transmission, and that the same measures of prevention are demanded. The fact that the affection is less widely spread or injurious, than in cattle, is largely due to the usual presence of but one, two or three breeding mares on a farm, so that there is little opportunity for a rapid extension of the infection. Multiply and encrease our studs of breeding horses, as cows have been in our dairying districts, and abortion, once introduced, would prove equally infective, spreading and injurious.

Therapeutics are useless in contagious abortion as the disease usually runs its course before any danger is suspected. If premonitory symptoms are observed, the abortion may sometimes be warded off for a time by secluding the animal in a quiet place and seeking to obviate labor pains by opiates and ounce doses of viburnum prunifolium.

Prevention. This is to be sought along two principal lines:

1st. The protection of a sound herd against the infection : and 2nd. The extinction of infection in a herd already diseased.

Protection of a sound herd. This requires the greatest possible care, because the infected animal usually presents the general appearance of perfect health, and there is no ready means of testing the presence or absence of the abortion bacillus. In purchasing a cow or mare in a public market the new owner may find her affected with this bacillus, and a serious danger to his whole herd. To protect the latter he must learn that the herd from which she came has had no abortions for several years before, and that the offspring for the different years are present in numbers corresponding to the dams. In the absence of this a certificate, and guarantee against infection of other animals by the one purchased, may well be demanded of the seller. A certificate from the veterinarian attending the herd furnishing the animal, may also be sought as evidence of the absence of abortion from the locality. Imported animals should be safeguarded in the same way but with even greater care, lest the microbian sources of new types of abortion, should be brought into the country. A guarantee of this kind might well be demanded by the Federal Government in the case of all breeding animals imported.

In case of failure to secure the most perfect guarantee with the purchased animal it would be worth the purchaser's while to seclude it from his valuable herd, and not to breed it with the other animals of his herd until it has been proved to be entirely free from infection. If bought for a sire it should be subjected to a course of disinfection of the sheath and penis : if for a dam and unimpregnated, antiseptic irrigation of the vagina may be made daily for a week, and the external parts, hips and tail daily washed with antiseptics. If very important to have her bred, secure, if possible, a male that is not to be used on other animals. If this is impracticable, let the sheath and penis, and surrounding skin be thoroughly disinfected as soon as the service has been had.

If the newly acquired female is pregnant, keep her by herself until parturition and, even if this takes place at full term, irrigate the womb daily for a week with a disinfectant, delay having her served for a month, and if she must go to the sire of the herd, subject him to thorough disinfection after service. It may be that she carries the germ but has become tolerant of it.

Extinction of Contagious Abortion in a herd. 1. Two separate stables, or compartments, not having a common gutter should be provided, one for the sound animals, and one for those that are known to be affected, or that are open to suspicion.

2. The cow or mare that shows symptoms of abortion, or that has aborted, should at once be removed to the quarantine stable, and her stall including the gutter and drain leading from it thoroughly disinfected. The whole stable should be whitewashed.

3. The aborted fœtus with its membranes, should be at once removed and burned, or boiled, or deeply buried after it has been thickly sprinkled with chloride of lime or other active disinfectant.

4. The manure from the infected stable should be taken into an enclosure into which no animals have access, and freely watered with a solution of sulphate of copper (6 : 100).

5. All in the quarantined stable and even those left in the general stable, should have the external generative organs, the hips, and the whole length of the tail washed once or twice a day with an antiseptic solution. Carbolic acid or creolin (2 or 3 : 100) makes a very safe and convenient agent, but copper sulphate (6 : 100) or mercuric chloride (2 : 1000) may be substituted. The colorless mercuric solution though effective and inodorous, has an element of danger in it, when left in barns in large quantities.

6. Cows or mares that have just aborted, should have the uterus irrigated daily for a week with a disinfectant solution. Carbolic acid (1 : 100) with 1 sodium carbonate) or creolin (1 to 2 : 100) or mercuric chloride (1 : 1000 with 1 hydrochloric acid) are good examples. Use a long rubber tube and funnel. They should not be bred until all discharge has ceased.

7. It is a good practice to keep a separate sire for the aborting and quarantined suspected animals, but the bull for each herd should, after each service, have his sheath injected with the disinfectant liquid, the orifice being held and the liquid pressed into all parts and finally the skin around the sheath must be well washed with the same.

8. All cows or mares in an aborting herd, or one from which aborting animals have been removed within a year, should, after delivery, be injected with a disinfectant for one week exactly as if they had just aborted. This will guard against the danger

from animals that carry the germ, but have become so tolerant of it that they no longer themselves abort.

9. New born animals brought in from other herds should be sponged all over with one of the above-named disinfectant solutions before being added to the herd.

10. In case the breeding animals go to pasture, separate fields should be furnished for the aborting and suspected ones and those supposed to be sound.

11. Breeding ewes, goats and sows should be excluded from all pastures occupied by suspected herds or those under treatment. The fields should further be cleared of rabbits as being susceptible to the infection and capable of keeping it up and transmitting it.

12. It is important to reserve the herd sires for the exclusive use of the home herds. Where this cannot be done, disinfection of the sheath and penis should be practised immediately after each service.

Attempts have been made at different times to destroy the bacillus by subcutaneous or intermuscular disinfectant injections. Bäuer used in the cow a 2 per cent. solution of carbolic acid, of which he injected two Pravaz syringefuls under the skin of the flank every fortnight from the fifth to the seventh month. It was rather unreasonable to expect much from 10 minims of carbolic acid once a fortnight, even apart from the fact that this agent is converted into the inert sulphophenic acid in the body.

Lignieres followed in the same line by injecting into the bodies of the cervical or other muscles 10 cc (2½ drams) of a mixture of terpinol one part, olive oil nine parts. This was injected during pregnancy every second day for the first three months, every third day for the second three months, and every fourth day for two months. The claim for success is based on the alleged prevention of second and third abortions in the same animals, and this becomes rather shadowy when we consider that the rule with cows is that they do not abort a second or third time. We have met with veterinarians who claimed a splendid success with the single injection of the terpinol. Lignieres claims no such success with his long-continued treatment, as he had a percentage of abortions in every herd treated, and from first to last almost every animal in each herd aborted once, or was sold nymphomaniac. It is to be feared that the apparent immunity depended mainly on that tolerance which comes early in nearly every case to the aborting cow.

Blackhead. Definition : Infectious protozoan disease of cæca and liver and general toxicæmia. Microbiology : Amœba in cæca, liver, etc. Symptoms : Attacks young, dulness, drooping, ruffling, moping, anorexia, greenish diarrhœa, icteric or dusky mucosæ, blackening of gobble, etc. Lesions : Enlarged thickened cæca, epithelial degeneration and desquamation, exudation, mottled liver, with giant and round cells and amœbæ, degeneration, necrosis, caseation, no abscess. Diagnosis from bacteridian typhlitis or diphtheria. Treatment : Intestinal antisepsis. Prevention : Disinfection of buildings, yards, manure, seclude from other flocks, birds, flies, breed new flock on fresh ground from eggs of affected or other flock, set in incubator or under hens.

Synonym : Blackhead.

Definition. An infectious disease of turkeys (and hens, Chester), especially destructive to the young, due to a protozoon (amœba meleagridis), and characterized by inflammatory thickening of the walls of the cæca, diarrhœa, brownish, yellowish or greenish areas of degeneration of the liver, and congestion with blackish discoloration of those portions of the head which are uncovered by down or feathers.

Area infected. New England, New York, Delaware and other states.

Microbiology. The microörganism (amœba meleagridis) is found in the thickened walls of the cæca, in the exudate, in the lumen, and in the degenerating patches of the liver. The most common and simple form is that of a rounded body, varying slightly in form, and containing a group of very minute granules situated somewhat eccentrically. They may be enclosed in lymph spaces or less frequently in giant cells, or have portions of broken up cells adherent. Their size varies from 10 to 15μ. They may be stained by the following process: Harden in 95 per cent. alcohol saturated with mercuric chloride, then in the same with an equal amount of a 5 per cent. solution of bichromate of potash, and finally in Hemming's solution. After a day in these solutions they are washed for a day in running water, then treated with ascending strengths of alcohol, dried by passing through alcohol and chloroform, imbedded in paraffin, sectioned dry, and stained in Delafield's hæmatoxylin and eosin. The spherical or slightly oval amœbæ have a homogeneous, bluish red tint, feebler

and therefore distinct from the tissue nuclei. Near the centre in most a blue circular line shows the outlines of the nucleus.

Th. Smith compares this affection with the amœbic dysentery affecting the large intestine and liver of man, and notes these differences, that amœbic movements have not been observed in amœba meleagridis, and that hepatic abscess does not occur in the turkey. The indisposition of the bird to suppuration may perhaps account for the latter distinction. Smith found rounded organisms in the tubules of the cæca and flagellates in the lumen of the gut, but did not attach any importance to their presence. Bacteria were only found in the lesions in the solid tissues where the subject had not been killed and immediately examined, but left over night for examination in the morning. There was no constancy in their species as in the case of the amœbæ, all indicating that their invasion was post mortem.

Symptoms. The disease is most frequent and fatal in the young (1 to 4 months, exceptionally 6 to 10 months), and the symptoms vary much in different cases, according to the intensity of the disease and the relatively extensive implication of the different organs. Among the general symptoms are those of general suffering and ill health, dulness, spiritlessness, drooping of the head between the wings, a pendent condition of wings and tail, erection of down or feathers, separation from the flock, the bird moping alone and sitting much of the time. The more characteristic phenomena which are rarely sufficient to identify the disease unless it is known to be prevalent, are loss of appetite, a greenish diarrhœa, yellowish or brownish discoloration of mucosæ, emaciation which becomes extreme if the subject survives long enough, and more or less blackish discoloration of the protractile caruncle of the forehead and bare portions of the skin covering the head.

Lesions. These are characteristic, the cæca being greatly enlarged, the walls thickened by a yellowish submucous exudate, the epithelium disintegrated and desquamating, and the mucosa covered by a solid yellowish gelatinoid exudate arranged in superposed layers, while the contents are soft, pasty or of a greenish liquid appearance. The comparative stagnation of the contents as in the appendix of man appears to favor microbian infection. Amœbæ are especially abundant in the exudate into the submucosa

and in the lymph spaces. The liver changes show infective inflammation in the spots of mottling, followed by degeneration, and necrosis, the liver cells disappearing under the compression of the giant and round cells and the amœbæ, the surface over the affected parts is depressed, and the mottling may show a variety of colors, as brown, brownish red, or yellow, pale yellow, grayish or dirty white. The degenerating tissue may become caseated, but abscess appears to be unknown, though so common in amœbic dysentery of man. In the skin of the head the blackish color predominates; there is more or less congestion, capillary embolism, and tissue degeneration.

Diagnosis. Th. Smith notes three cases of diseased cæcæ without great thickening of their walls, and an exudate in the lumen having the general appearance of that seen in the amœba disease but with no amœbæ, only the bacillus coli communis. The odor was strongly feculent. In all three cases the liver was healthy. In one there was an abundance of tapeworms in the bowels. Manifestly the absence of liver lesions may be held to indicate the absence of the protozoan disease. VonRatz (Budapest) gives two similar cases, with many nematodes, those in the exudate being 8 to 14 mm. long.

Zurn describes the presence of the diphtheria of fowls in the intestines of hens, turkeys and palmipeds and sometimes confined to the cæca. It is characterized by great prostration and debility, and an offensively smelling diarrhœa at first pultaceous and mucous, later bloody, and followed by constipation, in which case cæca and rectum are ulcerated and blocked with the yellow croupous exudate. It lasts 2 to 3 weeks or even months, is subject to relapse, and sometimes occurs as a sequel to the diphtheritic affection of the head and throat. The absence of marked thickening of the cæcal wall, and of the protozoon, the presence of the diphtheric exudate in the lumen, and the croupous condition or congested appearance of the head and throat are distinctive. Th. Smith, however, refers to two cases, supposed to be of this kind, in which the walls of the cæca were greatly thickened, the result of reparatory inflammation following a slough of the mucosa. Siedamgrotzky also describes thickening of the walls of both cæca in a hen, the mucosa being covered with a thin pseudo-membranous exudation, without ulceration.

It would seem that the cæca of birds, like the vermiform appendix of man is very subject to invasion microbes, bacterian and protozoan, and should always be carefully examined in case of intestinal or hepatic disorder.

Treatment would be in the line of intestinal antisepsis with carbolic acid, salol, sulphurous acid, or the sulphites, with a laxative of castor oil, to carry these agents unchanged to the cæca, but no success has attended attempts in this direction, and the danger that comes from preservation of the infected animal, and consequent multiplication of the microbe would as a rule far more than counterbalance any probable recoveries.

Prevention. Moore has shown that the amœbæ, passed with the fæces, contaminate the food and water and thus actively propagate the disease, so that preventive measures must be mainly directed toward the purification of these infecting media. To be thorough, new ground must be secured on which no diseased turkeys have been, and through which no water from contaminated or suspected land can flow ; if necessary this must be closely fenced to prevent all ingress or egress, and on this ground we can place, turkeys from sound flocks or hatched from eggs obtained in noninfected localities. The eggs of infected birds can convey the disease(Curtice). The amœba is not known as a parasite of other birds than turkeys and hens, but if it should eventually be found to be so or to occupy any other animal body as an intermediate host, the local extermination of such host will become a necessary precaution.

When a new flock has been started in this way, the birds of the old flock may be fattened, killed and marketed, and as suggested by Cooper Curtice the grounds they have occupied may be secluded by fencing for one or two years, in the hope that the amœba will perish by this break in the chain of its life history. If this should prove successful with the land, the infection might be easily exterminated in the whole infected district or state.

The poultry buildings will require thorough disinfection. All manure and droppings must be carefully removed and the building whitewashed, using freshly burned quicklime and ¼ lb. of chloride of lime to the gallon of the mixture. The litter should be burned, and all nests, roosts, drinking vessels and troughs soaked with a mixture of sulphuric acid ½ gallon, carbolic acid

½ gallon, and water 20 gallons, (Th. Smith). The agents are mixed slowly in a vessel set in cold water. The same may be liberally applied on the surface, fences, etc., of the yards. Or quicklime, freshly burned, may be used freely on the yards holding the infected flocks (Moore). Mercuric chloride is dangerous. When the infected flock has been finally disposed of, the buildings and yards should be again thoroughly disinfected, and together with the field runs, abandoned for at least one year.

ASTHENIA IN CHICKENS.

Microbiology: bacillus of colon group. Pathogenesis to Guinea pigs: rabbits, pigeons and chickens refractory to artificial cultures. Treatment.

Synonym. "Going Light," Bacterial Infection of the Duodenum.

C. F. Dawson, in investigating a wasting disease of well fed Brahma fowls in Maryland in which there was no appreciable symptom except the gradual and extreme loss of flesh and weight found in all cases a catarrhal duodenitis, with the presence in the contents of a bacterium in pure cultures, and apparently allied to bacillus coli communis.

Bacteriology. The bacterium is 1 to 1.3μ long, by .5μ wide, with rounded ends, often in pairs. It does not stain in acid nor alkaline methylene blue, carbol fuchsin, nor alcoholic dyes, but stains easily in aqueous solutions of the same stains and by Gram's method. It is ærobic, facultative anaerobic, grows at 50° to 120° F. in acid or alkaline beef bouillon, with fœtor; in glucose, saccharose, or lactose bouillon with the production of acid; in milk causing coagulation; on gelatine, agar and potato. Growth in Bouillon ceased at 131° F. and sterilization took place at 135° to 140° F. Vitality was not lost under freezing nor drying. It was killed by a 1 per cent. solution of carbolic acid in five minutes, or by formaldehyde gas, but not by lime water.

Pathogenesis. Inoculated subcutem, in a *Guinea pig* caused death in 24 hours, with a necrotic œdematous condition of the adjacent tissues like malignant œdema, and containing the microbe. It was further found in the liver, spleen, abdominal exudate, heart, blood and lungs, but not in the kidneys.

It proved harmless to *rabbits* when injected subcutem, but fatal in 24 hours when thrown into the peritoneum. The lesions were severe duodenitis and omental inflammation, and the bacterium was found in the liver and duodenal wall.

Pigeons, rats and mice proved refractory to the artificial cultures, also chickens. The latter were injected subcutem, intra-abdominally and intravenously. Yet in view of the constancy of the lesion in hens with the microbe in pure cultures and the absence of all other morbid conditions, Dawson felt justified in attributing the disease to the microörganism. It is possible that his experimental hens had become immune from a previous attack, or failed in some condition of food or environment which is essential to pathogenesis, or finally there may possibly be some other infinitesimal microbe present which escaped observation, but which furnished the occasion for the coincident development of the colon bacterium.

Treatment. The indications are: the expulsion of the offending bacterium to be followed by nutritious, easily digested food, pure water and tonics. Dawson advises castor oil in dose of two teaspoonfuls, or calomel ¼ grain and repeated till purgation occurs; and to be followed by powdered fennel, anise, coriander, cinchona of each 30 grains; powdered gentian and ginger of each ½ dram; powdered copperas 15 grains. The addition of bismuth, pepsin, or orexin may be suggested.

APOPLECTIFORM SEPTICÆMIA IN CHICKENS.

Streptococci infection in hens. Most violent in pullets and hens; less so in capons and cockerels. Found dead, or sick 24 hours, ruffled, prostrate, neck limp, head drooped, indisposed or unable to rise, liquid icteric fæces, blood stained skin of neck and breast, death without agony. Lesions: Soiled anal feathers, plump carcass, skin pallor save neck and breast, extravasation of blood subcutem and intramuscular, mucosæ anæmic, peritoneal engorgement and effusion, enlarged, engorged liver, cell proliferation, degeneration, coagulation necrosis, gall bladder full. Enlarged congested spleen, degeneration. Congested swollen kidneys, epithelial degeneration, casts, streptococcus. Congested, consolidated bronchioles, alveoli, lungs, with microbe. Meningeal congestion, exudate. Bacteriology: Streptococcus, pure culture in lesions, size, chains, tetrads, staining qualities, culture media, colonies, action on sugars, acid, no gas, on milk no

coagulation, on gelatine no liquefaction. History: Stagnant water; no evident source of infection, buzzard, etc. Pathogenesis: Inoculation, intravenous or intramuscular, feeding. On duck, pigeon, rabbits, mice, dogs. Guinea pigs and sheep immune. Immunization by injection of sterilized cultures.

Under this name Norgaard describes a hæmorrhagic streptococcic infection which prevailed in 1901 in a flock of 200 to 250 Plymouth Rocks in Loudon County, Va. Forty birds died within six weeks, and later the mortality reached 200, death often occurring suddenly while feeding, or the birds would drop from their roosts in the night and be found dead in the morning. An outbreak has been seen in New York.

Symptoms. In the majority, (the pullets which died suddenly,) no symptoms whatever were observed, yet a certain number of the capons and cockerels were noticeably ill for 12 to 24 hours. In these the feathers were ruffled, the prostration extreme, the head drooped between the wings, the neck was weak and wobbling (limber neck), the wings and tail drooped, and the animal lay on its breast often unable to rise. Sometimes there was a passage per anum of liquid bile-stained mucus. Close examination of the neck and breast might detect hæmorrhagic discoloration of the skin, though the skin elsewhere was pale and smooth. Death takes place without a struggle.

Lesions. The anal feathers were usually stained, indicating diarrhœa; the picked carcass was plump and fat, with pale, healthy looking skin, except on the neck and breast, where it was discolored by extravasated blood; at such points the connective tissue and muscles were infiltrated with blood in areas of an inch in diameter, more or less, and with irregular outlines. The buccal mucosa was clear, pale and bloodless. The cavity of the body contained an abundance of sero-sanguinolent exudate, and the mesenteric veins were engorged. The *liver* was greatly enlarged and congested, the greater part of its substance being apparently composed of the distended blood vessels. The hepatic cells were granular and fatty, and if death had been delayed over twenty-four hours their outline became indistinct and the nucleus stained only faintly. In the foci of disease were clumps of round cells and leucocytes in and between the acini, and in still more tardy cases points of coagulation necrosis. The streptococcus was abundant in the necrotic centres, among the clumps of leuco-

cytes, in the parenchyma and capillaries. The *gall bladder* was usually distended with bile. The *spleen* had the large size and well developed Malpighian bodies that characterize the well-fed bird, but it showed still greater enlargement, combined with sanguineous congestion and circumscribed blood extravasations. With a hand lens were seen numerous round, semi-transparent points, the size of pin holes, which the microscope revealed to be centres of coagulation necrosis, surrounded by embryonic tissue. The centres consisted of a granular debris with abundance of the streptococcus. The *kidneys* were congested and swollen, and in cases that had survived two or three days there was granular degeneration of the epithelium of the tubules, and the lumen of the tubes contained casts with leucocytes and streptococci. The organism was also present in the capillaries. The *lungs* presented areas of congestion and consolidation, exudation into the walls of the bronchioles and the alveoli, and an abundance of streptococci in the lesions. In the cranium were a subdural exudate and meningeal congestion. For examination of the tissues they were hardened in specimens of alcohol of encreasing strengths, embedded in paraffin and stained with carbol fuchsin with a counterstain of methylene blue.

Bacteriology. The streptococcus was found in pure cultures in all the diseased centres except in the intestinal contents. Smear preparations were made from the blood, the abdominal exudate, the intestinal contents, the bone marrow, the cerebral exudate and the sanguineous effusions into the muscles.

The coccus is .6 to .8μ in diameter, in short chains of 2 to 8 cells, or in some media much longer. Involution forms are common, and Norgaard claims to have seen indications of fission in two directions to form tetrads. The organism stains in the usual aniline dyes, as well as by Gram's and Gram-Weigert's method. It is nonmotile, ærobic, facultative anærobic, grows in solid and liquid media that is neutral, or slightly acid or alkaline (not if strongly acid), best at 98.6° F., and slower at the room temperature. In alkaline peptonized beef bouillon in 24 hours it forms threads and balls on the sides and bottom of the tube, leaving the liquid clear. On agar there are formed small shining, grayish colonies 1.5 mm. in diameter with brownish centre and bluish periphery. With all the sugars it produces acid, but no gas. It

does not coagulate milk. On gelatine colonies are formed in four days, and there is no liquefaction.

History of the Outbreak. The outbreak commenced early in January, without any obvious occasion for the introduction of infection. There had been no chicken disease on the place for 25 years, and no chickens had been purchased except from a neighboring farm where the stock remained healthy. They had been fed on corn meal, wheat bran, wheat tailings and whole corn, together with scorched wheat from a burned barn. The poultry houses were clean and well aired, and after they had been closed there was no abatement of the disease. The water was from a stagnant pool receiving drainage from the stable, but this was no new condition and January is not the driest month with the foulest water. The suggestion may be hazarded that the infection may have been introduced by the usual infection bearer, the buzzard, or by some other wild bird, or mammal.

Pathogenesis. Among the *chickens* the most rapid and fatal cases were in pullets, then among the laying hens, while the capons and cockerels were less severely affected, and some survived from three to seven days.

Those *inoculated intravenously* as a rule sickened on the second or third day with a temperature of 110.7° F., and were found dead the following morning.

Some took injections of .25 to 1cc; in the *pectoral muscles* with impunity.

Chickens that had fasted 24 hours, took each daily for 3 days, a few cubes of bread soaked in a fresh bouillon culture. Death followed in four out of six, in from four to thirteen days from the beginning of the experiment. The birds gradually become listless, refused to eat, and remained quiet in a corner of the cage, with closed eyes and head drooping until it rested on the ground. Diarrhœa was frequent but not invariably present.

Chickens fed on the chopped up viscera of rabbits that had died of the disease perished in 3 to 10 days.

A *white duck* inoculated intravenously with .5 cc of peptonized beef bouillon culture, took ill on the 8th day, and died on the 12th. There was loss of coördination and use of the wings, temperature 109° F., and necropsy showed valvular endocarditis containing the streptococcus. A second duck sickened on the 11th

day, but recovered after three day's illness. Killed on 21st day, all cultures from heart, spleen and kidney remained sterile. Subcutaneous, intra-muscular and feeding experiments failed to produce the disease in ducks.

A *pigeon* injected with .5 cc. bouillon culture intravenously died on the fourth day with the internal lesions of chickens, and blood extravasations on head and neck. Intra-muscular injections and feeding experiments were fruitless.

In *rabbits*, *intravenous* injections of 1 to 1.5 cc. led in 24 hours, to temperature of 105.3° F., without impairment of appetite, or other marked sign of illness, and as a rule the subject was found dead next morning. *Intra-abdominal* and *intrapleural* injections kill in two to four days, and *subcutaneous* ones in three days. In addition to the lesions found in chickens, there is often bloody urine, a sanguineous lymph on and beneath the cerebral meninges and in the fourth ventricle, and deep congestion of the cancellated tissue of the vertebræ. Streptococci are abundant in the lesions.

In *mice intra-abdominal* injections of .01 cc killed in 48 hours, and *subcutaneous* injections in 2 to 5 days. The lesions were like those seen in birds, and streptococci were abundant.

In *dogs* after *intravenous* injection there was hyperthermia (103.6° and 104.1° F.) and lameness of the right fore shoulder joint, followed in 5 to 8 days by recovery. Feeding on the viscera of the diseased chickens, was followed by anorexia, and vomiting only.

Guinea pigs and *sheep* proved refractory.

Immunization. The injection into the wing vein of .5 to 2.5 cc. of bouillon culture, sterilized by heat proved protective to the chickens against inoculations of the virus while the check animals invariably died.

TUBERCULOSIS.

Synonyms. Definition : infectious disease caused by bacillus, attended by productive inflammation, tubercles, necrotic degeneration, caseation, fibroid change, calcification or ulceration. Animals susceptible : python, salamander, carp, fish, birds, rodents, sheep, goats, solipeds, cattle, dogs, cats, apes, swine, wild ruminants, kangaroo, carnivora, cage birds, civilized (housed) man. Geographical distribution : with dense population, Central Europe, Britain, Eastern States ; little in absence of cattle products—Scottish Western Isles, Iceland, Newfoundland, Greenland Highlands, Arctic North America, Northern Norway, Sweden, Lapland : open air life protects. Virulence : ancients records, modern, early antituberculous legislation, decimation of herds, researches of Vilemin, Gerlach, Chauveau, Semmer, Parrot, Tappeiner, etc. Bacillus tuberculosis : evolutionary changes, relation to actinomyces, 1.5 to 3.5μ by 0.2μ shorter and thicker in ox, solitary, exceptionally short chains, on blood serum may be filamentous, staining slow, carbolated fuchsin, technique, sections ; biology, adaptation to environment, to genera, variation in same genus, experiments showing real identity of variant forms ; vitality : in water human bacillus 50 to 70 days, bovine bacillus indefinitely, avian 117 days, dried sputa 9 months, cow's lung 102 days, putrid material 43 days to years, survives gastric juice, sunlight kills in some hours, if in thin layer, daylight 7 to 18 days, dry heat (212° F.) for an hour left living bacilli, moist heat 140° F. for 1 hour kills, low temperatures (—25° F.) fail, after 15 days in salt failed in rabbit, after 30 in Guinea pig. Chemical disinfectants. Accessory causes : racial vulnerability, close buildings, dark, foul, damp stables, poor scanty food, heavy milking, conformation, early breeding, inbreeding, age, predisposing diseases, traffic in animals, dairy extension, admission of tuberculous animals from other states, assorting tuberculous for sale in a given State, sale of sound from tuberculous herd, lack of indemnity for animals killed, private testing of herds and sale of tuberculous, accustoming to tuberculin test, anti-pyretics during tuberculin test, false certificates of tuberculin tests, feeding hogs on tuberculous offal, feeding calves and hogs on tuberculous milk, feeding hogs after tuberculous cattle, feeding from a common trough, dry, dusty stables, extension through vermin, flies and other insects carry virus. The tubercle : miliary, pinhead, conglomerate, proliferation of cells—tissue and leucocytes, in nests in stroma, giant cells in center, then epithelioid, then lymphoid, caseation, calcification, fibrosis, pearl disease. Localization : *Cattle :* pulmonary : miliary tuberculosis, aggregation into larger tubercles, caseated centres, fibroid, cretified, abscesses, vomicæ, complex infections, infiltrations, difference from broncho-pneumonia, lesions of different ages ; pleural lesions ; congestion, exudation, fringes, nodules, grapes, adhesions, caseations : bronchial and mediastinal glands, congestion, swelling, softening, induration, caseation, cretefaction : cardiac lesions : lesions of mouth and

throat, pharyneal glands: gastro intestinal, peritoneal, mesenteric, glandular; œsophagastoma; liver tubercle; splenic; pancreatic; genito-urinary; mammary; cerebral; spinal; orbital; skeletal; cutaneous; muscular; glandular; table of distribution. *Swine:* lesions: pharyngeal, intestinal, mesenteric, muscular, hepatic, splenic, glandular, pulmonary, skeletal, caseation, liquefaction. *Horse:* lesions: thoracic, abdominal, glandular, of serosæ, vertebræ, etc. *Sheep* and goat: lesions: thoracic, abdominal, glandular, hepatic, pharyngeal, facial, etc., verminous affections. *Dog and Cat:* lesions: respiratory, abdominal, pharyngeal: tonsillar, hepatic, pancreatic, splenic, skeletal, arthritic, cutaneous. *Apes and Menagerie Animals:* lesions. *Chickens:* lesions: intestines, liver, spleen, peritoneum: lungs and kidneys often escape. *Pheasant:* lesions: as in hens, centre zone has epithelioid cells, fibroid, cretefaction, amyloid degeneration. *Parrot:* lesions: eye, beak, tongue, palate, larynx, bones, joints, lungs, liver, intestines, muscles, skin. Primary and secondary infection, extension by lymphatics, blood channels, tonsils, inhalation, deglutition.

Synonyms. Consumption; Tabes; Scrofula; Pining; Grapes; Great White Plague, etc.

Definition. An infectious disease common to man and a large number of animals, caused by the bacillus tuberculosis, and characterized by a productive inflammation giving rise to small, rounded bodies (tubercles), or diffuse infiltration, with a tendency to necrotic degeneration, and caseation, or to fibroid degeneration (sclerosis), calcification or ulceration.

Animals susceptible. Tuberculosis comes near to being a panzoötic, since although reptiles, fishes, birds and some mammals do not readily contract it under normal conditions, yet under abnormal and debilitating conditions nearly all will succumb to it.

Reptiles. Sutton found tuberculosis in a python which was kept so warm in the London Zoological Gardens that a thermometer between its folds registered 85° F. Kráhl, Battaillon and Ferre cultivated the bacillus in frogs, Kráhl in snakes. Blauvelt found tuberculosis in a salamander. Lechner found it in amphibia.

Fishes. Broussais records the prevalence of tuberculosis in carp in a pond which received the sputa of a consumptive man. Under the skin were found rounded tumors, containing abundance of bacilli, that infected rabbits and Guinea-pigs on which they were inoculated.

Birds. In birds of the farmyard—hens, pheasants, turkeys, ducks and pigeons—it is very frequent and often occurs as an

epizoötic. While the tuberculosis of barnyard fowl is a manifest variety and not readily interchangeable with the varieties affecting the mammal, yet, with a special predisposition, it can be transferred and can then be conveyed from animal to animal in the new genus in which it has been implanted. That of cage birds is interchangeable with that of man.

Rodents. Guinea-pigs have a strong susceptibility to tuberculosis, whether from human or bovine source, and rabbits for that of birds, and by continuous transmission through the body of the rodent, all alike seem to tend toward acquiring common characters. The Guinea-pig, therefore, has been especially availed of for the experimental transmission of tuberculosis, and, as the disease in them becomes acute and rapid in its progress, these subjects permit the multiplying of experiments in a short period. *Rabbits* are less vulnerable to the bovine or human form.

Sheep and goats, kept under usual conditions, show a remarkable immunity from tuberculosis, yet if directly inoculated an inherent susceptibility is easily shown. Habitual immunity may be in some degree due to their open air life, to the heavy winter fleece protecting them against chills, to the comparative absence of the heavy and continuous milk yield demanded of cows, to the more restricted development of the lymph plexuses in the lungs and elsewhere, and to the limited opportunity offered by the small tonsils for infection entrance.

The *horse*, *ass and mule* rarely contract tuberculosis casually, the more spacious stall, outdoor life, hard muscular condition, the very small tonsils, the exclusive nasal respiration, the paucity of connective tissue lymph plexuses, and the abundance of red globules, probably favor immunity. Yet on inoculation by Chauveau, Nocard and others, the horse readily succumbed to infection, generalization taking place more certainly than in the ox.

Cattle. The bovine races are remarkably subject to tuberculosis. This is probably due in part to the great amount of connective tissue lymph plexus in the lungs and elsewhere, the habit of using the mouth in hurried breathing, the deep, sudden inspiration through the mouth and over the tonsils that follows a cough, the habitual restricted size of the cow stables, the absence of individual separated stalls, the habit of feeding from the same trough with the cattle adjacent, the great drain of yearly breeding

and heavy milking, the retention in the herd of old, failing cows for their milk product and high priced offspring, and the bovine habit of licking each other with the infected tongue. In many European cities and even in country districts the disease is very prevalent. In Copenhagen a few years ago 17.7 per cent. of all oxen and cows killed in the abattoirs were tuberculous; in Berlin 15 per cent.; in Holland 20 per cent.; in Pomerania and Bomberg 50 per cent.; in Hildesheim, Hanover, 50 per cent.; in Berlin dairies 75 per cent. (Ostertag); in Leipsic and Edinburgh 20 per cent. The great variation in the data for the different cities is suggestive of different inspection standards. American figures as given by the Bureau of Animal industry are from Baltimore (mostly cows recently from the country) 2.5 to 3.5 per cent.; and for the packing centers (among 2,273,547 mostly fat steers, and therefore selected) 0.02 per cent. It is here largely a matter of locality and infection, I have seen large herds with 100 per cent. tuberculous, and extensive districts, in the north and especially in the south, entirely free from the disease.

Dogs and cats in their natural condition rarely show the disease, but contract it readily on inoculation. Cadiot found 40 cases in 9,000 *post mortems* of dogs. The young are apparently more susceptible than the old, and primary lesions in the abdomen are common and suggest infection through the food. The majority belonged to consumptive persons, and gnawed the bones that had been first picked by the owner, and ate from his plate what he left. Jacobi records the case of a dog, with general tuberculosis, which habitually licked up the expectorations of his phthisical master.

Apes and Monkeys, in confinement, almost all die of tuberculosis.

Swine contract tuberculosis readily, the large tonsils, the habit of breathing through the mouth, and the abundance of connective tissue and lymph plexuses in the lungs and elsewhere contributing to this. Yet in them the affection is mainly a dietetic disease. Swine kept in the country and fed on vegetable food are rarely affected. In Saxony, where 17 per cent. of the cattle are tuberculous only a shade over one per cent. of swine were so, and in Baden only 0.02 per cent. In hogs raised on our western farms and corn-fed the proportion is much less. Yet in those fed on uncooked skim milk, kitchen scraps and the uncooked refuse of

slaughter-houses, tuberculosis becomes very common. In one case, in a large public institution, where the dairy herd was universally affected, and where, on their slaughter, their raw offal had been thrown to the hogs, I found that the latter were almost all tuberculous. Similarly, in feeding experiments, from the time of Gerlach, pigs and especially young pigs, have shown themselves to be very susceptible.

Among the less domesticated animals that contract tuberculosis may be named *deer*, *elk*, *gazelle*, *antelope*, *camel*, *dromedary*, *giraffe*, *kangaroo*, *lion*, *tiger*, *jackal*, *jaguar*, *bear*, *arctic fox*, *rat*, *mouse* and the *common cage birds*, *etc.* Fröhner found 36 per cent. of parrots affected in Berlin.

Man has long been recognized as standing in the front rank of susceptibility to tuberculosis. Yet even in his case the prevalence of the affection bears an intimate relation to his indoor life. In large cities *post mortems* often show that one-third have suffered, and that one-seventh and upward of the whole population die of tuberculosis. Natives of tropical islands and countries, living in the open air and apart from close association with civilized man, often escape entirely. In South Africa the herdsmen living in close buildings suffer encreasingly, while their cattle, kept constantly in the open pasture, escape.

Geographical Distribution. The prevalence of tuberculosis shows a direct relation to the opportunity for infection rather than to climate, latitude or altitude. It is the prevailing disease of civilized communities with dense populations, and especially in large cities where the general vitality is lowered and the crowded buildings give every opportunity for infection. It is preëminently the disease of Central Europe, of Great Britain and of our Eastern States, where there is the greatest activity in business, manufacture and commerce. Yet in the Scottish Hebrides, Iceland, Newfoundland, the Greenland Highlands, Arctic North America, the northern parts of Norway and Sweden, Lapland and Finland it is hardly known. The absence of commerce and cattle and the sparse population have much to do with this. The coasts of Greenland with their Danish stations have two-thirds of the population tuberculous (Cook); the Indians of Barrow's Straits, when infected in 1899 by the frozen-in whalers, suffered from it as from a plague; in Christiana, Stockholm and St.

Petersburg it is as prevalent as in the large cities of Central Europe. Elevated and dry climates have often been found to be free from the affection, yet it prevails inside the double windows of houses in the higher Alps, and has, within a generation established a notorious prevalence in Minnesota, Dakota and westward, as it has in Australia, mainly owing to the advent of consumptives who sought for health in the dry atmosphere. The hills and dry tablelands in North and South America, Africa and Asia which have acquired a reputation for salubrity, owe this largely to the sparse population and the absence of facilities for infection. In South Africa, in spite of the dry and genial climate, the disease is constantly encreasing in the human (housed) population. The sea-coasts, which are the seats of large cities and the centres of population and trade, are also the most extensively affected with tuberculosis—in the West Indian Islands, Panama, Nicaragua, Mexico, Yucatan, Columbia, Equador, Peru, Chili, Ceylon, Guinea coast—while the sparsely peopled highlands of the same countries are practically free.

What is true of man is equally so of cattle. In the Gulf states of this country, herds living in the open air are practically free from tuberculosis, while in New Orleans and other large cities, the cows shut up in the confined stables and yards, are as tuberculous as those of the great cities of the north. So in many districts in the northern states we find no bovine tuberculosis, while in others, differing in no essential particular as regards geological formation, altitude, exposure, and industrial use of the animal, a large proportion are affected.

Virulence. Tuberculosis has long been recognized as virulent. "It is indicated in the Mishna and other Jewish works as rendering the meat unfit for consumption, but to come to more modern times, we find that throughout the seventeenth and eighteenth centuries the flesh of tuberculous animals was excluded from human food, alike by the civil and ecclesiastical laws of Europe. Tuberculosis in cattle was erroneously identified with syphilis of man, which made a frightful extension in the end of the fifteenth century, spreading from the army of Charles VIII which had been engaged in the siege of Naples. This conviction lasted until it was disproved about 1782. Though mistaken in the identity of the two diseases, the fact that for centuries the com-

mon people and physicians both associated tuberculosis with a malady so notoriously contagious as syphilis, speaks strongly for the forcible evidence of contagion manifested at that time. Morgagni, who must have begun practice about 1700 A. D., testifies to the strong conviction of the contagious element in tuberculosis. Indeed it became a common practice to isolate the consumptive person from the public, and after his death to burn his clothes and sometimes even the house, or at least to subject them to a careful disinfection. It is recorded that in 1750, in Nancy, the magistrates ordered the burning, in the public square, of the personal property of a woman who had died of phthisis, from sleeping in the bed of another consumptive person.

"At Naples, a royal edict of September 20, 1782, prescribed the sequestration of the phthisical, the disinfection of the rooms, chattels, movables, books, etc., with vinegar, eau-de-vie, lemon juice, sea-water, fumigations, etc., under a penalty of three years at the galleys, or in the case of nobles, of three years imprisonment and a fine of 300 ducats. A physician who failed to report a case of consumption was fined 300 ducats for the first offense, and banishment for ten years in case of a second. Any one assisting in such evasion of the law was sent to prison for six months.

"Chateaubriand found that, in 1803, he could not sell carriages in Rome, because Mme. Beaumont, who had died of consumption, had ridden in them three or four times. George Sand, who was with the phthisical Chopin in Minorca in 1839, was refused a lease of the house for the second month, and the price of repainting and purifying was demanded. Later, in Barcelona, they were assessed for the bed on which Chopin had slept, as the police regulations prescribed it should be burned.

"This was not a mere survival of vulgar prejudice. Jacobi tells us of a dog which died of consumption from eating the sputa of his phthisical master. Laennec, the discoverer of auscultation, and the great authority on pulmonary consumption, records that he himself contracted a tuberculous nodule, through a wound with a saw, while making a necropsy in a case of phthisis. Laennec died of tuberculosis later, although he seemed to have checked this lesion by caustics. Andral joins Laennec in enjoining the greatest caution and cleanliness in taking care of, or associating with persons having advanced tuberculosis.

"Cullen, who started with a strong prejudice against the doctrine of contagion, leaves us the following instance of its occurrence: 'A young man predisposed to phthisis married a Dutch girl of a sanguine temperament and good constitution. Some days after the marriage the woman lost her fresh color and was attacked by a bad cough; a month later she commenced spitting blood. The physician advised her not to sleep in the same bed with her husband, but she refused to follow his advice, and six months later she died of phthisis. The servant who took care of her, and the domestic, who avoided as far as possible staying in the sick chamber, both died of consumption.'

"Wickmann, court physician in Hanover, in 1780, pronounces emphatically for contagion. In Zurich, at that time, one death in every six was from phthisis. The contagion of phthisis was slow in its operation, and was, therefore, less evident than that of plague, smallpox, scarlatina and other affections attended by a skin eruption, but it was no less real and deadly. It was also less frequently indirect, or carried from victim to victim by intermediate agents. He cited instances of the transmission of consumption from husband to wife and vice versa, and claimed that the marriage of a phthisical person should be legally prevented. As a means of preventing the disease, he proposed a strict surveillance of establishments for the sale of old clothes, and the avoidance of leaving infants with consumptives.

"Valsalvi and Sarconi refused to make necropsies of persons who had died of phthisis.

"Dr. Ruhling, of Gottingen, writing in 1774, of the disease in animals, says: 'The malady is transmitted to sound animals by direct contact of animals standing side by side in the stall, and licking each other, and breathing the expired air direct from the diseased lungs; the frequenting of the same pastures will also serve to propagate it.' In Krunitz's encyclopedia, published in Berlin in 1787, is the following: 'The heifers show an ardent desire for the male, and remarkably enough, do not become pregnant, but part with the fruit of conception. When opened these animals show the first stomach, kidneys and surface of the lungs, covered with pustules like dried mulberries, or in suppuration. The affection is contagious, and communicates itself from one animal to another by contact.'

"Fromage, in the dictionaire de Rozier, Article Phthisis, says: 'Men of art are very much in accord that this malady is noncontagious, but some stock owners think differently.'

"Huzard, who saw much of tuberculosis in the Parisian dairies in the concluding decade of the last century, said that most of the veterinarians looked upon the disease as contagious, and that some of the physicians believed the same of the phthisis of man.

"Dewar, speaking of his experience in Scotland in 1839, and succeeding years, in a particular herd, says: 'The proprietor agreed to remove partition walls, and to make openings opposite one another in the outside walls, so that there might be a current of fresh air passing through. These alterations combined with cod-liver oil, tonics and stimulants, may have prolonged life but yet no cure was effected, and that herd died out. I resorted to every precaution when new cattle were purchased to prevent infection; as I had an idea, even at that time, that the disease might be communicated from one to another when in immediate contact. The precautions adopted when fresh cattle were purchased had the desired effect, for in a few years afterward, they were as good a lot of cattle as any in the district. That herd left an impression on my mind that never can be effaced, and made me always dread the evil consequences of tuberculosis. Having practiced in the same locality, which is an extensive breeding and rearing district, I have seen several herds decimated. Though I have sometimes foretold the owner what he might expect, yet he did not believe me until so many were infected that the byres were infested with the fearful malady, and your remarks in regard to nose contamination are so true that I do not think it possible any can escape that are so exposed. I have also seen, as Grad has, the same stall infecting beast after beast until a thorough cleaning and disinfecting, with plenty of fresh air had removed all infection.'

"Spinola, writing in 1858, of bovine tuberculosis, gives contagion as a possible cause, and Lafosse, in 1867, says: 'physicians begin in imitation of Morgagni to believe in the contagion of phthisis. The facts published by Villemin appear to support this opinion, which has been held by our colleague, Dupont, of Bordeaux, for several years.'

"Cruzel in his work of date, 1868, is much more positive; he says: 'This foetid expired air, inhaled immediately by another

cow upon a sound lung, gives the latter tuberculous infection. It is a matter of every day experience to the veterinarian. Two oxen or cows are kept in the same stable, take their food from a common rack or manger, lie in the same stall, and respire nose to nose. The one is, to all appearances, perfectly sound, the other is in as good a condition, and is vigorous, but it coughs from time to time, and its breath is foul. Soon we notice that the animal that does not cough, eats with less appetite, he loses flesh and soon he is unequivocally affected with the same malady as the first.' The foregoing quotations show clearly that in spite of the misleading teachings of Broussais and others, the doctrine of contagion in tuberculosis could not be overcome, and that up to the time of the remarkable experiments of Villemin, in 1865, it maintained its hold upon the minds of extensive and careful observers. In animals, especially, the evidence was so frequent and clear, animal following animal in the same stall, and eating from the same infected manger, only to be infected in turn, and two animals stalled together, and licking the same manger with their prehensile tongues, transmitting the infection with certainty the one to the other, were facts that could not possibly be ignored. Other cattle in the same building might escape for a length of time, but the eating in common from the same fatal manger, by a tuberculous and a healthy animal, quickly sealed the fate of the latter.

"Physicians, too, who were compelled to investigate the causes of the extraordinary fatality from tuberculosis in the armies and navies, could not shut their eyes to the fact notwithstanding that they came to the task strongly prejudiced through education against the acceptance of contagion. Thus, Dr. Bryson, in his report to the Epidemiological Society, in 1860, on consumption in the several ships of the English navy on the Mediterranean station, says distinctly that the disease appeared to be propagated by contagion. Dr. Parkes, quoting this in his Practical Hygiene, says: 'It may be inferred that pus cells were largely thrown off during coughing, and floating through the air, were received into the lungs of other persons. The production of phthisis in animals confirms this view. The case of monkeys in the zoological gardens, narrated by Dr. Arnot, is a striking instance. Cows in close stables frequently die of

phthisis. But not only phthisis may reasonably be considered to have one of its modes of origin in the breathing of an atmosphere contaminated by respiration, but other lung diseases, bronchitis and pneumonia, appear also to be more common under such circumstances.'

Villemin in 1865, published his investigations in which he had produced tuberculosis in a great number of animals, by inserting particles of fresh tubercle or injecting the tuberculous sputa of man into the subcutaneous connective tissues, the peritoneum and the trachea. There resulted in nearly every case a chronic disease, the marked phenomena of which were caseated centers devolving from firm inflammatory nodules in the seat of inoculation, in the adjacent lymphatic glands, in the lungs, serous membrane, liver, spleen and kidneys. The centers of such nodules were at first transparent and grayish, but soon the center underwent necrobiosis, forming a soft cheesy mass the size of a pin's head, and gradually enlarging to that of a pea, a bean, a hazel nut or larger. After a period of about 14 days the lymphatic gland nearest the seat of injection could be felt as a firm pea-like nodule, and in two weeks more a second gland on the line of the lymph circulation, had become enlarged and nodular, while the first, now the size of a large bean, had probably undergone distinct softening. This became adherent to the skin, burst and discharged continuously or at intervals with little or no tendency to heal. If killed at this date, the animal showed only the open sore and a chain of nodular pea-like caseated lymphatic glands leading up from it. The lungs might be apparently sound, but the spleen and liver usually showed miliary elevations on the surface, with clear grayish centers, as viewed under a magnifying glass, opaque patches were found on the omentum, and the lymphatic glands of the mesentery, of the portal fissures of the liver and of the hilus of the spleen were distinctly enlarged and resistant. If not killed, the subject usually died 100 to 120 days after inoculation, and then the lungs were found studded with tubercles miliary or larger and more or less caseated ; the bronchial mediastinal, subdorsal, pectoral, prepectoral, phrenic, mesenteric, hepatic, and splenic lymphatic glands were more or less enlarged and caseated, while the liver and spleen were enlarged and studded with multiple tubercles. Guinea pigs give the most

extensive and intensive lesions, rabbits much less so, but in both, emaciation and wasting are very marked. In short the lesions are essentially and unmistakably those of generalized tuberculosis.

"At first, however, incredulity prevailed and experiments on a large scale were inaugurated all over Europe and America to put the question to crucial test. Martin, Conheim, Burdon-Sanderson, Simon, Wilson, Fox, Lebert and Wyss, Fraenkel, Waldenbourg, and others inoculated with all sorts of non-tuberculous organic materials (bronchial mucus, foul pus, cancer juice, diseased liver, simple cotton setons, etc.) and from these in Guinea pigs there resulted local caseated foci, and implication of the adjacent lymphatic glands. It was found that the liquids from low types of pneumonia produced these apparently tubercular deposits, while that from acute sthenic pheumonia did not; that the cotton thread seton failed to produce the disease if it were first steeped in carbolic acid, and that the lesion caused by breaking a bone without injury to the skin failed to produce the characteristic lesions even in the very susceptible Guinea pig. It became evident, therefore, that the morbid results in these cases, were due to accidental inoculations with the poison of the tubercle, which is widely distributed, or with some pyaemic or other germs.

"Gradually the truth triumphed, and those who had at first been loudest in their opposition were led by their own experiments to become the firmest supporters of the new truth.

"As early as 1874, Gerlach had experimented largely in feeding the milk of tuberculous cows, and had infected calves, pigs, sheep and rabbits in this way, Chauveau in (1874), Klebs (1873), Semmer (1880) and Parrot (1870) fed calves, cats, Guinea pigs, pigs, sheep and a variety of other species of animals with tuberculous flesh or lungs, or saliva, producing tuberculosis in a large proportion of cases. In this manner they escaped the charge of the operation of a wound and of aerial bacteria, and yet succeeded in producing infection. The lesions in this case began in the intestinal mucous membranes and mesenteric glands.

"Another essay in the same direction was made by Tappeiner (1878), who infected rabbits, Guinea-pigs and dogs, by enclosing them in boxes and compelling them to breathe tuberculous

liquids intimately mixed with the air by atomization. In these cases lesions began in the lungs and bronchial and mediastinal glands."*

Bacteriology. The *bacillus tuberculosis*, the essential cause of tubercle, was demonstrated by Koch in 1882. It was at first supposed to be peculiar in its indisposition and tardiness to take a stain, and in its retention of the stain once imparted, even when it is treated with acids. Later observations have shown that these characteristics are common to certain other bacilli, notably to those of leprosy, smegma, and to specimens found in timothy hay and other grasses, cow dung, milk and butter. These furnish sources of fallacy in the milk and butter especially, but when the specimens examined are taken from the interior of a tubercle, lymph gland or serous cavity the danger of error may be practically ignored. What is quite as important is the fact that the tubercle bacillus is subject to evolutionary changes in adapting itself to a habitat in different genera of animals, and to a lesser extent in different parts of the same animal, by which its pathogenisis is modified, but these variations do not materially affect the staining qualities. Still more striking variations have been found in old cultures, ovoid forms (Metchinkoff), club-shaped microbes and filaments (Metchinkoff, Klein, etc.), branching filaments (Fischl, Coppen Jones), divergent club-shaped groups, like actinomyces (Babes and Levaditi). The last named, seen in inoculated rabbits, have been held to establish a relationship between the microbes of tuberculosis and actinomycosis, as the club-shaped cells do not take the usual Ziehl-Neelsen stain for tubercle bacilli, but the Birsch-Hirshfeld's actinomyces stain. Apart from the rabbit such variations are not likely to prove sources of fallacy in identification of the microbe.

Morphology. As met with in the tubercle of man or ox the bacillus is a minute rod with rounded ends, 1.5 to 3.5μ long, by 0.2μ in thickness. In the ox it is shorter and thicker on an average than in man. They are usually solitary, but two and exceptionally even three or four elements may be united. In stained specimens unstained portions are frequently seen (spores?) When cultivated on blood serum there is a tendency to form

* (From Report to the N. Y. Legislature, 1895. James Law.)

elongated rope-like colonies having a waving outline. Other evolution forms have been already noted.

Staining. The resistance to staining is overcome by a heated alkaline watery solution of methylene blue (Koch), or better by a carbolated solution of fuchsin (Ziehl-Neelsen). To a 5 per cent. watery solution of carbolic acid add one-tenth of its volume of saturated alcoholic solution of fuchsin. The suspected sputum, or scraping from a section of a lesion, is spread in a thin layer on a cover glass by drawing over it the straight edge of a second cover glass, dried in the air, and passed three times through an alcohol flame with the smeared side up. It may then be floated on the surface of the staining fluid with the specimen side down, then gently heated for three or four minutes almost to ebulition, drying being prevented by the addition of more of the carbol-fuchsin solution as required. It is then washed in water to remove excess of the staining fluid and treated with a bleaching acid solution (sulphuric acid 5 per cent. or nitric acid 15 per cent). When decolorized it is well rinsed in two or more portions of a 60 or 70 per cent. alcohol and then in water. Next stain with a 1 or 2 per cent. aqueous solution of methylene blue for one minute and wash off the excess with water. The tubercle bacilli will appear of a deep red and other bacteria blue.

Gabbett's counter-staining and decolorizing solution is especially useful in economizing time. After staining, as above directed, in the carbol-fuchsin solution for one minute, wash in water, then cover with a solution of 2 grams methylene blue (powder) in 100 cc. of 10 per cent. sulphuric acid, until the film has a faintly bluish tint : then wash thoroughly in water and examine. Tubercle bacilli come out red, others blue.

In *examination of tissues* the microscopic sections are steeped for twenty-four hours in the carbol-fuchsin solution (or 1 hour at a temperature of 45° to 50° C.), then decolorized in a 5 per cent. sulphuric acid for a few seconds, then in a 70 per cent. alcohol ; they are then counter-stained in an aqueous solution of methylene blue, washed in water dehydrated with absolute alcohol, cleared up in xylol or oil of cedar, and mounted in Canada balsam.

Biology. The *bacillus tuberculosis* is notorious for its variant forms, assumed in adapting itself to given environments, and which it retains with considerable tenacity for a time, even when

placed in a new and different medium. It is largely held to be an obligatory parasite, and incapable of survival as a saprophyte, yet Straus, Nocard and others, have shown that the form obtained from the tuberculosis of birds can easily be made to live in suitable dead mixtures of organic matter. It is also notorious that the bacillus taken direct from the tubercle of the mammal, and which, as a rule, fails to grow at once in glycerined bouillon, yet after a first successful culture in the new medium, often adapts itself completely, and thereafter it can in many cases be transferred from bouillon to bouillon, with as great certainty as it could formerly be inoculated from ox to ox. The difference is one of habit and adaptability, rather than any primary and permanent distinction of species. There is every reason to believe that the microbe has lived, and under given conditions can still be made to live as a saprophyte, with a greatly reduced adaptability to parasitic life in the animal, just as we see to-day that it is only with great difficulty transferred from certain genera of animals to certain other genera (from bird to ox and vice versa). Trudeau tells us of a culture of a bacillus tuberculosis from man, inoculated on the rabbit and then cultivated *in vitro* in successive generations for six years, that was in this way robbed of its pathogenesis for Guinea pigs, which, after inoculation, lived for many months, some two and a half years, and some even recovered. The average duration of life in the Guinea pig, after tuberculosis inoculation from man, rabbit, Guinea pig or recent culture is but seventeen days. The bacillus of human sputum often produces only localized tuberculosis in the ox. Even in the same species and individual the pathogenesis often varies materially. Nothing is more familiar than the slow progress of tuberculosis in the bones and lymph glands of man, on the one hand, and its frequently rapid progress in the lungs, liver and brain, on the other. As inoculated on the lower animals bacilli from the lungs of man are usually more virulent than those from the lymph glands (Creighton, Arloing, F. Craven Moore, Courmont and Denis). Among seven specimens of human sputum, the bacilli in six cases showed a fair average vitality, whereas those of the seventh failed to grow on blood serum of the dog (Theobald Smith). In both man and cow a large proportion of cases of tuberculosis remain localized, the

disease lasting for years with little or no appreciable advancement, and not a few subjects appear to make a permanent recovery.

On every hand this evolutionary tendency of the bacillus tuberculosis places itself in evidence, demanding a recognition of the fact, that more or less transient or lasting variations in accordance with previous or existing environment, control to a large extent the transmissibility of the disease among different genera and even among individuals of the same genera and species, and also the fatal progress, or the mild and evanescent issue of the infection.

Even the most widely divergent of these evolutionary forms can often be made to approach each other and apparently merge into one type. Profiting by the example of Metchnikoff, Nocard enclosed in collodion capsules the bacilli of the human sputum and inserted the capsules into the peritoneal cavity of chickens which had proved refractory to their direct inoculation. This extended the direct action of the leucocytes from the encapsuled bacilli, but allowed the endosmosis of the serum of the fowl for their nourishment. After a sojourn of four months or more they were transferred to other capsules and again enclosed in the peritoneum and after a second and third transference of this kind it was found that the bacillus had become actively pathogenic for the chicken, having acquired the infecting potencies of the bacillus of avian tuberculosis.

In keeping with the above is the fact that both rabbit and horse are easily infected by the bacillus obtained from birds, and that after a certain number of transmissions, through the rabbit the issue of the bacilli of bird and mammal appear to become identical. No less instructive are the cases of the infection of carp by human sputa and the conveyance of tuberculosis to rabbits and Guinea pigs by inoculation with the nodules of the infected carp.

The *vitality* of bacillus tuberculosis is strong but variable. In sterilized water at 46° to 64° F. the human bacillus survived for fifty to seventy days (Chantemesse and Vidal), the bovine indefinitely (Galtier) and the avian bacillus at a higher temperature one hundred and seventeen days (Straus and DeBarry). In dried expectoration the bacillus of man still infects after nine or ten months (Koch, Schill, Fischer, De Thoma). In infected cow's lung, dried and pulverized, it infected Guinea pigs at 102 days (Cadeac and Malet). In putrid matter it infected after 43

days (Schill, Fischer), 167 days (Cadeac and Malet), several years in a grave Schottelius).

It is not destroyed by gastric juice (Fischer, Falk). The bacillus from tubercle of birds has a much greater vitality than that of mammals, Marfucci successfully seeded new media from a culture of two years old, and cultures in artificial media can be started more successfully.

Full *sunlight* renders sputum on a solid non absorbent surface non infecting in several hours, varying according to the thickness of the layer (Koch, Straus). When sputum is mixed in soil it may survive until the 137th day (Feltz). On woolen cloth it may live five hours (Migneco).

Diffuse daylight, with shading from the sun, killed in seven days (Koch), to eighteen days (Lucibelli).

X-*Rays* do not arrest the growth of cultures (Blaikie, Pott, Ausset).

A *dry temperature* of 212° F. for an hour left some of the bacilli still infecting to Guinea pigs (Lartigan).

A *moist heat* of 140° F. for an hour sterilizes (De Man, Th. Smith, etc.). The scum formed on the milk may still prove infecting (Th. Smith). Half an hour of a moist temperature of 212° F. is sterilizing. Yet in the case of steaks, roasts and boiled meats the size of the piece often prevents the reaching of this temperature throughout, and it becomes unsafe to use any meat in which the redness of the juice shows that the albumen has not all been fully coagulated (162° F.).

A *freezing temperature*, —16° to —26° F., does not devitalize the bacilli, even when alternated with thawing at intervals for several weeks (Galtier, Cadeac and Malet).

Heavy salting of meats has been thought to kill the bacillus in a month. After 15 days in salt the microbe failed to kill rabbits, but still killed the Guinea pig, whereas after 30 days it killed neither (Galtier). The fact that salted meats are always unequally impregnated in different parts, renders this extremely unreliable and more of a snare than a guide.

The action of chemical disinfectants varies not only with the agent but also with the medium in which the bacillus is found. In simple liquid media (bouillon) the following results were obtained :

	Parts per 1000.	*Killed in.*	
Carbolic Acid	50: 1000	30″	(Yersin)
" "	10: 1000	1′	"
Alcohol (absolute)	1000: 1000	5′	"
Ether	1000: 1000	10′	"
Iodoform ether	10: 1000	5′	"
Mercuric chloride	1: 1000	10′	"
Salicylic acid	25: 1000	6 hrs.	"
Thymol	3: 1000	3 hrs	"
Formalin vapor	60: 1000	40 hrs.	(Murray)
Sulphurous Acid (Sulphur 1 oz. to cubic metre)	———	14 hrs.	(Vallin)

The following agents proved ineffective: Saturated aqueous solution of creosote, or of B-naphthol, of naphthalin, of potassium iodide, of potassium bromide :—bromine water, iodine water (1:500), iodoform solution or vapor, vapor of oil of turpentine.

ACCESSORY CAUSES OF TURBERCULOSIS IN ANIMALS.

While recognizing that in the absence of the tubercle bacillus there can be no tuberculosis yet we must not ignore the fact that many conditions of the animal system and its environment contribute largely to the propagation of the disease on the one hand, or to hinder its progress on the other. None of these conditions can call the germ into being *de novo*, but in its presence, they greatly favor its diffusion or even its malignancy. Like any other seed, this bacillus requires a suitable soil and favorable climate, to bring out its most destructive development. In striking the balance, we have to guard against the error of so many, who would attribute to the germ alone the deadly results and who assume that these should be the same under all conditions, and the opposite error no less prevalent, that ascribes the evil to the condition, and holds that without these the germ would be harmless.

Hereditary Predisposition. Racial Vulnerability. Before the discovery of the specific bacillus, when as yet tuberculosis was held to result from a constitutional weakness, or cachexia in which deterioration of cells was held to be the main factor, the disease was held to be mainly hereditary, and its every day transmission in the line of descent, and the increasing mortality to extinction of given families were confidently appealed to in support of the

doctrine. Now, however, we recognize that congenital tuberculosis in man or beast is very exceptional, and that the morbid process almost invariably takes its start from the germ implanted after birth. In Saxony when the tuberculous cows were 16.5 per cent. tuberculous calves were but .2 per cent., though the latter had been fattened on the milk of the former. In Munich but two calves were found tuberculous in 400,000 killed, and in Lyons but five in a similar number. Up to the present the number of calves recorded as tuberculous at birth does not exceed seventy.

That the young almost always contracted the disease, only after birth, virtually disposes of the alleged heredity of the tuberculosis but it by no means antagonizes the heredity of the racial vulnerability. As man, cattle, swine and Guinea pigs show a much greater vulnerability than the carnivora in general, so certain families in each of these genera show a more decided susceptibility to tuberculosis under similar conditions, than do certain other families. This goes far to explain the appearance of tuberculosis, in certain lines of blood, and its advance to the extinction of the family, while under no better environment, other families can count on a practical immunity. In the Burden herd of Jerseys in 1877, I condemned eleven animals, verifying my diagnosis by necropsies, and found to my surprise that I had taken every representative, even the grades, of a given family, and left all the pure bred members of a second family untouched. Both families had mingled freely in the pastures and yards, yet the second family furnished no cases of tuberculosis then, nor for many years afterward. The case is all the more striking that the non tuberculous family gave the largest yield of milk and might have been expected to run down rather than the other on account of this drain.

Close Buildings. Lack of Ventilation. Air rendered impure by being breathed again and again, predisposes strongly to tuberculosis, and has been even looked upon as the sole cause (Macormac). Everywhere city dairy cows, kept in confined, close buildings, suffer severely (6 to 20 per cent. and upward ; in Berlin 75 per cent. (Ostertag), in Denmark 61 per cent. (Bang), while in the same districts country cows are comparatively immune (often 1 to 2 per cent.), and steers raised in the open air still more so (0.02 per cent. for our Plains cattle). For the slighter

cases of tuberculosis in man and beast, life in a pure open air, day and night, in a genial climate, gives the best hope of improvement or recovery. In the Burden Jersey herd above referred to, animals condemned in spring as tuberculous, were turned out to pasture during the summer where they maintained an appearance of robust health, yet when returned to the barns in Fall they fell off rapidly so that some had to be helped to rise in the stall. "The stabled cow, the tame rabbit, the monkey, the caged lion, tiger or elephant are almost invariably cut off by scrofulous affection" (Aitken). It has long been noticed that sailors sleeping in close spaces (Bryson, Parkes), suffer much more than the officers in more spacious rooms (Clark). Monks and nuns (two-thirds of the deaths, Leudet) occupying confined cells, and the inmates of prisons (four times the average outside, Baer) have shown an extraordinary prevalence of tuberculosis and attendant mortality. While this can be attributed mainly to the preservation and concentration of the bacillus in such places, a considerable allowance must be made for the impure and rebreathed air.

Dark. Foul, Damp Stables. Dark stables are usually close, dirty and damp as well, and all these conditions alike conduce to tuberculosis. Darkness hinders the development of organic coloring matter in living bodies, whether chlorophyll in plants or hæmoglobin in the blood of animals. Hæmoglobin is the main oxygen carrier in the blood, and in case of its deficiency the tissues are not properly ærated. The result is as if the inhaled air contained little oxygen, so that darkness further intensifies the evil of rebreathed, deoxygenated air. The extraordinary mortality from tubercle among prisoners, monks, nuns and miners serves to further accentuate this conclusion.

Trudeau's experiment with rabbits is instructive in this connection. Of a number inoculated with the same number of tubercle bacilli, one half were kept in the open air and the rest in a dark, damp, underground place deprived of sunlight. When killed on the same day, it was found that the open air rabbits showed only slight lesions or none, while the underground lot had extensive tuberculosis.

The impure stable air lowers the vital tone of the animal, especially if the impurity has come from animal exhalations.

The same is true of damp air, "a damp ship is an unhealthy one," and Bowditch and Buchanan pointed out the especial prevalence of tubercle in cold, damp, undrained, foggy localities. This does not hold for all damp lands, yet damp, fog, and cold are especially hurtful in presence of the germ.

INSUFFICIENT OR UNWHOLESOME FOOD.

Starvation and indigestible or innutritious food contribute to debility, and lessen the power of resistance. Hence in the poor, half starved denizens of city slums, and among neglected herds of cattle, tuberculosis, once introduced, makes rapid and extensive ravages. Conversely, overfeeding often acts in the same way, developing indigestion and thereby robbing the tissues of their proper nourishment.

HEAVY MILKING.

In dairy cows, of milking breeds, the drain on the system is liable to prove too great, under a ration-for-milk, warm sloppy mashes of grains, bran, middlings, roots, distillery swill, exclusive diet of silage, saccharine roots, or marc, warm drinking water, a warm atmosphere, liberal salting, and enforced idleness in the stall, with careful stripping of the bag. The butter cows, (Jerseys, Guernseys, Alderneys, etc.) are especially liable to suffer, as the greater the yield the more the system is robbed of the adipose material which is so needful to a vigorous health. When the cow has been reduced to a high-pressure milk factory her physical size reduced to obviate the need of a large sustaining ration, and her milking capacity stimulated to the highest degree, the presence of the tubercle bacillus is especially dangerous.

CONFORMATION.

It was formerly held that cows with heads narrow between the horns, small thin necks, narrow chests defective also in depth and length, loose projecting shoulders and elbows, pendent, pot bellies, with hollow flanks, and a general laxity of the frame were

especially predisposed to tubercle. In many such cases the suspected animal was already the victim of the affection, which had brought out these characteristic features of weakness and emaciation. In other cases the unthrifty appearance was due to poor feeding and care or to chronic disease, which in robbing the system of vigor and hardihood laid it open to the attack of the tubercle bacillus whenever it was introduced. Even when there was no such depressing influence affecting the individual, the inheritance of such a frame, betokened a constitution lacking in vigor, and with little power of resistance to the invading microbe. Some milking breeds which tend to the above conformation, show an unusual development of the lymph glands and plexuses, and as tuberculosis attacks the lymphatic system preëminently, the bacillus finds an especially favorable field for development in such systems. It would, however, be an error to assume that the compact, rounded frame, with circular chest and abdomen, and full, firm neck and shoulders, with a great disposition to fat and little to milk, is in any degree immune. Under the presence of the tubercle bacillus, and close stabling, they often succumb quite as rapidly as the most susceptible milking breeds. The meat producing breeds with a strong propensity to fatten, have an extraordinary development of lymph spaces and plexuses in the intermuscular and subcutaneous connective tissue, and the microbe finds a welcome home in their sluggish, inactive and atonic systems as well as in the typical dairy cows.

BREEDING TOO YOUNG.

Breeding from immature animals undermines the vitality as the system is overtaxed to sustain at once the demands for further growth, for the nourishment of the unborn offspring, and for the yield of milk. Some families of Jerseys have been undoubtedly injured in this way, through the desire to diminish the size of the cow, and yet retain the highest milk and butter yield.

CLOSE-BREEDING.

Inbred families are proverbially subject to tuberculosis. By pairing individuals that show in a marked degree the desired qualities of early maturity, heavy milking, or fattening, the

greatest stimulus is given to the enhancement of such qualities, and with this there is the correspondingly ready receptiveness to the tubercle bacillus. To this is added a degree of constitutional delicacy which comes from too close breeding, and which otherwise shows itself in an encreasing infertility, when bred with an animal that is nearly related. In addition, the high bred animal is kept much indoors, where the bacillus, once introduced, is easily preserved and propagated, and is denied the free outdoor life and exercise which might develop muscle and vigor. This is no valid argument for introducing inferior blood to the deterioration of the economic value of a race, but rather for the complete exclusion of the bacillus which may find in our treasured herd an especially inviting field for its ravages.

AGE.

In both man and beast, age appears to have a predisposing effect, but apart from debility or trauma, this may be fully explained on the ground of often repeated infection, an indoor life, and, in dairy cows, the excessive drain upon the system. In thoroughbred herds of cattle, in which the cows are preserved as long as they will breed, and in dairies where they are kept as long as the milk yield is satisfactory, the oldest show by far the largest percentage tuberculous. In secluded individual dairies we have found 100 per cent. affected, Ostertag gives the average for Berlin at 75 per cent., and Bang for Denmark at 69 per cent.

PREDISPONENT DISEASES.

As already noted all diseases which undermine the health tend to diminish the powers of resistance to the tubercle bacillus. To be especially dreaded, are long standing diseases which produce emaciation and debility, fevers that for a length of time impair sanguification, metabolism and nutrition, diseases of the digestive organs which cut off the requisite supply of nutritive material, and, above all, diseases of the lungs and air passages that impair the tone of the tissues and invite the attack of the invading microbe. Bronchitis, laryngitis, pharyngitis and tonsilitis in all their varied forms are to be particularly noted. Even in man it is believed that one of the most common channels of infection entrance is through the open tonsillar crypts, and the same doubt-

less is true of animals. Pigs, with their large tonsils and frequent infection through feeding, are probable examples of this, and when, in addition to the detention of the microbe in the cavities, there is an inflammation and debility of the tissues, the opportunity for infection is greatly enhanced.

ENCREASED TRAFFIC IN (TUBERCULOUS) ANIMALS. DAIRY EXTENSION.

In common with all infectious diseases, tuberculosis owes its great extensions to the sale and purchase of animals. Where an indigenous race of cattle is raised and sold, without any additions from without, tuberculosis is usually rare and confined to the one herd, or to those having intimate intercommunication with it. Where, on the other hand, a large stock is kept up, as in milk dairies, and few or no calves are raised, but the bulk of the cows are sold off yearly and replaced by new purchases, tuberculosis, sooner or later, finds admittance through the body of an infected animal, and once implanted in the herd, it goes on encreasing, without limitation, except by the sale or death of the more seriously affected. The larger the herd and the greater the number of yearly changes, the greater the number of opportunities for the introduction of an infecting animal. Where a great part of the herd is turned off yearly, and the new animals are drawn from any or every source indiscriminately, it would border on the miraculous if such a herd were to remain free from the infection for any great length of time. The recent extension of tuberculosis in herds has been coincident with the great development of commerce in live stock, and the rapid transit on land and water by steam. Before the days of the steam engine, animal plagues advanced slowly and uncertainly, excepting in the presence of a great European war, which drew animals from all available sources, congregating them in large mutually infecting droves in commisariat parks, and marching them in the wake of the army for its daily supply. Inevitably every country invaded was infected, and the plagues spread from the line of march in all directions. The more deadly plagues, like rinderpest and lung plague, were the first to appear, and in this lay a certain measure of palliation of the evil, as the exposed and often weaker animals were killed off, so that the slower contagion like that of

tuberculosis had not time to develop. This mitigating influence held good so long as the deadly plagues were not placed under effective control. When, however, a nation successfully extirpated and excluded lung plague and rinderpest, the way was opened for a freer extension of tardily developing plagues like tuberculosis. The great development of beet sugar factories and the accumulation of herds to consume the marc; the extraordinary extension of manufactures which have made England and certain countries of Western Europe consuming rather than agricultural lands, and the wonderful modern expansion of dairy husbandry have combined to encrease and concentrate the cattle industry in ratio with the manufacturing and commercial progress of the nations, and as the live stock are kept under a milk-stimulating regimen and a life passed largely indoors, the way has been open for an ever-advancing encrease of tuberculosis. In the United States the centralization of population in the Eastern States and in all great centres of industry and the concentration of cows for the milk supply, and in certain districts in connection with butter and cheese factories, have contributed to wide local extensions of consumption. Hence it has been no uncommon thing to find herds in the vicinity of cities with 20 to 100 per cent. affected, in striking contrast with the 0.02 per cent. found in the fat cattle from the plains.

The modern railway traffic brings to all these swarming centres of animal industry, live cattle from long distances to supply the constant depletion through deaths and the disposal of dry cows, and thus the whole Atlantic slope is drawn upon to fill the eastern stalls. Infected animals thus shipped from Ohio, Indiana, Illinois, Wisconsin and Michigan for the good of the herds in those states find new homes in New York, or New England, where they have four to twelve months to propagate the infection before they are themselves disposed of. As in the days of the lung plague in America, the eastward trend of bovine traffic, becomes to a certain extent a protection and benefit to the herds of the west, but in equal ratio it operates to the detriment of our eastern stock. What is true of the planting of new centres of infection in the busy dairy districts of the east, and of the steady increase of disease in already infected herds, through the constant addition of new cases purchased, is no less true of the cor-

responding districts devoted to dairying interests in the west. It is the natural order of things, that, everywhere, the new accessions of infection, coming in the lines of trade into large dairy herds, kept to a great extent indoors for months at a time, must hasten the general infection of such herds. When, therefore, no intelligent measures are interposed to check the evil, we must expect that the marked increase in the prevalence of tuberculosis, which we have witnessed of late years, shall become more and more evident year by year. Journals have claimed credit for befriending the stockman, in opposing all control of the cattle traffic, dealers have denounced all control as ruinously oppressive and injurious, and legislators have turned a deaf ear to the warnings of science and experience, but time, in this as in all other plagues, will justify the demand for an intelligent control, and the increasing losses will one day open the eyes of the stock-owners to the fact that their truest friends are those that would exclude the baneful seed, and kill it ere it has had time to germinate and bring forth its ever increasing harvests.

In the absence of any systematic and efficient government protection in this line, the stock owner can only protect his sound herd by the rigid exclusion of all animals that are not of his own breeding, or in case of purchase, by an exhaustive inquiry as to the occurrence of sickness or deaths in the herd from which he buys, and by the professional examination and test of every animal to be bought. Even then he must promptly separate, test, and, if necessary, destroy any animal that proves unthrifty, or which by cough, diarrhœa, wheezing or other sign gives evidence of probable tuberculosis.

Unregulated Traffic in Tuberculous Animals from Other States. The danger of buying animals untested is in no degree lessened when they are sent in from other states. While some make a business of supplying the store market with what they believe to be good stock, there is always the temptation to turn off animals that are unthrifty or poor milkers and which have proved less profitable than the others. Some even have the herd tested and sell off those that show evidence of tuberculosis. Unless, therefore, it is held in check by the tuberculin test, the traffic is made to the extent of such sales a direct means of disseminating tuberculosis.

ASSORTING THE TUBERCULOUS ANIMALS FOR SALE IN A GIVEN STATE.

A still more injurious result comes from the order of given States, that the admission of store cattle shall be guarded by the tuberculin test of each animal, and the supineness or worse, of adjacent states in establishing no effective safe guard against the disposal of the tuberculous culls in the unprotected State. Cattle from the west or elsewhere in the United States, arrive in a great public market as, for example, New York, they are here tested, those that stand the test are shipped into one of the States requiring the test (Massachusets, Pennsylvania, New Jersey, Canada), and those that have shown the reaction of tuberculosis are sold into the herds of the State in which they were tested. The most malicious enemy of the New York stock owners, could not devise a surer means of stocking the herds of the State with tuberculosis than this atrocious system. Yet when the present writer (1898) had a bill introduced into the legislature to correct the evil the opposing interest proved strong enough to prevent it from coming to a vote. The legislative committee on tuberculosis which sat later, and upon whose attention the subject was pressed, also failed to correct the evil and left the matter as bad as before. Under the present law the New York purchaser must secure his own interests by having every animal he buys tested by a trustworthy veterinarian.

Sale of Sound Animals Concentrates the Tuberculosis. The action of one or more States in admitting store cattle only after a tuberculin test, acts directly in encreasing the relative number of tuberculous animals in adjacent States. The purchaser from Pennsylvania for instance, goes into a New York herd and purchases all the best animals on the condition that they pass the tuberculin test uncondemned. It follows that the New York herd is left with the tuberculous cattle only, and those that, aside from tuberculosis, are of low value or profitless to keep. Further as the advance of tuberculosis is proportionate to the relative number of tuberculous subjects in the herd or building, and the concentration of the poison, the depreciated herd is almost certain to become rapidly and generally affected by the disease.

The Denial of Indemnity for Tuberculous Animals Killed.

Several American States forbid compensation to the owner for any animal affected with the contagious disease charged. All history attests, and any consideration of human nature might teach, that such a measure is only calculated to spread the infection. The owner of an animal, affected with a contagious disease, who can get little salvage by turning it into beef, and none at all if he hands it over to the State for slaughter, will naturally think of putting it on the market, where he can secure a good price. How much more is he tempted to do this when the disease is an occult one, and the animals show the outward appearance of health, as is the case in nine-tenths of many tuberculous herds! Crime cannot be fixed on the seller, for he is not an expert, and cannot be expected to diagnose the disease. If the infected cow is of little value for the dairy, she is passed on, from hand to hand, leaving infection in every herd she has entered. The ultimate owner (in whose hands the State finds her and diagnoses her disease), though he may have bought her in good faith as a sound animal and paid a correspondingly high market price, is made to lose the whole value of the cow. The real offender who knew her to be a tuberculous animal, and sold her in consequence at the price of a sound cow is shrewd enough to keep himself out of the clutches of the law, while the honest purchaser who has been already swindled, has his income and property cut off without compensation. Such a law is self-evidently unjust; it plays into the hands of the swindler at the expense of the just man; with the object of protecting the community against infection, it refuses to call on the public for any contribution toward its own protection. The system is a direct bid for extensive and encreasing violation of the law and diffusion of the infection and must be accorded a prominent place in the list of causes. It would be surprising to find that any country ever extirpated an animal plague by working on such a system. As a matter of fact no country ever did; all such sanitary successes from the extinction of sheeppox or rinderpest in Western Europe, to the recent *stamping out* of lung plague in the United States, were based on a just compensation to the owners of the stock. When, therefore, veterinary sanitary principles and experience have been so far ignored as to allow the passage of a law, which at once favors the diffusion of infection

by the crafty sale of the infected, the expatriation of the healthy animals from the home herds, leaving only the diseased, and finally the selection from herds in transit of the tuberculous ones to be scattered widely among the herds of the State, we must not wonder at the continuance or encrease of tuberculosis in the commonwealth. Until a more rational and common sense legislation can be secured, the unfortunate stock owner must defend himself by the expensive alternative of testing every animal he buys.

Private Testing of Herds and Sale of the Tuberculous. A most reprehensible practice is the private testing of herds and the sale as store animals of those that re-act. In this way a herd may be cleared of infection, but at tenfold expense to the public at large, as every animal sold may convey the infection into a separate herd. It is, however, a natural outcome of the unwise system of refusing indemnity for the infected.

Habituating to Tuberculin Testing and then Selling under Test. Many (not all) cattle, when injected with tuberculin repeatedly at short intervals, acquire a tolerance of the agent and fail to react as at the first test. We have in such cases examined the animals *post mortem* and found active tuberculosis. Unscrupulous owners, aware of the fact, have their diseased cattle injected repeatedly every few weeks, and as soon as they fail to respond, sell them under the guarantee of the tuberculin test. The cattle, of course, carry the infection into other herds. This swindle could be obviated if it could be made a misdemeanor to have or use tuberculin except as a State or Federal official acting for the government. This would imply the corollary that the State should test the herds when called upon.

Antipyretics During Tuberculin Test. Another method of undoing the tuberculin test is to feed large doses of antithermic agents to depress the temperature at the time the rise from tuberculin would take place. Tuberculous cattle may thus be sold as sound animals, with a certificate of having successfully passed the tuberculin test, the operator having given such certificate in perfect good faith. It suggests the importance of withholding tuberculin from public use, or of securing an absolute control of the feeding and watering of the animals during the test.

False Certificates of Tuberculin Tests. Stock owners have reported to the writer alleged tuberculin tests of their herds which

were completed in three hours, and others have named instances of marking animals for shipment and giving certificates of testing where no tuberculin had been used and no examination beyond the most cursory glance had been made. This might be expected of some non-graduates, registered on a basis of alleged practice, shamefully ignorant of veterinary medicine and conscious of their false position, and some educated veterinarians thus placed at a disadvantage may have been tempted to follow suit, but it can only end in personal disgrace and an inevitable extension of tuberculosis. Begotten in a legislative wrong and nourished by moral weakness, it can only grow into greater evil. Unfortunately such unworthy actions throw discredit on the very name of sanitary police. To avoid the evil every one aspiring to the responsible work of testing cattle should be thoroughly examined as to fitness and licensed to practice under a heavy penalty for neglect or malpractice.

Feeding Hogs on Fresh Offal from Abattoirs. It has been a common practice, especially in country districts, to turn the raw offal of slaughterhouses to pigs, and as the tubercle is usually concentrated in internal organs, the hogs become infected in large numbers. In public institutions which slaughtered their own meat I have found the hogs all but universally tuberculous. The danger is only slightly lessened when the hogs are fed raw butcher and kitchen scraps in swill. It suggests the compulsory boiling of all swill or garbage containing raw meat.

Feeding Calves and Hogs on infected Milk. Though it has been repeatedly shown that the majority of moderately tuberculous cows do not yield infected milk, yet in every tuberculous herd, at irregular intervals, one or more are attacked with tuberculosis of the mammæ, and the drinkers of the milk take in the tubercle bacilli. This will happen in the most strictly supervised tuberculous herds, while in those that are less carefully managed, the milk that is considered unfit for human consumption is fed to pigs or calves. In one dairy, I found that the calves, all fed in this way afterward reacted under the tuberculin test, while the following year the crop of calves, though fed on the milk of the same diseased cows, with this difference that the milk had been first heated to 180° F., without exception grew up healthy, and not one reacted under the tuberculin test.

Feeding hogs after Tuberculous Cattle. Where cattle and hogs are kept on the same place, it is a common practice to let the swine clean up all food left by the satiated cattle. If there are tuberculous cattle, affected either in the air passages or alimentary tract, the pigs become infected by taking in the expectoration by which the food is soiled, or by rooting around where the cow manure has fallen. This evil is encreasing yearly.

Feeding from a Common Trough. In an infected herd, a common cause of extension is found in the use by the whole herd of a common feeding trough, in which the food soiled by virulent discharges, is taken by healthy animals. The habit of tying a cow in different stalls in succession as she happens to strike one, in place of keeping each cow by its own stall, is a fruitful source of infection. Even when each cow is kept by its own stall, she often becomes infected by reaching into the feeding trough in front of the next cow on the left or right and taking in soiled and infected fodder. In swill stables the evil reaches its maximum, as the feeding trough for 50 or 60 animals is slightly inclined so that the liquid food runs from the supply end to the other, and infecting expectorations are carried in front of all in turn.

Dry, Dusty Stables. Tubercle bacilli are not carried out on the expired air, unless there is forced expulsion as in coughing, snorting or sniffing. In such cases the solid particles are thrown off in masses or fine spray and lodged on surrounding objects. These, together with infecting discharges from bowels, urinary or generative organs, open sores, etc., dry up, and rise on the dust, and, as sterilization occurs slowly indoors, they cause more or less infection of the animal inmates. Cornet, Tappenier and others have thoroughly established this as a common form of transmission, and shown the great importance of cleanliness, disinfection and the removal of infecting materials without raising dust. In an establishment in Paris, a consumptive had served for 3 years. In the following 10 years, 15 of the 20 employés died of phthisis.

Extension through Vermin. As rats and mice (and other rodents) are susceptible to tuberculosis by ingestion (Galtier) it follows that they may become the media for extension of the infection through fodder in which their droppings are scattered,

or from their feeding in the same troughs as the cattle or swine. For swine in particular the danger is greater because of their carnivorous habits ; the rat acquires tuberculosis through eating the offal of the abattoir, or the scraps of the butcher's stall, or kitchen, and the sick rat is thereafter easily caught and devoured by the pig to its own undoing. To block this channel of infection the destruction of vermin about slaughter houses, stables and pig pens is a most important consideration.

Flies and Other Insects as Carriers of Tuberculosis. These congregating on tubercular sores, around the nares or lips, on the skin contaminated by the virulent bowel discharges, on dishes holding infecting milk, on objects soiled by infecting discharges, on diseased carcasses at abattoirs, rendering works and elsewhere, (Spillman, Hofmann, Lartigau, etc.,) and on graves where the earth worms have brought the bacillus to the surface, (Lortet, etc.) become more or less active agents in disseminating the virus. In this way food and water are contaminated, and exceptionally, infection may even be implanted on sores. As the excreta of the flies contain the virulent bacilli, the latter are deposited on windows, walls, furniture, etc., and may be later disseminated in the dust of the apartment.

Dewevre found tubercle bacilli in the bedbugs infecting a bed in which successive cases of consumption had developed, showing that other parasitic or rapacious insects besides flies must be looked on as possible propagators of the bacilli. There is reason to suspect lice, fleas, ticks and acari especially. The same is true of leeches and other rapacious invertebrates.

LESIONS. THE TUBERCLE.

The characteristic lesion in tuberculosis is the tubercle, taking its name from the small rounded nodule which, at first virtually invisible, encreases often to the size of a millet seed, or a pinhead or even larger, and which by confluence with others, forms conglomerate masses of all sizes to which the term tubercle is still applied. Where the bacillus tuberculosis is implanted, the fixed tissue cells are stimulated to an undue proliferation, and a diapedesis of leucocytes takes place from the neighboring capillary blood vessels, the whole eventuating in the formation of a rounded cluster or nest of epithelioid and giant and later small rounded

lymphoid cells in a fine fibrous stroma. Baumgarten and his followers claim that the larger epithelioid and giant cells result from the karyokinesis and proliferation of the fixed tissue cells, epithelial and endothelial cells, and that the lymphoid cells which later (often after the eighth day in experimental cases) invade the lesions, are alone the product of the migrating leucocytes. Metchnikoff, Yersin and others contend, on the other hand, that epithelioid and giant cells are directly derived from the leucocytes and endothelial cells and like these are possessed of actively phagocytic qualities. As the final outcome before caseous degeneration, is the predominance of the small lymphoid cell, it may well be questioned whether this is not the result of active encrease in both kinds of cells, as appears to happen in many other inflammations. For our present purpose it is well to note the early characters : *Centrally* often a large (giant) cell with a number of peripheral nuclei and at times, branching processes ; around this as a *second zone* epithelioid cells of large size, with round or oval nuclei, and sometimes giant cells ; outside this a *third zone* of small rounded lymphoid cells with large nuclei. There is a delicate fibrous network between the cells, but no indication of capillary blood vessels, the absence of which may partly explain the constant tendency to degeneration, necrosis, and caseation. The tissue around the miliary tubercle is red and congested.

Caseation. A striking characteristic of tubercle is the occurrence of coagulation necrosis, beginning in the centre of the specific nodule as a whitish or pale softening and degeneration of the cell elements and gradually extending toward the circumference. The cells, and even the proximate tissue elements, die and disintegrate passing into a structureless, granular or hyaline debris, which has been named from its supposed resemblance to old, ripe, soft cheese. Baumgarten has observed that the small lymphoid cells are the first to degenerate, followed by the epithelioid and giant ones.

While the formation of tubercle is at first a productive inflammation of which the cell clusters and nodules are the result, yet the tendency to necrosis, and caseation is so great that it must be looked upon as one of its most prominent features, and is rarely absent, whether the lesion exists in the connective tissue of the

lungs, liver or other organ, in the lymph glands, in the respiratory or intestinal mucosa, or submucosa, in the oseous cancelli, brain or skin. It is, therefore, largely pathognomonic, yet it is not peculiar to tubercle, being common in glanders, and other infective inflammations. Its presence should always lead to a search for the primary tubercle nodule, with its nonvascular cluster of lymphoid and giant cells and above all for the specific bacillus resistant to staining.

The tendency to extensive degradation and caseation is especially marked in swine, in which the resulting debris is often so liquid that the tubercles bear a strong resemblance to abscesses.

Calcification. The deposition of lime salts (mainly phosphates) in the tubercle is a common feature of advanced cases in man and pig, but especially in cattle. It is unknown in rabbits and Guinea pigs. The tubercle assumes a hard cretaceous aspect and feeling, grates under the knife and crepitates when pressed. This is always an evidence of chronicity, but it has been observed in swine in 3½ months.

Fibroid Degeneration. Fibrosis. In this case a productive fibroid inflammation takes place in the tissue of the tubercle, and it is resolved more or less extensively into a hard, white, resistant body. This is the pearly mass which has given rise to the name of the *pearl disease* (perl-zucht) in Germany. It may be fibrous throughout, but usually a number of the nodules show a caseated centre while the external zone, that nearest to healthy tissue, has alone taken on the conservative fibroid development. Like cretefaction fibrosis is an indication of chronicity in the lesion. It is often seen in man, but still more so in cattle, in which it affects particularly the abdominal cavity, but also to a lesser extent the chest and other parts. It is remarkable for the paucity of bacilli to be found in its substance, often requiring many sections and infinite patience to reveal the presence of the microbe. This comparative lack of actively multiplying bacilli is doubtless one factor, operating in the direction of conservative processes, chronicity and even partial recovery. In experimental tuberculosis the extent and rapidity of development of tubercle, as also of the degeneration are found to be closely allied to the number of bacilli introduced.

Cattle: Pulmonary Tuberculosis. One of the most frequent

seats of tuberculosis, the lung, may in acute cases show a diffuse miliary tuberculosis, a considerable part of a lobe or of several lobes being congested, infiltrated, and filled with small individual tubercles, grayish or transparent in the midst of the general redness. A second form, often of old standing, is in the shape of hard masses, often circumscribed or isolated, and easily felt when the soft lung tissue is manipulated. They are respectively formed by the local aggregation, and confluence of the small miliary nodules and may vary in size from a pea to a mass of ten or twelve inches in diameter. On section the miliary tubercle may not yet show central caseation, but the larger ones do so as a rule. The caseated nucleus may be soft, cheesy and somewhat homogeneous; it may be dry, granular and yellow, resembling damp farina of maize; it may be calcified in its outer portion and invested by a firm fibrous envelope. The tubercle may be the seat of general fibroid degeneration, constituting the pearl nodules (perl-knoten) dense as cartilage and either with or without a soft caseated centre; it may show an agglomeration of nodules in all stages from the early congested nodule to advanced caseation or cretefaction, the whole embedded in a solid congested and infiltrated tissue, largely fibroid. There may be extensive abscesses, the seats of complex infections, having thick uneven walls, often showing *grape like* tuberculous outgrowths, and containing thick, caseo-purulent, viscous, granular, yellowish or greenish and comparatively odorless contents: in other cases the abscesses have burst into the bronchia, leaving *vomicæ* and with the new resultant infections the contents have acquired an offensive putrid odor; finally, there may be extensive inflammatory infiltrations, affecting lobules or lobes, and interspersed with tubercles in the form of the early grayish or transparent nodule, or even more advanced caseous or cretaceous centres. These latter serve to distinguish the condition from broncho-pneumonia, the grayish centres of which might suggest miliary tubercle. The broncho-pneumonia, however, has the whitish centres confined to the bronchioles and their terminal air sacs, and represent their thickened walls and muco-fibrinous contents; they do not show the same tendency to caseation, nor necrosis of the tissue, nor to caseation of the dependent lymph glands, and, above all, they do not contain the bacillus tuberculosis.

A noticeable feature in the tuberculous lungs is the great frequency of tubercles of all different ages from the initial transparent nodule to the caseated or calcified mass side by side. A tuberculous bronchitis is a familiar accompaniment, with lines of miliary tubercles, ulcers and a flocculent (sometimes gritty) muco-purulent discharge, containing elements of the necrotic tissue and bacilli. Emphysema and interlobular œdema are also met with.

The *pleuræ* often suffer by continuity of tissue from the diseased lung, but they may be affected primarily through infection of the circulating blood. The earliest pleural lesions may be congestion, exudation and the formation of false membranes in fringes, but soon these become the seats of minute nodules or forming tubercles which steadily encrease to form pale red cauliflower-like growths—which have been spoken of as *grapes*, from their supposed resemblance to bunches of that fruit. Extensive areas of the mediastinal, costal or pulmonic pleuræ are often completely covered by these productions. Dense, and thick adhesions sometimes form, holding in their substance characteristic tubercles.

The *bronchial and mediastinal lymph glands* receiving as they do the afferent trunks from the lungs and the greater lymph sac of the pleura, offer in their sluggish currents the most favorable culture vessels, and almost always become affected in consequence. Not infrequently they are found to be tuberculous when the tissues which they drain appear to be sound, and we must therefore, conclude that the primary slight lesions in the latter have recovered, or that the bacilli have passed through the tissues and lymph channels without establishing any centre of disease. The glandular lesions are primarily congestion and redness, with more or less infiltration and swelling, followed by a nodular induration, with enlargement or exceptionally shrinking. When bisected they may show all stages of the tubercle from the miliary granule to the caseous, dry, yellow, granular or cretaceous necrosis. By aggregation these often grow to a large size, a long diameter of 6 to 12 inches being not uncommon.

Pericardial and Cardiac Lesions. The pericardium may be implicated from the pleuræ or independently, and though tuberculosis of the heart is rare, it may be the seat of primary tuber-

culosis or of extension from the pericardium or endocardium. In the N. Y. State Veterinary College Museum is a cow's heart, greatly enlarged, and completely invested and invaded by tubercle several inches thick.

Tuberculosis of the Mouth and Throat. Tubercles sometimes form in the *tongue* causing nodular swelling, with a caseated centre. Much more frequently they attack the *pharynx* or *larynx* with the formation of nodular necrotic swellings, followed by ulcers and the implication of the adjacent lymph glands. The glands are liable to be invaded through the tonsillar and other follicles of the faucial and pharyngeal regions, which like the solitary and agminated glands of the intestines form excellent culture vessels. The glands most frequently attacked are the *retro-pharyngeal*, but the *lateral pharyngeal*, the *intra-parotidean* and *sub-maxillary* lymph glands occasionally suffer. They often become indurated, yet the formation of abscess-like sacs is not uncommon especially in the retropharyngeal.

Gastro-Intestinal, Peritoneal and *Mesenteric Tubercle.* Tubercles of the interior of the stomachs are rare, though they are frequent on the peritoneal surface of the first three stomachs, as rounded, subserous nodules varying in size from a pea upward. The *mucosa of the small and large intestines* may suffer, by preference in the seats of the solitary or agminated glands, and the resulting ulcers may extend in the lines of the lymph vessels from the convex to the attached border of the gut. The small greenish caseated and calcified nodules on the intestines, which are so often mistaken for tubercles, are the degenerated cysts of the œsophagastoma. Tuberculosis of the mesenteric lymph glands is much more common, the successive stages being essentially like that seen in the bronchial glands. Besides these the surface of the mesentery, omentum and abdominal parietes often becomes the seat of congestion, exudation and cauliflower-like neoplasms or grapes, as already stated of the pleuræ. In a certain number caseation or cretefaction may be detected.

Liver Tuberculosis. The liver is greatly exposed to tuberculosis as the single destination of all the blood from the gastro-intestinal tract. Tubercles also form on its surface by direct infection from the peritoneum. The hepatic tubercles are often very large and numerous, adding greatly to the bulk and weight

of the viscus. There is usually coincident tuberculosis of the lymph glands of the porta.

Tubercle of the Spleen. This is also a favorite seat of the morbid process, exposed as it is to the reflux of infected blood in the portal vein, and to access of the bacillus from the peritoneum and omental lymphatics.

The *pancreas* is less exposed to the channels of the circulation and is less frequently affected.

Genito-Urinary Tubercles. The *kidneys* are always liable to suffer in generalized tuberculosis, in accordance with their function of elimination and the great quantity of blood that passes through them. The tubercles may be numerous, encroaching upon and destroying the glandular tissue, and determining congestion, nephritis and hæmorrhage (Schütz). The *ovaries*, when tuberculous, lead to nymphomania and sterility; they become swollen, with rounded projections, indurated, and on section show all stages of infiltration, caseation and calcification. They may encrease to a great size. Tubercles are found on the *Fallopian tubes*, the *serous and mucous surfaces of the womb*, and in the *broad ligaments*, in all their characteristic forms. The *bladder* and *vagina* are less frequently involved. In the male the *testicle*, *epididymus*, *tunica vaginalis* and *prostate vesicles* are sometimes affected. The swelling and induration of the affected organ, or the existence of hydrocele, may be noted.

Tubercle of the Udder. From the mass of blood passed through the udder it is specially exposed to infection whenever the bacilli enter the circulation. It may also be directly infected by the entrance through the teat, or a trauma, of the bacillus of the stable dust. There may be for a time only a slight general swelling which leads to no suspicion on the part of the milker, and as the secretion is not arrested, a dangerous product may be distributed. At this stage the lobules on section appear swollen, gray, with paler or yellowish points and minute hæmorrhages. The milk ducts contain coagula and bacilli (Bang). In other cases, usually more advanced, the gland is hard, nodular, enlarged, and shows a marked thickening of the walls of the smaller milk ducts and secreting follicles, with yellowish and even caseous and calcified centres. The gland is often greatly enlarged, the milk suppressed or completely altered, and tubercular neoplasms or ulcers exist in the larger ducts.

Tuberculosis of the Brain and Spinal Cord. As noted in Volume III. this occurs in young cattle especially in the form of tubercles of all ages attached to the pia or arachnoid, on the brain, or cerebellum, or in the ventricles, also in the cerebral matter, in or on the lumbar enlargement of the cord or on its pia. Several cases have come under my notice in the mature cow.

Tuberculosis of the Eye. This has been seen mainly as a result of experimental inoculation, yet casual cases also occur in the cornea, sclerotic, iris or choroid, the product undergoing early necrosis and softening into a yellow mass filling the different chambers.

Tuberculosis of the Bones. Though less frequent than that of internal organs this is not rare. It usually attacks the spongy tissue, near the articular extremities, or the vascular line between epiphysis and diaphysis, giving rise to considerable exudate, thickening of the bone and arthritis. The vertebræ, ribs, sternum, petrous temporal, frontal and occipital also suffer. Sections of the bone show dilatations of the cancelli, filled with the characteristic non-vascular groups of giant, epithelioid and lymphoid cells with, at times, softened and caseated centres. The adjacent bone is congested and softened, so that the detachment of epiphysis and apophyses is not uncommon. In case of invasion of the joints the cartilages and ligaments are the seat of tubercular deposits, softening, fibroid change and caseation, and there is general synovitis. The cartilages of the ear (concha) and nose (septum) may also be invaded.

Tubercles of the Skin. These are not very uncommon in cattle being the counterpart of the tuberculous warts and ulcers of the hands of butchers, tanners and others that handle the products of diseased animals. They may show as little pea-like nodules in the substance of the skin, or immediately beneath, very commonly on the side of the abdomen, where their presence in life furnishes a useful suggestion, as enlarged masses forming raw, warty projections with centres of caseation, or as clusters of warty-like growths of this kind (grapes).

Tuberculosis of the Muscles. This is comparatively rare in cattle, though by no means unknown, appearing as tubercles of the size of a pea and upward, in or between the muscular masses, often showing a caseated centre.

Tubercle of Lymph Glands. No organs in the body suffer more than the lymph glands as they receive through their afferent trunks and develop the bacilli coming from any adjacent tissue to which they are subsidiary. There is also evidence to show that bacilli entering from the lungs or bowels may pass through these without apparent effect, to develop in the connected lymph glands. The frequent infection of the pharangeal, bronchial, mediastinal and mesenteric lymph glands has been already sufficiently noticed, and if the other groups suffer less it is mainly because the tissues from which they derive their lymph are less frequently infected. An intimate knowledge of the different groups of lymph glands is a most essential prerequisite to the diagnosis of tuberculosis in life, and no less to a satisfactory *post mortem* examination. The order of relative susceptibility and importance is somewhat as follows: Bronchial, mediastinal, mesenteric, hepatic, sub-lumbar, mammary inguinal, sub-dorsal, phrenic, intercostal (especially the first and second), pharyngeal, parotid, sub-maxillary, prescapular, prefemoral (stifle), prepectoral, axillary, subital and popliteal.

Relative Frequency of Tubercle in Different Parts in Cattle. The following table gives the seat of tubercle as noted by a number of observers. It is open to the objection that it is the result of examination for diagnosis, and therefore gives the most obvious rather than the complete list of lesions. Examination of the brain, bones, deep muscles and intermuscular glands, etc., was usually omitted. Again, as the animals were often steers, the indications as regards the generative organs are unreliable. The œsophagostoma nodules on the bowels have been so often mistaken for tubercles that I have omitted all reference to intestinal tubercle.

DISTRIBUTION OF TUBERCLES IN DIFFERENT ORGANS OF CATTLE: PER CENT OF AFFECTED CATTLE.

	Right Lung. %	Left Lung. %	Mediastinal Glands. %	Bronchial Glands %	Costal Pleura. %	Visceral Pleura. %	Mesenteric Glands. %	Liver. %	Spleen. %	Uterus. %	Lumbar Glands. %	Liver Lymph Glands. %	Peritoneum. %	Pharyngeal Glands. %	Diaphragm. %	Stomach. %	Udder. %	Iliac and Inguinal Glands. %	Lymph Glands of Udder. %	Lymph Glands of Flank. %	Lymph Glands of Shoulder. %	Pericardium. %	Heart. %	Kidneys. %	Bones. %	Muscles. %	Larynx %
German Abattoirs,	75		29		47 – 55		------	28	19	10	5	1	----	4	0.2	0.16	1	0.06	----	----	----	0.9	---	0.7	---	0.1	0.13
Pearson, (Penna),	63	57	60	7-33 8	22		23	25 20 7	5.7	5.5	---	10.3	14	15.15	11.4	7.2	8.75	----	13.5	9	7.75	8.2	---	2.1	0.4	---	----
Bryce (Canada)	77.27		56.8		11.36		11.36	40.9	---	---	---	----	4.5	-----	2.27	---	----	----	----	----	----	---	---	---	---	---	----
Reynolds (Minn. Agr. Ex. Station),	89.4		33 3-64.9		79		12.5	35.4	27	---	---	8 3	----	18.7	----	---	16.6	----	----	----	----	--	---	2	---	---	----
Law (N. Y. Grade Herds)	34.8		64		6.4		9 6	1.6	---	---	---	----	17.7	6.4	----	---	6.4	----	----	----	----	---	---	---	---	---	----
Nelson (N. J. Exp Station Cows),	33,9		----	----	------		------	28	---	---	---	----	----	-----	----	---	----	----	----	----	----	---	---	---	---	---	----
Russell (Wis. Exp. Station),	55		72 4		------		24	10	---	---	---	----	----	31	----	---	6 9	----	----	----	----	---	---	---	---	---	----
Stalker & Niles (Ia. Exp. Sta.)	36		61		------		------	8.3	---	---	---	----	----	5	----	---	----	----	----	----	----	---	---	---	---	---	----
Reick (Leipsic),	100		----		*57		------	83	18	---	---	----	*57	-----	----	---	17	----	----	----	----	---	---	52	9	49	----

*All serosæ.

Swine. The *lesions* in swine, though essentially like those in cattle, differ in some particulars. The primary tubercles are more commonly pharyngeal or intestinal and mesenteric, in keeping with infection by ingestion ; caseation will often proceed to liquefaction, so that the tubercles appear like grumous abscesses with irregular outlines and vascular growths on their walls ; there is less tendency to cretefaction than in cattle ; the muscular and intermuscular tissues and the lymph glands are far more frequently affected ; ulceration of the pharyngeal and intestinal mucosæ is more common. The lungs, liver, spleen and visceral lymph glands are very subject to tubercle, the kidneys, uterus and testicles somewhat less so, and the nerve centres least of all. The serosæ are often involved and the seat of clusters of vascular or caseated neoplasms (grapes). The bones and joints also suffer, particularly in young growing animals.

Horse. The horse suffers much less frequently than cattle or swine, probably largerly because of his outdoor life, his better tone as the result of muscular work, and the absence of the excessive milk secretion which is secured from cows and of the dangers from ingestion that attend on swine. Yet according to Nocard, it is even more susceptible, and the disease once established is liable to advance more rapidly to a diffuse generalization. The *lesions* in the lungs and abdominal cavity resemble those of cattle, both in nature and abundance ; tuberculous polypi and ulcers are more common ; the visceral lymph glands (bronchial and mesenteric) are early and severely attacked ; the liver and spleen suffer extensively, the serosæ somewhat less so ; lesions have been noted in the vertebræ, skin and muscles (Cadiot). In rare instances tubercles have been seen in the heart, and the aorta has been atheromatous (Cadiot). Necrotic degeneration, caseation and cretefaction occur as in cattle.

Sheep and Goat. Tuberculosis is infrequent in the goat and especially so in the sheep, owing perhaps largely to open air life and their predilection for high, airy pastures. When inoculated they show a marked susceptibility, and under favorable conditions they contract the infection casually. The tubercles may be found in all parts of the body,—lungs, thoracic lymph glands, intestines, mesenteric and sublumbar glands, liver, spleen, serosæ, lymph glands at large, vertebræ, etc. There is less tendency to calci-

fication than in the ox, the older tubercles remaining in the caseated condition or bursting and forming vomicæ. A common seat of casual tubercle in sheep is around the throat or on the sides of the cranium or face.

More frequently than in cattle, verminous affections (œsophagostoma columbiana, and venulosum in the bowels, strongylus filaria and rufescens in the lungs, linguatula denticulata in the mesenteric glands,) are mistaken for tuberculosis, hence the necessity for a careful investigation into the nature of the neoplasm, for the presence of the tubercle bacillus, and the absence of the worms and their eggs. Except in the case of the linguatula the lymph glands are little affected by the worms.

Dog and Cat. The *lesions* are often concentrated on the respiratory or alimentary tract, but they have been noted also in the pharyngeal glands, tonsils, posterior nares, serosæ, liver, pancreas, spleen, nerve centres, ovary, uterus, testicle, epididymus, tunica vaginalis, prostate, heart, aorta, bones and joints. They follow the regular development of bovine tubercle, and caseation and cretefaction are prominent features. These animals are especially liable to infection by eating the left victuals from the plate of a consumptive owner, as well as by devouring consumptive prey, and the primary lesions are to be looked for along the line of the throat, bowels, liver and lungs. For the dog Cadiot records caseating polypi and ulcers on the mucosa of the larynx, trachea and bronchia, and Müller and Cadiot several cases of pharyngeal caseating adenitis in the *dog* bursting externally and developing intractable fistulæ, having abundance of bacilli in the discharge. These he attributes to expectorated virus from old-standing tubercles in the chest infecting the pharyngeal mucosa and indirectly the lymph glands. The infection entering with the food and lodging in the follicles of the tonsils would act in the same way, and infected wounds received in fighting must also be quoted. *Cats* suffer from similar intractable sores, some of which may be traced to a tuberculous origin. The lungs are often extensively hepatized and of a general pale grayish color, but the early miliary lesions, the caseating and cutaneous centres, the vomicæ often intercommunicating, the tuberculous bronchial and mediastinal glands and the bacilli show the true nature. Intestinal ulcers are common, especially on the agminated glands,

and small tubercles, in all stages of degeneration are met with in the enlarged liver, the spleen, pancreas, kidney, etc.

Apes and Menagerie Animals. In these tuberculosis is common alike in the thoracic and abdominal forms, and the lesions in the main are those of domestic cattle.

Chickens. The *lesions* are common in the abdominal cavity, the intestines, liver and spleen being the most frequently attacked, while the subcutaneous connective tissue, bones and joints also suffer. The lungs and kidneys usually escape. The intestinal mucosa shows small nodules, often caseated, or ulcers; the enlarged and friable liver is studded with tubercles from the size of a hemp seed upward, gray or translucid, homogeneous or with central necrosis, singly or in conglomerate masses, with congested or hæmorrhagic periphery; spleen is swollen and permeated by similar deposits; fibrinous ascitis is not uncommon; the abdominal lymph glands are enlarged and congested.

The early tubercle shows a central, necrotic, hyaline area, consisting of the debris of disintegrated cells, which is colored brown by picro carmine, unlike the nucleus of the pheasant tubercle (Cadiot). Around the hyaline centre is a zone of large epithelioid cells, the nuclei of which stain strongly in carmine. Outside this is the usual zone of small, round, lymphoid cells. In the whole the tubercle bacilli can be made manifest by the carbol-fuchsin (Ziehl-Neelsen) stain. In the older and larger tubercles the central necrotic mass has encroached in part or in whole on the epithelio-lymphoid zone.

Pheasant. The *lesions* have the same seats and naked-eye aspect as the chicken tubercle, but under the microscope the smallest and most recent tubercles show epithelioid cells to the centre, or later, the central zone presents a dense fibrous network enclosing open spaces and giving a mahogany stain with Lugol's solution (iodine and potassium iodide) (Cadiot). There has been an organisation of connective tissue which has submitted to amyloid degeneration, making a clear distinction from the tubercle of chicken.

Parrot. The *lesions* were thus located by Eberlein and Cadiot:

	Cadiot	Eberlein.
Eye and periocular region,	12	14
Commissure of the beak,	7	11
Tongue,	8	9
Palate,	4	—
Larynx,	—	2
Bones and Articulations, Upper limbs (wings),	7	14
Bones and Articulations, Claws,	3	—
Bones and Articulations, Cervical, Dorsal and Caudal	5	—
Lungs,		7
Liver,		4
Intestine,		3
Muscles,		1
Heart,		1

The *skin lesions* are vascular neoplasms containing bacilli and usually invested with a covering of horn, but sometimes, on the legs and feet, raw. The morbid growth may be rounded or conical, narrowing to a point. The *lesions of the buccal mucosa* begin as small, grayish swellings on the angle of the mouth, palate, tongue or larynx which grow out into more or less rounded vegetations. The *lesions of the liver and lungs* are mostly miliary with the usual tubercular features, but they may grow to larger size, as a pea or bean. In the *cancelli of bones* and on their surface, the lesions resemble those of the mammal.

The cutaneous form has been held to be the counterpart of warty lupus of man, the more plausibly that the disease is developed by inoculation from tuberculous men. The arthritic type represents what is described as gout in parrots.

In any case the recent miliary lesion presents the true tubercular type of a central giant cell or cells with bacilli, surrounded by epithelioid cells, and they in turn by small, rounded lymphoid globules.

PRIMARY AND SECONDARY INFECTION.

The estimate of the relative, early or late infection of two organs or tissues may often be made with reasonable accuracy from the fact that the lesions in one organ are old, caseated, calcified or sclerosed, while those in the other organ are all recent, with vascular environment and almost devoid of caseation or other degenerative process. We cannot safely predicate our decision on the greater number of old lesions in one organ rather

than another, as the disease may have advanced much more rapidly in the one issue. Still less can we state with certainty that the disease has not entered by a given channel because no lesions are left to show the transit of the bacillus along its supposed course. We frequently find tuberculosis of the bronchial or mesenteric glands, when we can detect no lesion in the lung, nor intestine. The bacillus has been passed on without establishing any lasting lesion in transit.

It is often too confidently asserted that the infected dust inhaled, falls directly on the air cells and determines the extensive pulmonary tuberculosis that ensues. So on the other hand, it is often too arrogantly assumed that tubercle bacilli ingested with food, must necessarily show their results mainly in the intestines, mesenteric glands and liver. That solid particles can find their way directly into the lungs, has long been demonstrated by the pulmonary anthracosis of the miner, and the deadly phthisis of the stone hewer and cutler. The experiments of Cornet, Tappeiner and others in producing pulmonary tuberculosis, by compelling the inhalation of infected spray, corroborate this experience very satisfactorily. Yet it does not follow that all of the offensive matter penetrated the air cells at once on the air inhaled. The heavy particles of steel, quartz and even of coal dust must be mainly arrested on the surface of the moist air passages, yet, under the irritation caused by their presence and the consequent arrest of the ciliary motion, they would slowly gravitate downward to the pulmonary cells. In the case of the inspired tubercular spray or dust, we must recognize the possibility of the approach of the bacillus to the lungs through the lymph and blood channels as well. The bacilli lodged in the pharynx, and, above all, in the tonsillar follicles, can readily enter the lymph vessels, and are finally poured into the lower end of the jugular vein, but a few inches from the right heart, by which they are instantly propelled into the lungs. If then the lung is the most receptive and least resistant organ, it may easily be that this is the first point where the bacillus can establish a strong and effective colony. Apart from this the colonization of the tonsillar follicles may determine a constant supply of fresh bacilli, which may gravitate down with the abundant mucus toward the lungs.

This tuberculous colonization of the tonsillar follicles is doubtless the main source of the infection of the pharyngeal lymph glands, which is so common in ox, pig and dog. It tends further to intestinal tuberculosis through the frequent swallowing of the products of the infected follicles. Then the infection of the pharynx and tonsils, whether established by inhalation or deglutition, may be the first step toward a secondary infection of the intestines.

Again the bowels can be infected by the frequent swallowing of the expectorations brought up from the diseased lungs or bronchia.

Conversely the lungs may be easily infected secondarily from preëxisting disease of the abdominal organs, and again primarily through the lymph channels. With tuberculosis of the cardia or liver the bacilli can follow the lymph vessels of the œsophagus or vena cava so as to reach the mediastinal glands, and from these glands in a state of disease they can easily pass into the adjacent pleural cavity and reach the lung. Or by following the mesenteric lymphatics they reach the thoracic duct and enjoy what is virtually for them a culture fluid, until discharged into the jugular, which, as already stated, is but a few inches from the right heart and lung.

Extension through the general blood stream usually takes place only when tubercles have already become numerous and extensive in a given region of the body, and its occurence is the signal for a generalized tuberculosis.

The extension from the gastro-intestinal organs, pancreas and spleen may be considered as a partial exception. Here the infecting blood is not the general blood stream, but has to run the gauntlet of the liver capillaries by which the bacilli may be sifted out and delayed. This arrest subjects the liver to secondary tuberculosis in almost all cases of abdominal tuberculosis, and goes far to explain the extraordinary frequency of the disease in this organ. Ingestion tuberculosis almost necessarily leads to hepatic tuberculosis, and this notwithstanding that the primary lesion may have been very circumscribed.

The tuberculization of such a large vascular organ as the liver, however, paves the way for further extension, and if once extensively diseased, the early generalization of the infection is to be dreaded.

TUBERCULOSIS CONTINUED.

SYMPTOMS OF TUBERCULOSIS IN CATTLE.

Microscope and staining; by centrifuge; by agglutination test; by inoculation; by tuberculin test; tuberculin; reaction; precautions, temperature before injection, exclusive of other illness, of parturition, abortion, heat, isolation, of hot building, of cold draughts, of hard floor, of faulty milking, privation of water, of change of food, of journey, of rude handling, of previous recent tests, of anti-pyretics, make special examination in unthrifty, test excluded animals later, danger of infecting through thermometer; technique; dose; time of injecting; identification and record of subjects; seat of injection; sterilization of syringe and skin; temperatures hourly or every second hour from 8th to 16th or later; typical reaction; accurate record of feeding, watering, milking or other occasion of hyperthermia or hypothermia; passing on the value of a rise; local swellings; chills; tremors; effect of test on general health of reacting and non-reacting animal; action on parturient cows; reliability of test. Relation of human to bovine bacillus; varying character of microbes generally, of tubercle bacillus in man; bacillus of man and bird interchangeable; tuberculosis in man and ox, similarity, coextensive, apparent exceptions, direct infection—man to cattle—cattle to man; encrease of tabes; experiments of Adami, Ravenel and others; experiments on cold blooded animals; bearing of variations of susceptibility on sanitary work. Treatment: when admissible; hygiene; rich feeding; oleaginous seeds; cod liver oil; pneumatic cabinet; grooming; warm bath; medicated inhalations; sulphurous acid; chlorine; formaline; calmatives; carbonate of creosote; derivatives; streptococcic serum; air or oxygen in peritoneum; excision of tubercle. Prevention; Extinction in cattle; expense, supply of tuberculin and efficient veterinarians; appraisment; indemnities; vermin; disinfection; scheduling; control of purchases. Breeding healthy stock from infected; raising healthy offspring without sterilizing the milk. Removal of unthrifty and suspicious animals. Removal of animals showing objective symptoms, or, tuberculin reaction. Generally applicable measures. Hygiene of milk, butter, cheese, whey, oleomargarine. Hygiene of meat.

Tuberculosis may be acute or chronic, yet as seen in cattle, from casual infection, it usually comes on very slowly and insidiously and follows a chronic course. As the symptoms vary according to the organs involved it will be convenient to consider these in turn, beginning with such as show the most diagnostic phenomena.

Pulmonary Tuberculosis. Though one of the most common and dangerous forms of the disease this may last for months or

years without any suspicion on the part of the owner or caretaker of anything amiss. There may be an occasional cough, short, weak, dry, wheezing, perhaps repeated and roused by opening the stable door in cold weather, by leaving the stable for the cold outer air, by rising suddenly in the stall, by being driven on a run for a short distance, by drinking cold water or by eating dusty food. If driven for some distance, or put to draught work (ox) the subject blows more than the others. Sometimes even at rest, breathing is slightly accelerated. Yet the spirits may be as good, the eye as clear and full, the coat as smooth and sleek and the skin as soft and mellow as in health. Some such animals give as much milk, of as rich a quality, or, when put up to fatten, lay on flesh apparently as well as their healthy fellows. In favorable cases percussion may elicit circumscribed areas of dulness, and wincing or other sign of tenderness, and auscultation may detect crepitation or wheezing over the same points. By covering the nose and mouth with a sac or blanket the breathing is rendered more labored and the morbid sounds become clearer and more definite. The use of a stethoscope or phonendoscope may also render them more distinct. The morbid râles are more significant of tuberculosis if found in a number of isolated and circumscribed spots, with healthy respiratory murmur between, than if simply surrounding a single extended area of flatness as is usual in pneumonia. Much, however, stands in the way of success at this early stage. The heavy muscular and bony mass of the shoulder covers the anterior lobe and partially muffles the auscultation sound, while it renders percussion useless. The thick covering of the ribs in fat animals proves a serious barrier to successful auscultation and percussion. The varying plenitude of the abdominal viscera, and the rumbling, trickling, gurgling, and in the case of the rumen the crepitation of the contents, tend to complicate, obscure or cover up the pulmonary sounds. There is usually no appreciable elevation of temperature, or a slight rise of about one degree takes place at distant and uncertain intervals so as to render it useless for purposes of diagnosis.

When the disease is more advanced and the pulmonary lesions more extensive, the animals usually appear less thrifty on the same feeding, yet fat animals are habitually killed for food that show quite extensive pulmonary tuberculosis. With loss of

condition, the coat loses its lustre somewhat, the hair becomes dry and stares in patches, and the skin loses its mobility and mellowness. The cough may be more frequent, perhaps paroxysmal under excitement, harsher, more short and broken, and either dry and husky or moist and gurgling, with a succeeding deep inspiration, perhaps a moist râle. When the skin on the last ribs is pinched up between the fingers and thumb it is slower in flattening down to its normal smoothness, pinching of the spine at the shoulder or back, or it may be of the sternum, may cause wincing and even moaning, and the same may come of percussing the ribs smartly with the closed fist. There is now more decided evidence of flatness on percussion on the various affected points, and of abolition or lessening of the respiratory murmur, which is replaced by wheezing, or by bronchial blowing sound, heart beats and abdominal crepitation or gurgling, conveyed to the ear more clearly through the intervening consolidated tissue. The breathing may be slightly accelerated even at rest, and becomes distinctly so on exertion. The appetite fails somewhat and the secretion of milk lessens, or it may become more pale and watery. Chronic tympany occurring after meals occasionally appears, usually indicating tuberculosis of the glands along the œsophagus with pressure on that organ impairing eructation, and on the vagus nerve so as to impair nervous control. In connection with this there come on signs of generalization of the tubercle, as irregularity of the bowels, or enlargement or nodular induration of some of the superficial lymph glands, as the pharyngeal, prescapular, prepectoral, axillary, prefemoral, inguinal or mammary. Expectoration is usually abundant but it is difficult to secure it for diagnostic purposes since the moment it reaches the pharynx it is instantly swallowed, while any that may have been projected into the anterior nares is licked out by the pointed tongue. Nocard tried to secure this through a cannula passed in between the tracheal rings but with very little success. Others have introduced the hand into the pharynx, rousing the cough by tickling the larynx, and attempting to bring out the expectoration in the hand. When it can be secured its solid and opaque flocculi may be stained and examined for the bacillus, or it may be inoculated on a Guinea pig, intraperitoneally, to test its virulence. If there are open vomicæ or complex infection the breath is usually heavy and mawkish.

In the most advanced stages the symptoms are very characteristic. The subject is miserably thin and wastes visibly from day to day, the hair is dry and erect, most marked along the spine, the skin is scurfy, rigid, lousy, and clings firmly to the bones, the eyes are pale, deeply sunken in their sockets and bleary, the tears running over the cheeks, while a yellowish, granular, fœtid, and often gritty discharge flows from the nose, and dries in masses around the alæ. The cough is weak, painful, paroxysmal, and easily roused by pinching the back or breast or percussing the ribs. The breathing is liable to be hurried, even panting, and the animal may stand most of its time with nose extended to obviate the oppression that comes of recumbency. All the visible mucosæ are pale and blanched, and the pulse weak and rapid with every indication of anæmia. The temperature is usually raised to 104° to 105° F. and the milk secretion is completely arrested. Indications of generalized tuberculosis become more marked in the enlarged glands, diarrhœa, and clouded (purulent), or blood stained urine with microscopic casts, and even anasarca. The morbid sounds in the lungs have become a complex variety according to the nature of the lesion, blowing, wheezing, amphoric, friction, creaking, mucous, with the other bruits conveyed from adjacent organs. Death usually occurs in a state of complete marasmus, after months or years of illness.

Tuberculosis of the Abdomen. This usually affects the intestines, mesenteric glands, peritoneum, liver, spleen and pancreas, and has been known as *tabes mesenterica.* The generative organs also occasionally suffer, in which case, an early and rather persistent symptom is sterility, with a too frequent or it may be persistent desire for the bull (nymphomania). There is usually a steady loss of condition in spite of good feeding, the impaired functions of the intestinal mucosa, but especially of the mesenteric glands, liver and pancreas, interfering seriously with absorption and assimilation. The victims are therefore known as piners. While there may be more or less fever, highest in the evening, this is by no means marked, and cough and respiratory trouble may be entirely absent. Indications are not lacking, however, of digestive trouble. Slight tympany may follow meals, and the bowels are irregular, costiveness alternating with diarrhœa. If heavy feeding is resorted to, diarrhœa is the usual result, accom-

panied, it may be, by colic and tympany. There is a tendency to formation of pea or nut-like nodes under the skin of the flank, and Kleinpaul claims that the tubercles or vegetations on the rumen can be felt by manipulation of the left flank. Clearer evidence can often be had by rectal exploration, the tubercles and enlarged glands being felt on the rumen, in the knotted mesentery and in the sublumbar and subsacral region.

In case of *uterine tuberculosis*, the nymphomania may be supplemented by a purulent discharge, and rectal exploration may detect the tubercles on its surface, in the broad ligaments or even in the enlarged ovaries.

Great fœtor of the fæces may indicate ulceration of the mucosa, indigestion or impaired hepatic function.

For a great length of time the disease may be virtually confined to the mesenteric or portal glands, or even to the spleen, while the animal enjoys fairly good health. Again, in some instances, the subject may be fat and sleek, though the rumen, omentum or mesentery is to a large extent literally covered with tuberculous vegetations. The tuberculization of the intestines, mesenteric glands, liver or pancreas interferes far more with the general health than does even extensive peritoneal tuberculosis.

As the case advances it tends to generalization and winds up with the general symptoms predicated above of pulmonary tuberculosis.

Genital Tuberculosis in the bull is associated with nodular swelling of the testicle, epididymus or cord, hydrocele, and exceptionally tubercle on the penis, or in the prostatic sac.

Mammary Tuberculosis. This may be primary and circumscribed under direct infection through a trauma or by the milk ducts, but more commonly it is secondary to generalized tuberculosis. It may be a rather firm, uniform, painless swelling of one or two quarters (usually hind ones) without at first serious interference with milk secretion. As the disease advances, the follicles and ducts being invaded, an irregular knotted condition is developed, the milk becomes pale, watery, semi-coagulated and filled with bacilli, and the climax is reached in a densely indurated condition of the gland. From the first the mammary lymph glands, behind and it may be in front of the organ, become

swollen, and they are finally indurated as caseation or calcification ensues. The superficial inguinal glands often participate.

Pharyngeal Tuberculosis. In this a rather common localisation the retro-paryngeal glands and those on the side of the pharynx especially suffer, though the parotidean lymphatic gland and the sub-maxillary often participate. Enlargement and induration of the tonsils and ulceration of the mucosa may be present. There is distinct swelling of the throat or displacement downward of the larynx, and the enlarged or hard nodular, perhaps even shrunken, glands may often be detected by manipulation. The nose is carried slightly protruded and a stertor or wheeze accompanies the breathing. A glairy liquid may run from the mouth or nose, and in this, bacilli may often be detected under the microscope. The retro-pharyngeal glands are very subject to softening and liquefaction, and in such a case an obscurely fluctuating swelling may be detected above the pharynx when the hand is introduced through the mouth.

This usually terminates in generalized tuberculosis, though it will often remain for a length of time the one appreciable localization.

Cutaneous Tuberculosis. The counterpart of *tuberculosis verrucosa* of man, this probably usually occurs by direct inoculation in a sore, yet the infection may reach the seat of lesion through the blood. It is usually represented by an irregular clustering warty growth, hanging more or less loosely from the skin and showing at points caseating centres. Bacilli may be recognized under the microscope.

Glandular Tuberculosis. Aside from the tuberculosis of the internal lymph glands already referred to, tubercles may form in any group of lymph glands, causing swelling, induration, fibroid degeneration or tuberculous abscess. Among these may be named the glands in front of the stifle or shoulder, at the root of the ear, beneath the zigoma, in the jugular furrow, the prepectoral, axillary, inguinal, etc.

Tuberculosis of the Brain and Meninges. Disorder of the cerebral functions occuring in generalized tuberculosis may be held to point to this disease. The earlier meningeal symptoms are often those of excitability, timidity, spasms, visual troubles, etc., merging later into vertigo, hepetude, paresis, unsteady gait, local paralysis and coma.

Tuberculosis of the Eye. This has been seen mostly as the result of experimental inoculation, with conjunctival and sclerotic congestion, corneal opacity, and the development of yellowish centres in the iris and choroid, from which the tubercle extends into the chambers.

Tuberculosis of the Heart, Pericardium or Pleura. Tuberculosis of the pleura is usually a concomitant of pulmonary phthisis, yet it may exist for a time independently, and its diagnosis presents serious difficulties. There may be tenderness of the intercostal spaces, a friction sound in case of raw granular surface or vegetations, creaking from false membranes or even flatness low down on percussion. If these escape notice, a short painful cough and slightly hurried breathing under exertion, in the absence of objective symptoms of lung disease, may lead to suspicion, but the true nature of the affection must remain in suspense. It usually leads to pulmonary or glandular (bronchial), and finally generalized tuberculosis.

Pericardial or Cardiac Tuberculosis is usually secondary and may be suspected when friction is synchronous with the heart sounds, when the heart beats or sounds are irregular or intermittent, or when the area of cardiac dulness is greatly encreased.

Tuberculosis of Bones and Joints. This is more common in calves and growing cattle, but may be present at any age. As affecting the vertebræ it causes stiffness and unsteady gait, perhaps what was looked on as a simple sprain causes persistent lameness in spite of treatment, and a point or area of tenderness on pinching is manifest. In the large bones and joints of the limbs, the cancellated extremities, or it may be a simple process with its inserted tendon or ligament, shows a firm, persistent swelling, and there is acute synovitis of the joint. In acute cases in calves the epiphysis may become detached from the diaphysis so as to make the limb useless. In the mature animal the enlargement and lameness may last for years without material change. The condition may be difficult to diagnose, in the absence of signs of tuberculosis elsewhere in the body, or unless the synovia withdrawn through a sterilized nozzle, with antiseptic precautions, should show the presence of bacilli.

SYMPTOMS OF TUBERCULOSIS IN SWINE.

In young pigs, infected by the milk of the dam, there are general unthriftiness, stunted growth, emaciation and unhealthy skin, encrusted with a dark unctuous matter or scurf, as in chronic hog cholera. Temperature is variable on successive days, or times of the same day. Digestive disorder is manifested by slight colics, diarrhœa, vomiting, tympany and abdominal tenderness. The pig becomes pot-bellied, with hollowness of the flanks in front of the iliac bones, and manipulation may detect the tuberculous bowels and mesentery in the form of a knotted mass.

Roloff describes a caseous colitis with ulceration of the mucosa, which is probably tuberculosis.

Enlargement of the superficial lymph glands (pharyngeal, inguinal, prescapular) may be present. Traumatic infection of the castration sore and inguinal glands has been noted. As the disease becomes generalized, implicating the lungs, there is a dry paroxysmal cough and hurried breathing, becoming more oppressed on the slightest exertion. If quiet and thin enough for auscultation and percussion the usual morbid lung sounds can be heard. Unlike cattle, pigs are very subject to muscular and intermuscular tubercles, and as there is a general tendency to caseation, these are usually to be found as saccular cavities with soft, sometimes liquid, caseated contents. The bones and joints may suffer, as in cattle. The tonsils are usually enlarged and even caseous. The outer auditory meatus and the interior of the eye have been found affected. Cases affecting the brain were manifested by nervous disorder, rearing up on the fence, turning in a circle, spasms, rolling of the eyes, paresis and paralysis, often hemiplegic. When one or other of these indications of local disease is found associated with the general disorder of the lungs or bowels, in a herd fed on raw meat scraps, milk, or the soiled food of tuberculous animals, the evidence is strongly in favor of the local tubercle corresponding to the symptoms. It is noticeable that diagnosis by microscopic examination is difficult and uncertain because of the relatively very small number of the bacilli. In the mature pig the disease may be difficult of diagnosis without tuberculin, and a *post mortem* examination may be necessary to identify the disease in the herd.

SYMPTOMS OF TUBERCULOSIS IN THE HORSE.

Though not a common disease in the horse yet a large number of cases are on record, and accidental and experimental cases alike show that this animal is peculiarly receptive to the disease. The early symptoms depend on the location of the primary lesions, yet the general phenomena of debility, langour, early fatigue, unfitness for violent efforts, perspiration on slight exertion, irregular appetite, occasional rises of temperature and emaciation may usually be noted. The advance is generally slow, almost imperceptible, with periods of improvement and aggravation.

Some cases have appeared in the submaxillary and pharyngeal lymph glands with sore throat and were for a time mistaken for glanders (Ehrhardt, McFadyean). Others show a swelling of the appearance of cold abscess in the seat of the prepectoral glands (Johne, Röbert). One showed widely distributed lymph nodes and enlarged and indurated lymph glands (prepectoral, inguinal etc.) with thickening of the intervening lymph vessels (Cadiot). This last suffered from bronchitis two months before. A number of cases reported by McFadyean showed special stiffness of the neck, with swelling and distortion of the vertebral joints, due to a tubercular osteitis and periostitis, and associated as necropsy showed with internal tuberculosis. Cases of this kind occurred in the practice of McConnell, Dawes, Insall, Malcolm and Hill, so that tuberculosis may well be suspected in cases of disease of the cervical vertebræ. In all of these cases post mortem examination, performed at once, or after a long delay, showed generalized internal tuberculosis.

When the chest is extensively affected, the symptoms are those of broncho-pneumonia or heaves (broken wind), there is hurried breathing with paroxysmal cough sometimes dry and wheezy, at others moist or mucous, a double lift of the flank in expiration, and a mucopurulent discharge from the nose, sometimes streaked with blood. Auscultation detects a varying force of the respiratory murmur at different points, with more or less wheezing. Blowing and other sounds conveyed to the ear through the solidified lung tissues are more rare or less marked. Percussion may show general resonance, encreased at emphysematous points, and diminished in small circumscribed areas, the seats of tubercle or

consolidation. There are of course the attendant debility, inappetence, fever and steadily advancing emaciation. In some cases the enlarged tracheo-bronchial glands are seen to bulge forward between the two first ribs, and by the sides of the trachea. Stocking of the limbs is frequent.

Abdominal tuberculosis is most common in the young foal and when not secondary, may be due to feeding on tuberculous milk, especially in those brought up by hand on cow's milk. There is debility, emaciation, anæmia, irregular appetite, digestive disorder, constipation alternating with diarrhœa, colics, pot-belly, falling in beneath the lumbar transverse processes, dry, harsh, scurfy skin and advancing marasmus. Rectal exploration will usually detect the enlarged sublumbar and mesenteric glands, and the simultaneous manifestations of disease of the lungs, superficial lymph glands, throat, or bones serve to identify the disease.

In the advanced stages of tuberculosis in solipeds polyuria is a frequent phenomenon (Nocard) tending to hasten the general anæmia and marasmus.

The patient often works for months or years but with gradually encreasing debility, which is soon fatal after the occurrence of generalized tuberculosis.

In estimating the nature of the disease, indications may often be drawn from the environment, feeding, etc. Nocard as the result of a careful study of the morphology and habits of the bacilli has shown that the more purely abdominal form of equine tuberculosis is near akin to that of the chicken, while the pronouncedly pulmonary form resembles the human type. McFadyean on his part adduces a number of cases of tuberculosis in horses that had been fed largely on cows' milk, a most significant fact considering that such a ration is rare for this animal, and that few horses contract tuberculosis casually. Exposure therefore to the sputa of man, of the nasal or bowel products of birds or to the milk of cows, may suggest the probability of tuberculosis in a horse with chronic anæmia, debility and wasting.

The discharges from the nose of the affected horse are more available than those of cattle for examination and experimental inoculation, yet in many occult cases the diagnosis by tuberculin is the only reliable resort.

SYMPTOMS OF TUBERCULOSIS IN SHEEP AND GOATS.

The tubercles have been usually found post mortem in these animals or as the result of experimental inoculation, and symptoms have not been well recorded. They follow the same order as in the ox, weak husky cough, wheezing and other râles in the lungs, disorders of the digestive organs, swollen lymph nodes and glands, and caseated products in those that were of some standing. I have found these latter especially, in the region of the throat in high bred rams kept in confined buildings or yards and highly fed to prepare for *letting* or sale. These lose in vigor and activity and *scrofulous* swellings form on the neck, head or elsewhere and become rapidly caseated. In German abattoirs tuberculous sheep proved 0.1 to 0.15 per cent. In Saxon goats the percentage was 0.6.

SYMPTOMS OF TUBERCULOSIS IN DOGS AND CATS.

These follow in the main those of consumptive cattle. As the infection generally enters with food, the early symptoms often point to disease of the throat and alimentary tract, while the later ones involve those of the respiratory organs as well. Impaired and capricious appetite, debility, early exhaustion under exertion, emaciation, sunken pallid eyes, apathetic expression of the face, lack of life and gaiety, a knotted feeling of the abdomen if the region is flaccid, and a tense fluctuating sensation if ascitic, with usually enlargement of the superficial lymph glands are noticeable.

When the chest becomes affected there is the hurried breathing, quickly encreased by exertion, panting, paroxysmal cough, wheezing, and the various morbid râles in the chest, crepitant, friction, creaking, blowing, cavernous, mucous, etc. On percussion, flatness is detected in limited areas in a number of centres. Expectoration is usually promptly swallowed and can only be secured with difficulty for examination.

When tuberculous sores and fistulæ occur in the region of the throat or elsewhere, the evidence is patent and the bacilli can easily be found in the discharges.

In *cats* the course and symptoms do not materially differ. In both animals the history usually shows the connection of house life and the habit of eating after tuberculous persons.

SYMPTOMS OF TUBERCULOSIS IN BIRDS.

In the gallinaceæ there may be inappetence, vomiting, diarrhœa, with hurried breathing, sneezing, and the general phenomena of debility, weakness, advancing emaciation and anæmia, the comb and wattles becoming pale and flaccid and the visible mucosæ bloodless. The eyes are sunken and lack lustre, the head sinks, the wings and tail droop, and weight is steadily lost. When the bones and joints of the legs and wings are involved the local swellings and distortions are visible indications of the trouble.

In *parrots* these local swellings and particularly the horn-covered vegetations on the face and around the beak are characteristic.

Canary. Tuberculosis is common in the canary, contracted, as in the parrot, from man, with whom alone the caged bird comes into dangerous contact. The interchange of the disease between pet birds and their owners would demand the exclusion of such from the rooms of consumptives, and a careful watch for indications of disease of the air passages with marasmus, that the bird may be disposed of before it has become a source of danger.

DIAGNOSIS OF TUBERCULOSIS.

It is needless to repeat the various symptoms of tuberculosis according to its different seats and the degree of its extension in the animal body. In cases in which the indications are slight, greater importance may be given to them through the knowledge of the existence of more advanced or decided cases in the same herd, or the necropsies of animals taken from it. Yet in the average herd it is safely within bounds to say that three-fourths of the affected cattle will escape condemnation if we employ objective symptoms alone. In one herd of seventy head, in which the tuberculin test condemned twenty-four head (being 50 per cent. of the mature animals), I left the examination after slaughter to the veterinarian of the A. J. C. C. who was at the time skeptical as to the value of the tuberculin test. He wrote me afterward of his surprise at finding every one of the twenty-four condemned animals tuberculous, when not one of them had shown symptoms by which he could recognize the disease in life. This is no exceptional case, and may be advanced rather as a typical example of the ordinary infected country herd.

Microscopic detection of the bacillus in the expectoration may be successful in the horse with pharyngeal or pulmonary tuberculosis, but fails in those forms that affect the other internal organs. It is all but useless for the expectoration of cattle and dogs. When there is cutaneous tuberculosis or a tuberculous fistula this is much more valuable, and it is especially useful in dogs and parrots.

The precipitate in the centrifuge will often show the bacilli that are present in milk, but in very many cases of tuberculosis the bacilli are not present in the milk.

The centrifuge used on the *urine* may also succeed when kidneys, bladder or prostate are affected, but the bacilli are rarely found in the absence of disease of these organs. The smegma bacillus is a source of fallacy.

The serous effusions in the affected *serosæ* (pleura, peritoneum, pericardium, synovial cavities) may also be centrifuged and the presence of the bacilli revealed.

The *agglutination test* of Arloing and Courmont though often giving positive results, (95.5 per cent. in pulmonary tuberculosis, 50 per cent. in surgical, Arloing and Courmont ; 40 per cent. Knopf ; 25 to 50 per cent., Lartigan) ; yet proved too unreliable, and frequently gave positive results when tuberculosis was absent. The best medium for cultures to be so used is 6 per cent. glycerine bouillon, and the age of the tuberculosis culture 8 to 12 days. One part of fresh blood serum of the suspected animal in a sterilized capillary tube is added to ten parts of the bacillus culture, and the tube placed in an oblique position. In 2 to 24 hours a fine sand-like material precipitates along the sides of the tube, and the microscope shows the bacilli in clumps, absolutely still without even Brownian movements. Gallemaerts found that it proved very satisfactory with the serum of Guinea pigs after three days from intraperitoneal inoculation, was less marked after inoculations subcutem, and that in man the agglutination appeared in influenza and pneumonia in the entire absence of tuberculosis. Such an uncertain test cannot be utilized in veterinary sanitary work.

Experimental inoculation with milk, expectoration, morbid discharges, the scraping of nodules, etc., is much more searching, and will detect more cases than the microscopic examination.

But it fails entirely in cases in which the milk of unquestionably tuberculous animals is free from bacilli, or in which the local nodule or discharge tested is itself free from tubercle. It is a test of the local lesion and not of the entire animal system.

In choosing a subject for inoculation, the first consideration is that it must come from a healthy stock and be itself free from tuberculosis. Next, it must be of a species actively susceptible to the habitual tuberculosis of the animal from which the inoculated matter is taken. Thus for man, ox, dog and parrot, the Guinea pig is especially appropriate, while for gallinaceæ and horses, the rabbit is to be preferred. Inoculation is usually made into the peritoneal cavity.

As a period of two or three weeks is usually necessary to allow of an extensive development of tuberculosis, the method must be too often discarded on account of the delay in obtaining results.

Tuberculin test. Many stock owners still entertain an ignorant and unwarranted dread of the tuberculin test. It is quite true that, when recklessly used by ignorant or careless people it may be made a root of evil, yet as employed by the intelligent and careful expert it is not only perfectly safe, but it is the only known means of ascertaining approximately the actual number affected in a given herd. In most infected herds, living under what are in other respects, good hygienic conditions, ⅔ or ¾ are not to be detected without its aid, so that in clearing a herd from tuberculosis and placing both herd and products above suspicion the test becomes essential.

Tuberculin is the bouillon in which the tubercle bacillus has been grown, charged with the toxic products of its growth, but which has been raised to a boiling temperature to destroy all germ life, and from which the dead germs have been removed by passing it through a porcelain filter. When a physiological dose of this has been injected, subcutem, into the suspected animal, it has no effect on the non-tuberculous, while in the tuberculous it produces, in the course of the next 24 hours (usually from the 8th to the 16th), a steady rise of temperature by 2° F. or more, followed by a slow subsidence to the normal. This may last for from three to ten hours in different cases.

Among the precautions may be named :

1. The temperature of the animal is best taken at intervals, or at least, morning, noon and night, on the day preceding the injection to see that the subject shows no habitual rise at any time of the day. Yet in busy field work the one night temperature taken just before injecting will rarely fail to give a satisfactory normal as a standard for the animal. Any quotidian rise almost invariably reaches its climax at night.

2. *The subject must be in good general health.* If there is present in the system any concurrent disease it may undergo an aggravation within twenty-four hours and give a rise of temperature that will be mistakenly set down for tuberculosis. At the very start, therefore, it is important that the general health of the subject should be first assured by a critical professional examination. If some other disease is present the tuberculin test had best, as a rule, be delayed until that has subsided, while if tuberculosis is found the test will be superfluous.

3. *The subject must not be within three weeks of parturition, nor about to abort.* In many cases, though not in all, as preparations are made for calving, the system becomes unduly susceptible to the presence of tuberculin and that agent will cause a rise of temperature, though no tuberculosis is present. Unless this source of error is carefully guarded against the most valuable cows in the herd may be condemned unjustly.

4. *The cow must not be within three days of the period at which "heat" would naturally occur.* Under the excitement of œstrum the body temperature usually rises two or three degrees, and if tuberculin has been used this rise may be attributed to tuberculosis and a sound animal may be condemned. Nor is it always enough that the animal is supposed to be pregnant. Abortions sometimes take place unexpectedly and unknown to the owner. If, therefore, a cow under the test and which is not well advanced in pregnancy should show a rise of temperature it should be at once ascertained whether the animal is not in "heat." If symptoms of "heat" are found she should be set aside along with any calving cows to be tested again when such a source of error is no longer present.

5. *The tested animal must not be exposed to a hot sun in a closed area.* In excess this will cause heat apoplexy, and the fever heat which ushers this in, may easily be mistaken for the indications of tuberculosis.

6. *Cattle taken from pastures must not be enclosed in a hot, stuffy stable.* While they must be tied up to allow of the temperature being taken at short intervals, coolness and ventilation should be secured in summer by a sufficient air space and the requisite ventilating openings.

7. *Exposure to cold draughts between open doors and windows, or to wet or chilly blasts out of doors should be carefully guarded against.* A chill proceeding from any source and alike in the presence or absence of tuberculin causes a rise of the internal body temperature.

8. *Heavy cows unaccustomed to stand on hard boards may have a rise of temperature in connection with resulting tenderness of the feet.* One must avoid hard floors on the day of the test or make examination of feet and allow for attendant fever.

9. *Omission of the previous milking or a change of milker and consequent retention of part of the milk* will raise the temperature of a nervous cow, and in careless hands secure an erroneous condemnation.

10. *Privation of water at the regular time will often cause rise of temperature* especially when on the dry feeding of winter. I have seen a general rise of two degrees and upward from the delay of watering for a single hour, while after watering the temperature went down to the normal and remained so. Water always tends to a temporary lowering of temperature but in the presence of tuberculosis it soon rises again.

11. *Change of food is liable to produce a slight indigestion and rise of temperature.* This should be avoided as far as possible, and when a herd is taken up from pasture for the test, it should have grass, ensilage or other succulent food.

12. Cattle just from a long journey by road or rail, or other cause of violent exertion are liable to have an elevated temperature from the leucomain poisoning. Such should be left at rest until the transient fever shall have subsided.

13. Violent handling of nervous cows in taking the temperature must be carefully avoided. The operator who cannot handle them gently is not fit for this work.

14. There must be evidence that the animals have not been repeatedly tested at short intervals shortly before. In a number of instances I have found a proportion of the cattle irresponsive to

tuberculin, though a *post mortem* proved the presence of tuberculosis. Unscrupulous men, wishing to sell on a guarantee, can avail of this in animals so unaffected by the test.

15. The operator must have absolute control, even of the feeding and watering of the animals on the day of testing. Otherwise the rise of temperature may be prevented by a liberal use of antipyretics and a false guarantee may be secured.

16. An unthrifty animal, having general symptoms suggesting tuberculosis, must be subjected to the most critical examination in addition to the tuberculin which in such animals often fails to cause hyperthermia. Fortunately in such animals the tubercles are usually numerous and extensive enough to be discovered through objective symptoms.

17. Animals excluded from the test by reason of some individual unfitness at the time (parturition, œstrum, obortion or any other disease) must be marked and held for the test later after such disqualification shall have passed.

18. The operator must bear in mind the possibility of transferring other diseases from animal to animal, by contact, by the use of the same hypodermic needle on two in succession, and above all by the clinical thermometer. Diseases like contagious abortion, which present no obvious symptoms in the intervals, are especially liable to be carried in this way, and instances of the active extension of this after a test, have come under my notice. The operator should always enquire carefully as to the existence of abortions and sterility in the herd, put the aborting animals by themselves, using a special thermometer upon them, and carefully washing the hands before going to other cattle. It is well further to clean the thermometer after each animal and disinfect it with carbolic acid solution (5:100).

Dose of Tuberculin. Of the usual American preparation 2cc. (30 drops) is adapted to a cow or ox of 1,000 to 1,200 pounds. For larger and smaller animals a moderate encrease or reduction must be made, yet a considerable latitude is allowable. The new-born calf will take five drops and the animal of 700 pounds 25 drops.

Technique of the Operation. Lapses are so easily made when dealing with a large herd and are so dangerous that a regular plan should be systematically followed. The following will be found simple and convenient:

1. Inject the herd at 10 or 11 P. M. to secure a good rest and be fresh for the rise of temperature early next morning.

2. Before injecting have the subjects arranged in order and record them by name or other means of identification, with age, sex, breed, weight, pregnant or not, past or prospective date of calving, abortion, indications of disease, temperature taken just before injection and appropriate dose.

3. Inject into the loose connective tissue on the side of the neck, the animal being held by the nose, if necessary, by an assistant.

4. Use a syringe which has not been employed for any infectious products, and see that it is thoroughy cleansed and disinfected by boiling or by filling it with a carbolic acid solution (5:100).

5. After drawing the appropriate dose into the syringe, wipe the nozzle and dip it in strong carbolic acid before inserting it into the skin. This safely disinfects any virulent matter that may be lodged on the surface of the skin, and obviates those infected swellings and abscesses that have been a cause of complaint by stock owners.

6. When the nozzle is withdrawn from the skin, wipe it and dip it again into the strong carbolic acid to prevent any risk of infecting the tuberculin into which it is to be plunged.

7. The nozzle is much more easily inserted in the skin if the latter is pinched up so that the needle will transfix it at a right angle, instead of passing through a greater amount of the dense tissue because of the oblique direction. An excited animal with a thick, dense hide and a contracted panniculus muscle will offer serious obstruction which lessens greatly as the subject gets over its excitement and the muscle relaxes.

8. Temperature should be taken at 6 or 7 A. M., eight hours after injection of tuberculin, and every two hours thereafter, until the sixteenth hour.

9. If any subject shows no rise of temperature until the 16th hour after injection, its examination may be discontinued, but if it shows a slight rise toward the 16th hour it should be continued until it has shown a distinct reaction with steady rise and fall, or until, without such distinct reaction, the temperature descends to the normal.

10. If one has shown a distinct reaction but is still rising at the 16th hour, it should be continued till it begins to fall. The typical reaction is one in which the rise and fall are both gradual, and extend over a number of hours.

11. In recording the temperatures, there should be noted the exact time of each feeding, watering and milking, or any other condition (change of weather), which may in any way affect the heat production or radiation.

12. In old, emaciated animals and in second or third tests, Pearson uses twice the usual dose of tuberculin or more.

Tuberculosis Reaction. With slight variations different operators make their estimate of tuberculosis reaction on nearly the same general basis. A rise of 2° F. over the highest temperature of the day or days before, in the absence of any other appreciable cause, and provided that the elevation has followed the tuberculosis type of gradual rise and fall, is held to condemn. If, however, this rise does not exceed the normal average, if the temperature before injection did not exceed 100° and that after injection 102°, the case may well be held in doubt and reserved for re-testing. If, on the contrary, the initial temperature of the animal was 103°, and there was, between the 8th and 16th hours, a gradual typical rise and fall, reaching 104° or a little over, in the absence of any other cause for this, the subject would be condemned. Cattle having an initial temperature of 103° or above are not favorable subjects for the test, except in the case of calves in which the temperature is normally higher and the reaction must reach a higher point. In all cases of doubt it is well to hold for a second test, unless urgent sanitary considerations demand that a herd should be freed from the infection in short order. Then it may be better to risk a single error, with the concurrence of the owner, than to leave a possible centre of infection in the herd.

Local swelling in the seat of injection may be charged to lack of antiseptic care, or the presence of septic germs in the system of the animal prior to injection.

A chill during the period of reaction is not uncommon, especially in cold weather, or in a draught of cold air. The coat may stare along the spine in patches, or generally, tremors may be seen on the body or limbs, and a clammy coldness invades the

ears and horns, and especially the points of the hocks and ischia. The back is sometimes arched and the feet drawn together somewhat.

In the absence of any source of excitement the head may be less elevated, the ears lopped forward or drawn back, and even the eyelids may droop somewhat. These phenomena may last for a few minutes or for an hour or two.

In testing other genera consideration must be had of the different normal temperature (horse 99.5°, dog 98.5°, sheep or pig 103°, bird 106°), and the varying susceptibility to tuberculin, the Guinea pig requiring a maximum dose relatively to its size and man or horse a minimum.

Effect of Tuberculin Test on the Later Average Health of the Animal Reacting. The transient fever and reaction on the day after injection modifies the milk secretion temporarily to a certain extent in ratio with the hyperthermia. The consensus of veterinarians of the largest experience, and the voice of the International Veterinary Congress at Berne in 1895, oppose the doctrine of any continuous effect on the health even of the tuberculous. Yet in the case of Governor Morton's large herd of Guernseys a careful record of temperatures showed that for weeks after the test the reacting animals presented oscillations which were not shown before, and which were not found to occur in the sound animals. In the activities of sanitary work such indications are easily missed.

Effect of Tuberculin Test on Sound Animals. In 1894 I tested this on a number of thoroughbred Jersey and grade cows, injecting them six times at intervals of from five to fourteen days. It led to no appreciable change of the general health as shown by the temperature, breathing, pulse, yield of milk or its quality. Careful analysis was made of the milk at each milking, and in two animals soundness was attested by *post mortem* examination. Similar tests made by the Bureau of Animal Industry and others led to the same results. Cows in which the yield of milk was on the gain continued to encrease in the same ratio as those that had not been injected, and those in which it was on the wane showed no more rapid decrease. The butter fats and total solids showed no variation more than appeared in the healthy.

Action of Tuberculin on Parturient Cows. The testimony of

Bang, Eber and Pearson, based on a very extensive experience, would indicate that the tuberculin test is not forbidden by the parturient condition. Eber concludes that unless the initial temperature materially exceeds 39.5° C. (103° F.) the parturient state is no barrier to successful testing. My own experience, on the contrary, is that a considerable proportion of parturient cows give a reaction when the initial temperature did not exceed 103° F., and when no sign of tuberculosis could be found. As an example, a cow in high condition, with an initial temperature of 102.8° F., rose gradually from the eleventh hour after injection and reached 106.3° by the eighteenth, a rise of 3.5°. From the record she was not due to calve for three months, but a fortnight later, when already killed and laid open, she showed all the signs of parturition, a fully matured calf, and not a trace of tubercle. This is far from unusual, and I am convinced that many errors will be avoided by refusing to condemn parturient animals or those within a couple of weeks before or after parturition on the tuberculin test alone.

Reliability of the Tuberculin Test. Even in the most careful hands the tuberculin test cannot be held to be infallible. A certain very small proportion of cows react without the recognition of any tubercle post mortem, some because of other bodily conditions, like parturition or abortion, but in skilled hands these may be ignored in ordinary sanitary work. Pearson claims to have had but 8 such cases in 4400 cows that gave a typical reaction. He suspects that some of these even had undiscovered tubercle, and Nocard thinks that all such cases are to be explained in this way. On the other hand a very few really tuberculous animals fail to react, some in connection with advanced disease, some because of repeated previous testing, and some because of the introduction of antipyretic agents into the system, but such cases can either be detected and controlled or are so infinitesimal in numbers, that they can safely be ignored in sanitary work. In skilled hands, the tuberculin test will show at least $\frac{9}{10}$ ths of all cases of tuberculosis, when other methods of diagnosis will not detect $\frac{1}{10}$ th. See above case of herd where objective symptoms showed nothing, yet tuberculin condemned half the mature cattle, and post mortem confirmed this, the skeptical veterinarian being judge.

The Relation of the Bacillus Tuberculosis of Man to that of Cattle. On the discovery of the bacillus tuberculosis it was largely assumed that it was the same in all tuberculous animals, in all organs and in all circumstances. But it was soon found that the bacillus of chicken tuberculosis differed materially from that of the mammalian, that it could be inoculated only with difficulty on cattle or Guinea pig, as could that of the latter on the bird. The bacillus of the chicken found a most receptive home in the rabbit and horse, and was more easily started in artificial culture in glycerine bouillon, than was that of man or ox. But presently it appeared that the affinities and disparities did not end here. The bacillus from man or ox led to much more pronounced lesions in Guinea pigs than in rabbits, and the abdominal bacillus of the horse was inoculable on the chicken. Both horse and parrot proved receptive to the bacillus from man. Swine, like Guinea pigs showed a receptiveness to the bacillus of man or ox. The bacilli from the sputum, open tuberculous sore, or bones of man showed less virulence for Guinea pigs and rabbits than did those from tubercles in the human lungs and liver. The bacillus from the ox showed a greater virulence toward rodents and other small animals than did the bacillus from man. The bacillus of human sputum inoculated on the ox did not habitually cause generalized tuberculosis, but often a local tubercle or group of tubercles, and sometimes the inoculation wound healed without permanent lesions. These last points were seized upon to sustain a doctrine of probable duality for the microbe of tuberculosis, but if duality it was quite evident it could not end there, but must be extended to multiplicity, each small group of genera having a tubercle bacillus peculiar to itself. Those who thought their interest lay in arresting all sanitary control of tuberculous cattle and their products, became urgent in opposition to active government measures, demanding mathematical proof of the infection of man from cattle, under conditions that would exclude the remotest possibility of the introduction of infection from another source. The clearest and most abundant circumstantial evidence would not suffice, they must have direct experimental inoculation under conditions of precaution against outside germs, which were practically impossible in any community, conveniently ignoring that such inoculation, if successful, would have amounted to man-

slaughter, and that no such experimental evidence has been had, or can be had, of any of the deadly diseases of man. Infection by exposure and accidental inoculations can be had in abundance, just as they can in tuberculosis, but never under the rigid precautions which would exclude the possibility of extraneous infection.

The subject has assumed such importance that I may be excused for introducing a portion of my paper read before the New York State Medical Society in 1900.

1. *This Variability is Common to Microbes Generally.* Certain bacilli, like those of anthrax, grow in the living body as rods only, but become long filaments in given artificial media. They produce no spores in the living tissue, but do so readily in the carcass or soil. Transferred from ox to ox they are generally fatal, but if grown for several generations in Guinea-pigs, and then transferred to cattle, the resulting disease is slight (Burdon-Sanderson, Duguid, Greenfield). Rabies passed from dog to dog is almost constantly fatal, but if passed through the ape and then back to the dog it is comparatively harmless (Pasteur). In both these cases the inoculated animals become immune from the more virulent germs, showing that they have passed through the actual disease in an unusually mild form. The later system of Pasteur is founded on this same general truth, as are also the methods of lessening the pathogenesis of germs by subjecting them to compressed oxygen, to graduated heating, to an altered chemical condition of the culture medium, to antisepsis, etc. For a time such weakened cultures often retain their lessened pathogenesis, even through a succession of cultures in a susceptible animal body, acting as if the germ were indeed a distinct species. But it might well have been considered that a microbe which had changed its aptitudes in a given environment could presumably revert to its original habits under the incentive of a suitable medium. And this is precisely what does take place. Pasteur has shown that the less potent rabic virus becomes more potent when passed several times through the body of a rabbit, and that the weakened anthrax germ acquires greater force when passed through a series of small birds or newly-born mammals.

To come to tuberculosis, Trudeau tells us that a culture of bacillus tuberculosis from man inoculated on the rabbit, and then

cultivated for two years *in vitro*, becomes much less destructive to Guinea-pigs, and that after six years of such artificial culture all the Guinea-pigs inoculated with it live for many months, some for two and a half years, and some even recover. The usual life of the Guinea-pig after inoculation is seventeen days.[1] All of our zymotic diseases have in a similar way cycles of malignancy and benignancy. For a series of years measles, scarlatina, diphtheria, smallpox, or grippe have an unwonted mildness, and, again, one or another merges into a cycle of extreme and fatal malignancy. Rinderpest on the steppes of Asia is comparatively harmless to the native stock, but among outside cattle imported into the steppes or attacked in their native lands it is habitually fatal. Texas fever is mild among the indigenous cattle in the Gulf States, but very deadly to Northern stock. Glanders is not at all fatal to horses of the plains, the Rockies, or the Sierras; but it becomes redoubtable when these horses carry it to the Eastern seaboard, and still more so in Western Europe. It is a common experience to see a malady transformed through the effects of heredity or acquired immunity, through environment or the temporary mitigation of virulence in the germ; and again we see the same disease, no longer restrained by such inhibitory conditions, bursting forth as a malignant and deadly plague. We have, therefore, no warrant for the hypothesis that a pathogenic germ which, under given conditions of life, has lost in pathogenesis, but not in vitality, should continue forever to exist as a harmless microbe.

2. *Varying Malignancy of the Tubercle Bacillus in Man.* Nothing is more familiar to physicians than the slow progress of tuberculosis of the lymph-glands and bones, on the one hand, and its frequent rapid progress in pulmonary, abdominal, or encephalic organs on the other. It has on this account been rather difficult to persuade many of the etiological identity of scrofula and consumption. In experimental tuberculosis the same truth constantly crops up. Arloing and his followers found that the tubercle bacillus from the lymph-glands of man proved less virulent and deadly than that from the human lungs (*Lecons sur la Tuberculose*). As early as 1880, Creighton drew attention to this in his work on *Bovine Tuberculosis in Man*.

[1] Johns Hopkins Hospital Reports, Bulletin 100.

But the bacillus from the lungs is subject to variations of this kind. Among seven specimens of human sputum, cultivated by Theobald Smith, six had a fair average vitality, while the seventh failed to perpetuate itself on dog serum.[1]

It should be strongly emphasized in this connection that the failure of extension and generalization of the sputum germ when transferred to cattle does not distinguish it from the tubercle bacillus as conveyed from ox to ox. Everyone at all experienced with the tuberculin test well knows that in most herds the majority of the tuberculous animals show no generalization, but only a localized tuberculosis. There is reason to believe that even recoveries take place after slight infection, and it is certain that many tuberculous cattle continue for years in what appears to be good general health. Unless in particularly susceptible subjects or under specially poor hygienic conditions, or unless in case of reinfection, the average bacillus of bovine origin habitually fails to produce in other cattle a rapid extension and generalization.

3. *Interchangeability of Bacillus of Man and Bird.* Of all known forms of tubercle bacillus that of birds is the most distant from that of man or ox, and yet the beautiful experiments of Nocard[2] serve to establish their essential identity. Taking the bacillus of human sputum, which would not infect the fowl, he enclosed it in collodion capsules, which confined the bacilli while allowing transudation of the animal fluids, and left these in the abdomen of the chicken for not less than four months. He repeated this three times in succession with the product of the original sputum germ, and obtained a bacillus which was actively pathogenic for the chicken, though it had been harmless after the sojourn of four and eight months respectively.

This may explain the reported cases in which a flock of poultry have developed tuberculosis a few months after they were placed in the hands of a consumptive caretaker. That such transmission does not always occur is not surprising, considering that transmission between man and man is infrequent in comparison with the number of exposures. So in cattle the majority of exposed animals usually escape, although in such a case there can be no plausible explanation on the ground of a difference of germ. We

[1] Journal of Experimental Medicine, 1898, No. 111.

[2] Annales de l'Institut Pasteur, September, 1898

have in every case to consider the necessity for receptivity as well as infectivity, and the lack of either is a bar to infection. When, however, we assume that the most diverse tubercle bacilli are descendents of one original stock, that a large herd must furnish some animals of more than usual susceptibility, and that such animals are subjected to continuous accessions of both bacilli and toxins, we can easily understand how some of the more adaptable germs will in time accommodate themselves to the new medium. A Pettinkoffer, with an immune constitution or a specially vigorous gastric digestion, may with impunity drink a culture of cholera spirillum, but the same is not true of the drunkard fresh from a spree and with seriously impaired digestion.

Bacillus Tuberculosis in Man and Ox. Points of Similarity. The bacillus tuberculosis of cattle is in general shorter and thicker than those of man, but many in both subjects are morphologically indistinguishable. Such differences are often far exceeded by different specimens of one stock of germ seeded on different media. There is no great difference in the thermal death-point, and the viability in light, dryness, cold and putrefaction. The tendency is in all cases to colonize the lymph-plexus or glands and to develop the specific lesions, with slight variation in detail. The slow development of the lesions from both forms of bacilli and their histological similarity is another argument for their essential identity. The slow growth of both on artificial media, the demand of each for a medium having the same approximate composition, and the similar pathogenic and diagnostic characters of the toxic matters elaborated by both germs bespeak a primary identity. The very remarkable staining qualities of tubercle bacillus, from whatever source it may be drawn, are no less remarkable.

Tuberculosis of Man and Ox Coextensive. The prevalence of tuberculosis in man and ox in the same country and district is so frequent that it may be safely set down as the rule. Among ichthyophagists and great fishing communities, like the people of the Hebrides, Iceland, Newfoundland, Greenland, and the coasts of Hudson Bay, tuberculosis is rare. In these countries cattle are few or absent, or, like the hardy highland kyloes of the Hebrides, they are kept in the open air. The immunity of the people is not due to insusceptibility, since they fall ready victims to tuberculosis when removed to infected countries and cities.

In Northern Sweden, Norway, Lapland and Finland, where cattle are scarce and reindeer plentiful, tuberculosis is said to be rare, though the inhabitants live in the closest of dwellings through the long winter. In most of the Pacific islands there are no cattle, and the natives are comparatively free from consumption. In Hawaii, since the introduction of cattle, consumption has increased. Australia and Tasmania, which forty years ago were the great resorts for English consumptives, have become increasingly the homes of infection since the development of the cattle industry and the influx of phthisical subjects. Minnesota and Dakota, in the early days, were held to be incompatible with tuberculosis, but since the advent of the white man and his stabled herds they have largely lost their sanitary reputation. The highest known mortality from tuberculosis to-day is that of the reservation Indians of these States, who feed on raw, diseased beef. In the Kirghiz steppes the Tartars subsist on the flesh and milk of their solipeds, and largely escape consumption. In Japan, Dr. Ashmead tells us that the common people escape tuberculosis, while the aristocracy suffer severely. He attributes this mainly to the debauchery of the ruling class ; but it must not be overlooked that they eat freely of beef and dairy products, which the rice-eating poorer population do not. The same remarks apply in measure to the mandarin and plebeian classes in China. Holden tells us that tuberculosis is rare in Columbia, Ecuador and on the eastern slopes of the Andes, where little or no milk or butter is used. It must be further borne in mind that in these countries all herds live in the open air, and practically escape infection.

These examples must be contrasted with the consumers of beef and milk in civilized temperate countries, where the stock are largely kept in-doors. A general average mortality of 7 or 8 per cent. from tuberculosis, and the *post mortem* evidence in European and American hospitals of 33 to 50 per cent. which show tubercular lesions, recent or remote, cannot be lightly passed over. The contrast with our reservation Indians is still more striking. Holden and Treon testify that the meat furnished to the Indians is always poor and often diseased, and that when the stock arrives our hungry wards devour the internal organs raw, or, later, the flesh as pounded preserved meat, and still uncooked. The deaths

of these Indians from tuberculosis is 50 per cent. of the total mortality.[1] Dr. Washington Matthews, who spent twenty-one years among the Indians, gives their food as the main cause of the disease, and states that when the supply of fresh meat is liberal the death rate from tuberculosis is highest (Census of 1880).

If we now contrast this fearful mortality with the immunity of the Indians of Hudson Bay, Great Slave Lake, Alaska, and the North generally, we have a most suggestive picture. It may be conceded that the extreme Northern Indians, being beyond the cereal region, have a slight measure of protection in their meat diet ; but the recent spread of tuberculosis like a plague among the inhabitants of Barrow Straits, when introduced by the frozen-in whalers and the relief party, is sufficient disproof of any claim of special insusceptibility. There can be no doubt that in this, as in other virulent diseases, the rule holds that the long absence of the infection secures the preservation of the susceptible lines of blood, so that when the contagion does come it finds a more inviting field than in countries in which the more susceptible strains have been killed off and the comparatively immune have survived. Toward the Arctic circle the Indian must crowd into closer quarters in winter than his brother further south ; but, in spite of all, the beef-eating Indian is being rapidly exterminated by tuberculosis, to which his brother of the north is a comparative stranger.

Exceptions : their Explanation. This statement would be incomplete without a notice of exceptions to the rule. The Cape Town branch of the British Medical Association reports "that tuberculosis is rapidly increasing there in the human population, while tubercle in cattle is almost non-existent." This finds an abundant explanation in the different conditions of life. The men live in-doors and concentrate the infection, whereas the cattle enjoy an out-door life and escape. In a latitude of 30° south, where frost is rare, and with a dry climate (12 to 30 inches of rain per annum), the colonists find no occasion for housing their cattle, so that the conditions for the prevention of tuberculosis are ideal. It may be added that cattle are far less numerous in Cape Colony than they formerly were. The destruction first by lung plague and later by rinderpest has made the cattle industry ex-

[1]Medical Record, August 13, 1883.

tremely hazardous, and even before the advent of the rinderpest many had abandoned cattle and taken to sheep.

Parallel cases can be found in other countries. In Egypt, the great resort of consumptives, cattle are almost immune, the abattoirs furnishing about one tuberculous ox in ten thousand killed. [1] From Tunis (Alix), Algiers (Sarciron, Plaise) and Senegambia (Lenoir) a similar testimony comes. Cattle imported from Europe may die of tuberculosis, which is liable to assume a rapidly fatal type; but the native cattle, kept in the open air, are practically exempt.

Jersey cattle in their native island, staked out at pasture all the year round, show little or no tuberculosis, whereas the housed Jerseys of England and America suffer severely. The cattle of our Gulf Coast States, kept on ranches in the open air, are largely immune, and the cattle of Columbia, Ecuador, Peru, and the Argentine Republic largely escape; but the housed dairy cows of our Southern cities show a very high ratio of consumptives. Consumption becomes more and more deadly in the Southern negro even in the country localities, while the out-door cattle of the same districts escape.

The absence of tuberculosis from the sanitarium herd at Saranac requires to be explained on a different basis. This herd is housed in winter, and infection, once introduced, would have opportunity to spread. The absence of tuberculosis is highly complimentary to the management of the establishment. But a similar immunity is the rule for all well-managed sanitariums, and not as regards cattle only, but man as well. At Argeles no case of tuberculosis contagion to attendants occured in ten years (Ferrand). At Soden baths, in a village of 1500, there were in thirty-four years 65 deaths, 15 from consumption (Hopt). At Falkenstein, in fifteen years, one attendant became tuberculous (Jousset). At Görbersdorf the cases of consumption in the village and environs decreased (Knopf). At Brompton, London, in thirty-six years, among 150 attendants, but one became consumptive, though they individually served for from fifteen to twenty-four years, and nearly 40,000 patients had been received. [2] A well-conducted

[1] Danzon. Études Expérimentale et Clinques sur la Tuberculose, Vol. i. p. 350.

[2] Etudes Expérimentale et Clinques sur la Tuberculose, Vol. iii. p. 408.

sanitarium is and should be a safer place than the average community, in which 15 per cent. and upward are tuberculous. The educational influence of such an institution should decrease tuberculosis in the surrounding districts.

Cases of Direct Infection from Man to Ox. Chauveau induced tuberculosis in cattle by feeding the tubercle from the lungs of man.[1]

Nocard relates that a Beauce farmer, with a finely appointed stable and healthy herd, in 1883 employed a dairyman who had cough, profuse expectoration, and occasional hæmoptysis, and who had been several times in the hospital in consequence. He slept in the cow stable directly over the cows. In 1886 two cows, stalled immediately beneath him, showed ill health and were put up to fatten, but did badly and showed extensive tuberculosis when butchered. The dairyman stayed until 1891, having to go to the hospital several times in the interval. In 1892 the tuberculin test was applied and seven more cows were found to be tuberculous.

Huon tells of a cow bought to furnish milk for calves used to raise vaccine. She stood the tuberculin test, and was carefully secluded from all other cattle, but soon began to fall off, and in six months was very much emaciated, responded to the tuberculin test, and when killed showed extensive tuberculosis. Her caretaker at the vaccine establishment had what was believed to be chronic bronchitis, but when he died, soon after, this was found to be extensive pulmonary tuberculosis.

Bollinger inoculated a three-months' calf with liquid from human tubercle and killed it seven months later. Fibroid pedunculated tumors, from a pea to a walnut in size, hung from the mesentery and spleen, and the mesenteric and retroperitoneal glands were tubercular.[2]

Sidney Martin furnishes the following: Four calves were fed 70 c.c. of sputum containing a large number of bacilli. Three were killed after four, eight, and twelve months respectively, and had severally 53, 63 and 13 nodules on the small intestine, mostly on Peyer's patches. Two calves received at one dose 440 c.c. of tuberculous sputum, and were severally killed after eight

[1] Arloing. Tuberculosis Congress of 1891.
[2] Münchener medicinische Wochenschrift, 1894.

and nineteen weeks. The first had tubercular nodules in the intestine and mesenteric glands.[1]

Frothingham injected into the peritoneum of two calves, three and thirteen weeks old, a culture of tubercle bacilli isolated one year before from the liver of a child. Slight local nodules were produced, some like spontaneous tubercle, others granulation tissue.

Theobald Smith inoculated sputum into the chest and abdomen of the following:

1. A yearling heifer, which was killed two months later and showed on the pleura near the seat of infection a mass of tubercles, one by one and a half inch in diameter, with partly caseated centres; also a nodule one-eighth of an inch on the right lung, and small tubercles attached to the diaphragm and omentum.

2. A yearling injected in the same way showed in two months on the diaphragm a mass of tubercles two inches in diameter, and a second mass one inch in diameter on the ribs near the seat of infection. Microscopical examination failed to detect bacilli, but there is no evidence that they were sought by culture or inoculation.

3. A cow injected in the chest and killed after two months showed tubercles of the lungs, pleura, and mediastinal glands, partly caseated and containing bacilli. Vascular fringes hung from the pleura.

4. A cow receiving a chest injection of sputum culture and killed in two months showed fringes and pendulous masses on the pleura, with small tubercles containing cheesy matter and a few bacilli.[2]

Crookshank injected tubercular sputum into the peritoneum of a calf, which died of streptococcus infection on the forty-second day. It showed extensive tubercular deposits in the seat of injection and an abscess the size of a walnut. Nodular fleshy neoplasms in hundreds studded the mesentery, omentum, liver, spleen, and diaphragm, and small tubercles disseminated through the lungs and liver contained tubercle bacilli. These abscesses contained streptococci.[3]

[1] Report of Royal Commission of 1895.
[2] Journal of Experimental Medicine, 1898, vol. iii. p. 482.
[3] Transactions of the Pathological Society of London, 1891, p. 332.

The experimental inoculations of cattle with sputum by T. Smith, Kruse, and Adami showed a decided lack of potency in the bovine system, but (1) they do not show that the germ at once perishes in the system of cattle; (2) they do not prove that this germ, if returned from the ox to man, would prove less pathogenic than if carried from man to man without the intervention of the ox. (3) The observations of Bollinger, Baumgarten, and Crookshank show that under certain conditions the sputum bacillus can and does produce generalized tuberculosis in cattle. (4) Diminished pathogenesis of the germ when passed from man to cattle is no guarantee that this germ, or the slightly modified germ of casual bovine tuberculosis will prove equally mild if transferred from the bovine to the human patient.

Cases of Infection of Man from Ox. Tscherning, of Copenhagen, attended a young veterinarian who had cut his finger in dissecting a tuberculous cow. The skin wound healed in three weeks, but a subcutaneous swelling persisted, an ulcer formed, and a tuberculous mass containing bacilli was removed. No secondary tubercles formed.[1] A parallel case occurred to a prominent American veterinarian. The diseased tissue was excised and the bacilli identified by the bacteriologist of the university with which the patient was connected, and a permanent recovery ensued.

Pfeiffer, of Weimar, attended a veterinarian who had been similarly inoculated from a tuberculous cow. The patient, aged thirty-four years, had a good constitution and no tuberculous taint. The cutaneous lesion healed, but six months later there was tuberculosis in the cicatrix; pulmonary tuberculosis followed, and the patient died of this two years later. At the necropsy were found tubercular arthritis of the wounded thumb and many vomicæ in the lungs.[2]

The post-mortem wart (tuberculosis verrucosa cutis) is familiar to surgeons as occurring in butchers and tanners, and there is every presumption that in many cases this is of bovine origin (Martin du Magny, Hanot, Senn, Riehl, Paltauf, Osler). Gerber testifies that in exceptional cases this extends to the lymph-glands and becomes generalized.

[1] **Nocard. Dictionnaire de Med. Veterinaire. Article, Tuberculosis.**
[2] **Zeitschrift für Hygiene, Band iii.**

Dr. Stang, of Amorbach, had a five-year-old, finely developed boy patient, of healthy parents, destitute of hereditary taint. He died after a few weeks' illness with miliary tuberculosis of the lungs and enormously enlarged tubercular mesenteric glands. The cow which supplied his milk had been killed a short time before with pulmonary tuberculosis.[1]

Dr. Demme, of the Children's Hospital, Berne, had four infants, the offspring of sound parents, with no hereditary taint of tubercle, die of intestinal and mesenteric tuberculosis, having been fed on the milk of tuberculous cows. Among 2,000 tuberculous infants treated in twenty years these were the only ones in which he could exclude the probability of hereditary and other causes.[2]

Mr. Howe, of North Hadley, Mass., lost a son, aged twenty months, from abdominal tuberculosis, three months after he had paid a week's visit to his uncle and had been fed the milk of the uncle's tuberculous cow. The cow showed at death generalized tuberculosis. The child had been strong and well, as were his parents.

The four-year-old son of Colonel Beecher, of Yonkers, died March, 1894, of tubercular meningitis, and the two Alderney cows which had supplied him with milk were then proved consumptive by the tuberculin test and post-mortem examination.[3]

The child of Dr. Brown, U. S. A., was similarly cut off by tuberculosis, having been fed on the milk of a tuberculous cow.

Dr. C. H. Peabody had a child patient die of tubercular meningitis three months after the family cow had been killed for generalized tuberculosis. There had been previously no tuberculosis in the family (Ernst, *Infectiousness of Milk*).

A. H. Rose, of Littleton, Mass., gives the case of a child which was fed for three years on the milk of a tuberculous cow and died with abdominal tuberculosis (Ernst).

Gordon, of Quincy, Mass., records the case of a ten months-old child of healthy parents and ancestry which had been fed on the milk of a cow with advanced tuberculosis, and which died after a few weeks with acute tuberculosis (Ernst).

[1] Lydtin. Veterinary Congress, Brussels, 1883.
[2] Nocard. Dictionaire de Med. Veterinaire. Article, Tuberculosis.
[3] New York Sun, March 29, 1894.

Gage, of Lowell, Mass., had an infant patient of healthy parents and surroundings, but which subsisted exclusively on a cow's milk that contained bacilli and infected Guinea pigs. The child died of tubercular meningitis. A second child fed the same milk suffered in a similar way (Ernst).

Andersen, of Seeland, reports the death from tuberculosis of a six-months-old child which had fed on the milk of a cow having tuberculosis of the udder. The mother developed symptoms of tuberculosis after the death of the child.[1]

Dr. Gosse, of Geneva, Switzerland, spent his Sundays with his family on an estate in the hills, and his daughter, aged seventeen years, took great pleasure in drinking milk warm from the cows. Early in 1893 she sickened with an obscure illness, and after ten months died, revealing at the necropsy intestinal and mesenteric tuberculosis. The five cows on the estate were tested with tuberculin; four reacted and were killed; two showed tuberculous udders (Nocard).

Dr. H. M. Pond reports four cases of tuberculosis in one family, three of them fatal. The cows supplying the family with milk were tuberculous.[2]

Dr. Faust, veterinarian, of Poughkeepsie, records the case of a family on Long Island that lost from tuberculosis 139 cows. A three-year-old child and two grown sons died of tuberculosis. Tuberculosis was unknown in the parents' families.[3]

Dr. Kelly, veterinarian, Albany, gives the following: In a family of five a son, aged nineteen years, was very fond of milk and drank it fresh from the cow, and contracted tuberculosis. Some months later the farm herd of seventeen registered Jerseys were tested with tuberculin, and thirteen reacted and showed extensive tuberculosis when killed.

Dr. Cooper, veterinarian, Paterson, N. J., furnishes this: A child, fed on the milk of a cow, contracted tabes mesenterica. Examination revealed the presence of tubercle bacilli in the milk. The milk was then fed to ten kittens, all of which became ill and emaciated, and when killed showed tuberculosis.

Such cases, in connection with the experimental inoculations,

[1] Hatch Experiment Station, Massachusetts Agricultural College, Bulletin No. 3.

[2] Pacific Medical and Surgical Journal, 1888.

[3] Report to the Board of Health.

furnish more than mere circumstantial evidence. They are corroborated and strengthened by the very uniform diffusion of tuberculosis in man and stalled cattle in practically all civilized countries. Of the closer connection in individual cases one or two instances may here be added as drawn from personal observation:

1. In one case a family cow and the owner's wife had both advanced tuberculosis. The lady consumed a good deal of the cow's milk, but when she gave up its use she felt decidedly better.

2. The owner of a thoroughbred herd of sixty head had suffered for years from consumption, and attributed the poor condition of the animals to lack of care since he had been laid aside. Without the tuberculin test, I diagnosed tuberculosis in fifty-nine of the animals.

3. In a second thorougbred herd there never lacked one or two cases of advanced tuberculosis, two of the family suffered, and the eldest son, who was fond of milk and vegetable food, died of pulmonary tuberculosis.

4. A dairy of common cows had seventeen out of twenty-six destroyed for tuberculosis, and the farmer's wife, father-in-law, and two brothers-in-law had shortly before died of consumption. The wife felt ill in the close house air, and with her father occupied herself much about the cattle.

5. A veterinary professor, who was meat inspector of the city abattoir, died a few years ago of tuberculosis, which he handled so constantly in his daily duties.

6. While printing this second edition, I have before me a veterinarian, a strong, splendid vigorous man who has contracted tuberculosis in the lungs, just after making necropsies of tuberculous cows.

Stalker and Niles report that 5 persons, 20 to 30 years of age, of healthy ancestry, died of tuberculosis within two years, on a farm where 17 tuberculous cattle were found, and others had died in previous years, (Bull. Ia. Agr. Exp. Stat., 29).

Leonhardt reports the death from tuberculosis, abdominal and meningeal, of two children, fed on the milk of a tuberculous cow, (Rep. N. H. Bd. of Health, 1892).

Sontag reports the death by tuberculosis of a six months old

child of healthy parents, that had been fed the milk of a tuberculous cow, (Rep. N. H. Board of Health, 1892).

Dr. L. Pearson quotes the following :

" A well known veterinarian, wounded in the hand in opening a tuberculous cow, had a tumid, intractable sore, the tissues of which when excised showed tubercle bacilli.

A veterinarian, of Chester Co., Pa., in opening a tuberculous cow, cut his knuckle, which healed tardily, remained swollen and when excised, showed typical tubercular lesions including giant cells. "

He quotes from Hartzell the cases of two men wounded in cleaning cattle cars, both of whom had resulting tubercle, arrested in one case by excision, but in the other advanced to generalized tuberculosis and death.

Bang gives the following Danish cases :

A merchant having two chlorotic daughters secured a fine cow to feed them fresh milk. The cow was killed tuberculous and was replaced by another which also showed tuberculosis, this time affecting the udder. The girls died of tuberculosis at 16 and 18 years. Two younger children fed on the milk of sound cows grew up healthy.

A healthy cow became tubercular, after having been placed in the same stall in which another had died of tuberculosis. A child fed almost exclusively on the milk of these two cows died of tuberculosis.

A peasant at Silkeborg drank freely of milk freshly drawn. He died of tuberculosis, as did also a cow, and later in the same stable, a pig.

A peasant had an 11 year old cow with generalized tuberculosis, implicating the udder. The wife of the peasant, formerly healthy, became tuberculous shortly after the udder became affected and died at 45. A daughter who, like her mother, used the milk of this cow, died consumptive in the same year. The husband who drank beer, and not milk, remained well.

A physician fed his two children on the milk of his tubercular cow, and lost both from tuberculosis. Neither parent nor grand-parents were tuberculous. (L. Pearson in Bull. 75, Tuberculosis of Cattle).

Thorne had reports from twenty-two Ohio physicians to the effect that they had traced tuberculosis in their patients to the use of the milk of tuberculous cows, and thirty-three who be-

lieved they had reason to suspect the meat and milk supply as the source of cases of consumption. (Ohio Exp. Stat. Bull. 108).

To these may be added the cutaneous forms of tuberculosis (tuberculosis verrucosa cutis), which occur on the hands of persons (butchers, tanners, coachmen, cooks, etc.), who handle infected products of animals. These have been described by Riehl and Paltauf, Senn, and a number of surgeons and dermatologists, and the relation of the occupation to the seat of the disease is conclusive as to the source of the infection. It is the exact counterpart of the verrucosa necrogenica of the hands of persons working in the dissecting rooms of medical schools, and the source of infection is equally well established in both cases.

A strong argument for the appreciable influence of the bovine bacillus upon man (acting directly or indirectly through the pig) is that the relative death rate of Jews from tuberculosis is materially less than that of other races. It is constantly claimed that orthodox Jews who eat only kosher (rabbi inspected) beef and no pork, suffer least of all the population from tuberculosis. Dr. Gerster, judging by the burial returns of the United Synagogue and the English Registrar General's returns, concludes that only about half the relative proportion of Hebrews suffer from consumption as do other races in the same country. Some remarkable facts come out in the report of the Royal Commission on tuberculosis in England. In England and Wales the disease had decreased 39.1 per cent. in thirty-five years, but this decrease has been mainly in pulmonary cases, while the abdominal forms decreased only 8.5 per cent. Sir Richard Thorne, indeed, says that in children of the first year there had been an actual encrease of 27.7 per cent. Northrup and Still, on the contrary, present statistics showing that in children the pulmonary form of tuberculosis is the most common (Brit. Med. Jour. 1898). If in the face of this there has been a very material encrease of the abdominal form, coincident with the notorious encrease of the disease in dairy cows and of the bottle-feeding of infants, may we not enquire how much of this is due to the greater prevalence of bottle-feeding and the infected cow's milk? It is not for a moment supposed that the majority of infections in children come from the cow. The question is whether this encrease in the minority is not in measure chargeable on the cow. The impaired nutrition

resulting in some instances from the use of cow's milk cannot well be charged with a marked encrease of cases in which the mesenteric tubercles point so directly to infection through the food. If it is held that the tubercle bacilli in the milk are harmless, we wait for evidence of the real cause of such encrease and localization.

The experiments of Adami show that tubercle is directly transmissible from man to ox though usually with decreased virulence. More recently, Ravenel using the bacillus derived from the mesenteric glands of a child, injected intravenously two tuberculin-tested calves, with 5 cc. each of the culture, producing exalted hyperthermia, miliary tuberculosis of the lungs, tuberculosis and caseation of the bronchial and mediastinal glands, and death in 17 days.

As showing accommodation to environment, Bataillon and Ferre found that the bacillus (mammalian and avian) grown in frogs, Dubard that grown in fishes, Kráhl that grown in frogs, snakes, fishes and lizards, and Müeller that grown in the glow worm, thereafter grew at summer temperature (68°. F.) and grew poorly or not at all in the bodies of mammalia.

The tubercle bacillus is primarily and essentially one, but this must not close our eyes to the fact that in different hosts and environments it takes on very different habits, so that for the time and in these surroundings, it is materially modified in its pathogenic attitude toward different races. Yet its ready variability when conditions are favorable to change, renders it desirable to destroy it in all its forms, and especially in those which approximate most closely to those that prey on man and animals.

An impartial review of the whole field warrants the conclusion that the nineteen young (and therefore comparatively unsusceptible) cattle which in Koch's hands failed to develop generalized tuberculosis after inoculation with the virulent sputum of man, and the smaller numbers that resulted in the same way under similar treatment in the hands of Th. Smith, Dinwiddie and Adami, while showing a very marked limitation in the susceptibility of the sound bovine system to weak bacilli from man, cannot disprove the many well authenticated cases of the transmission of tuberculosis from cow to man and the reverse. The greater potency of the bovine bacillus over that of man, in its

action on the small rodents and pigs, utterly forbids the unproved assumption that it is on the contrary harmless to man.

If the object of the sanitarian were merely to delay a fatal result in his tuberculous patient, while he accepted the prevalence of tuberculosis as inevitable for all future time, the acknowledged lessened receptivity of the ox for the bacillus from man would mean more and would be at least worthy of a hearing, but as the extinction of a disease germ and its representative plague must ever be the first object, any movement toward the preservation in cattle of a germ which is deadly to man and much more so to cattle, must be held as subversive of the prime purpose of sanitary work. This is true even if we allow, for the sake of argument, that only a few of the bovine bacilli are capable of dangerously colonizing the human body, and that special environment is needful to allow of such successful colonization. On the other hand the limited receptivity of the ox for the bacillus from man is the greatest encouragement to active work to exterminate tuberculosis from our herds. It is impossible to adopt in man the summary measures that are so successful in the speedy *stamping out* of the plagues of the lower animals, so that tuberculosis in the human family can only be eradicated by slow degrees, and therefore will long continue for our herds the danger from the human side, but just so far as the susceptibility of cattle to human tuberculosis is limited, in the same ratio are our hands strengthened in effective work for the extinction of consumption in our herds and for preserving their soundness after they have once been purified. If they were to be reinfected by the presence of any consumptive person we might well despair of success in face of a wide prevalence of tuberculosis in man, but since it is only exceptionally that cattle suffer from man, outbreaks coming from this source can the more easily be taken care of. In this view tuberculosis is approximated somewhat more closely to the other bovine plagues (lung plague, rinderpest) which can be stamped out with the greatest ease and certainty, so that as a purely economic measure the argument for the speedy extinction of tuberculosis in our herds is reënforced.

TREATMENT OF TUBERCULOSIS.

Like all deady infectious diseases in the lower animals, tuberculosis is not to be profitably treated as a rule. In the case of

specially valuable breeding animals, in which the prospective progeny will pay for large outlay, and when the disease is in its incipient stage, treatment may be warranted. The patient should be thoroughly separated from other animals, kept in the open air, or, in our northern winters, in roomy, well ventilated buildings facing the south, well lighted, kept immaculately clean, frequently whitewashed, and well drained. If there are more than one case every precaution named under the head of prevention must be adopted. Exercise to keep the muscular system in good tone is called for, but never to fatigue. Hence, a sheltered pasturage is ideal. Feeding must be liberal including a ration of grain or seeds, and oil bearing seeds like linseed, rape seed or cotton seed may be specially named. Cod liver oil alone, or etherized, is often of great value, with iron and bitters continued for weeks or months. Pancreatic extract subcutem delays the growth of bacilli, and in dilute solution cured one local tubercle ; with encreased phagocytosis. Concentrated preparations aggravate (Italia). In the case of specially valuable animals one may use a pneumatic cabinet the principle of which is to diminish the air pressure on the body at large by an exhaust, while pure air for breathing, at the ordinary atmospheric pressure, is introduced through a tube furnished with a face piece fitting around closely beneath the eyes. This serves the purpose of attracting (sucking) the blood toward the skin and other tissues from the lungs, which in their turn are compressed by the air at the atmospheric pressure. Pulmonary congestion is in this way lessened, exudates are absorbed, necrotic tissue removed, sepsis counteracted, hæmatosis encreased, circulation of both blood and lymph stimulated, digestion and nutrition improved, and general health invigorated.

Active grooming and even the stimulating effect of cold douches may be invoked, the skin being rubbed actively until dry and warm.

If the circulation is poor, a stimulating steam or hot water bath of fifteen minutes, followed by a cold sponge and rubbing till dry may be profitably substituted. In such cases it is well to put a cold sponge on the head while in the bath.

Medicated inhalation is often valuable especially when the lesions are on, or near the bronchial mucosa. In 1868, Dewar met with most successful results from inhalation of sulphurous acid gas impregnating the atmosphere as strongly as the patient can

breathe without discomfort. His own groom who had given up work because of advanced phthisis, under treatment of half an hour three times a day, became ruddy, gained weight, and betook himself to work again. In rabbits which I inoculated with human sputa, the same year, all died tuberculous excepting one which I fumigated three times a day for weeks. This rabbit remained plump and well.

As an example of a still more irritating inhalent, I watched the case of a phthisical man who secured employment on the government disinfecting corps, in Chicago, in purifying the lung-plague-infected stables with chloride of lime solution, and who very soon began to improve, gaining weight and strength, his cough meanwhile subsiding.

Cervello's use of formalin by inhalation though well spoken of by its inventor would seem too irritating on the delicate lungs, however good it is as an antiseptic.

Inhalents may be conjoined with the pneumatic cabinet.

Among agents used to moderate the cough may be named codeine, morphia, cherry laurel water, wild cherry bark, guaiacol, menthol, syrup of Tolu, or chloroform, or alcohol inhalation.

As an internal antiseptic, carbonate of creosote has often proved beneficial.

Mustard blisters on the skin covering the tubercle is claimed by Knopf to act beneficially by attracting the microbes from the delicate lungs to the more robust skin and connective tissue, where they can be better disposed of by the more abundant leucocytes.

He has also found excellent results in complex infections in animals from the use of Marmorek's streptococci serum in doses of 10 cc., followed after the second dose by 5 cc. every 24 hours. In other cases it failed of the effect (reduction to normal temperature) evidently indicating that the hyperthermia was maintained by other microbes than the streptococcus. The principle is good, and perhaps at some time in the future a bacteriological examination of the sputum may reveal the microbes present and suggest the sera for such complications.

The introduction of air into the peritoneum has long been known to exercise a retarding and curative action on abdominal tuberculosis. My colleague, V. A. Moore, has lately experimented by pumping air and oxygen into the peritoneum and pleuræ in cattle slightly affected, with encouraging results.

I should add that isolated superficial tubercles may often be excised to advantage, and the part dressed antiseptically.

It is only in exceptional cases, however, that one is warranted in preserving and running the danger of spreading the tubercle bacillus for the advantage that can be secured to individual animals from treatment.

EXTINCTION OF TUBERCULOSIS IN CATTLE.

As cattle are the great propagators of tuberculosis on the farm, the question of extinction necessarily centers around this race. As in all other dangerous infections, the most prompt and successful method would be in the time honored one of destroying the infected and thoroughly disinfecting all their products. The only barrier to success in such a case would be the conveyance of infection anew from man to cattle after the herd had been purified. The demonstration that cattle are less susceptible to infection by the human sputa than many had supposed does away largely with this objection, as in a generally purified bovine race, the few primary cases contracted from man could be easily taken care of. Then, if tuberculous persons were interdicted from attendance on cattle, the danger in this direction would become very nearly a negligible quantity.

There remains the question of expense and many honestly consider this as absolutely prohibitory. The estimate of 5 per cent. of our 68,000,000 head to be killed and paid for at $25 per head, would be $85,000,000. But there is no necessity for this. Our fat steers at the packing houses are tuberculous only to the extent of 0.02 per cent., speaking well for their dams and nurses. The estimate of 5 per cent. based upon the testing of those few herds that have been taken because they were already known to be tuberculous, is unquestionably far above the actual ratio for the United States. It may apply to dairy cattle in some infected districts, but, for the bovive race of the whole country, it is absurdly high. If we had 10, 20 or 50 per cent, infected, as in some countries of Europe, the objection of expense might be a formidable one, but when the ratio of the infected is but 2 : 10000, we have every encouragement on the score of expense to enter on a campaign of extinction. But again, we do not need to deal with 68,000,000 cattle as we can omit the steers which are so

little affected and which will all come to the slaughter house in two or three years. The source of their infection (a few cases excepted) is in the older cows and bulls of the dairy and breeding herds, and this brings down our total to a little over 17,000,000. The average census price for dairy cows is $29 and, as the condemned cows are depreciated by their condition, it would be a high average to estimate them at $20. Again, the average infected ratio of cows for the entire country would be set high at 2 per cent., and on this basis it might well be that the required indemnity would not aggregate much over $6,000,000. Five or even ten times that amount would be a mere trifle in comparison with the $3,000,000,000 value, constantly encreasing, of our domestic animals, with the $99,210,272, representing our yearly product of beef and beef products, with our annual dairy products, worth $500,000,000, (Alford), or with our yearly loss of 100,000 of our population in the very prime of life when they are of the greatest value to the country, representing a yearly drain of $100,000,000, beside all the suffering and loss entailed by their prolonged and too often helpless idleness. Though this last item is doomed to continue for a length of time after the disease has been extinguished in our herds, it is receiving constant accessions from the latter, and can never be entirely done away with until our cattle are above suspicion.

An even more serious problem is the demand for tuberculin and above all for accomplished, experienced and honorable veterinarians fitted to conduct the sanitary campaign over the entire country. The tuberculin cannot be produced in a week or a month, yet the problem of its production in any required amount in a few months is merely one of the encrease of existing plants under the management of the same careful hands now engaged upon it. As to veterinarians it would be impossible to secure at once the required staff of men capable of carrying out the work over the whole country. But this is not essential. The work can be begun in the counties supplying the large cities with milk, and in the great butter and cheese producing areas where it is so urgently needed, and it must be made to include all thoroughbred herds, which are so constantly drawn upon to improve the blood elsewhere, and each herd, county and district, as freed from infection must be scheduled and no additions made to it from outside,

except under the guarantee of the tuberculin test, repeated in six months. The reacting animals, must be appraised, excluded from the herd, and disposed of, it may be to the butcher to be killed under official expert inspection, and the salvage, if any, to be deducted from the appraised value ; or to be rendered and the salvage estimated ; or to be buried as the case may be. In all such cases the other animals (horses, pigs) that occupy the same buildings and yards should be tested, although the risk of the infection of cattle from these animals is comparatively small. Unless in badly infected herds, steers and young cattle, which can be kept in a separate herd need not be tested. Vermin must be killed. Thorough disinfection must be applied to buildings and yards, and the dairy herd must be retested at the end of every six months until no more reactions are met with.

In this way the campaign in any State can be begun with a small staff, which may be steadily encreased as men are trained to the work, and in no great length of time the dairying and breeding herds can be purified and the investigation carried into the more purely agricultural fields, where herds are small and usually free from infection. Many minor points would require the attention of a competent superintendent of the work. My object here is to make a plea for the approved and attested method which has never failed in the case of other animal infection on enclosed farms, and which is based on the absolute destruction of every seed of the disease in the area under sanitary control. The method has the apparent drawback, that it demands a greater relative outlay at the start, than do others proposed, but in view of its certainty, and the confident hope of an early abolition of all infection, loss, and expensive expert control, it must, in my opinion, be looked upon here, as it has always proved in the past, the course of the truest economy. It may be compared to the treatment of a field of thistles by removing the offensive weeds, root and branch, before they have advanced to seed, instead of merely cutting them down with a mower, and leaving the roots, to grow anew, to leaf, to blossom and seed, in spite of the temporary partial drawback. But as the prospect of early legislation along this line is not a bright one, the expert must accommodate his aims and efforts to what can be done under the existing laws.

Breeding Healthy Stock from Parents with Latent Tuberculosis. When a State is not pledged to exterminate tuberculosis by prompt and radical measures, it is quite possible to raise healthy stock from sires and dams that have the disease in a latent form. It is very exceptional that calves are born tuberculous. If, then, they are kept in a pure environment and furnished with the milk of sound nurses, or even with the milk of their own tuberculous dams, after it has been heated for one-quarter of an hour to 180° F. or 212° F. they can be preserved in perfect health.

This is especially adapted to herds of valuable thoroughbreds, the destruction of which would be a serious loss, and the preservation of the strain of blood a most desirable object.

The whole herd should be tested with tuberculin, and the advanced and generalized cases, that can be detected by objective symptoms, should be at once destroyed and safely disposed of. The animals in good condition and that have not reacted should be placed in a new barn and yard, or where no tubercle has been, or in places that have been thoroughly disinfected, under special attendants. There will remain, the animals in good condition with no objective symptom, but that have reacted, and these are placed in separate barn, yard and pasture well away from other stock, under their own attendants, for breeding purposes. They should have the best of food and air, clean, well lighted, roomy buildings with shelter from storms, clean sheltered yards, and in summer, pasturage. Any cow showing indication of active advance or generalization of the disease (cough, wheezing, dyspnœa under exertion, excessive pallor of mucosæ, unthriftiness), should be at once separated and destroyed as endangering the reinfection of others, and the stable subjected to disinfection. The calf, as soon as born, must be removed to a special building or park, where it shall get milk from a sound cow or that of its own dam after it has been carefully sterilized. After sterilization the milk cannot safely be returned to the unscalded pail into which it was drawn from the cow, and it should be fed by separate attendants who have not milked nor handled the affected animal. Any loss of condition, unthriftiness, cough or scouring on the part of any of the calves should be the signal for its separation from its fellows, subjection to the tuberculin test, and, if it reacts, for its destruction and the disinfection of the building

where it was. It is well to test each calf at six weeks old and to remove the reacting ones. The success of this method is now well established.

Goodman, of Dorpat, applied it largely as early as 1891, rearing the healthy calves of reacting cows on the milk of cows that had stood the test. Bang, of Denmark, raised such calves to sound maturity on sterilized milk. Reynolds, of Minnesota, reports the raising of twenty-four healthy calves from infected cows on the milk of tested cows, while three fed on milk of reacting cows, which was supposed to be sterilized, all became tuberculous. McEachran (1899) in an extended experiment succeeded perfectly with the milk of tested cows only. I have now in hand a Jersey herd in which the progeny, fed on the milk of their reacting dams, became tuberculous without exception, and in the years following, those fed on the milk of the same reacting cows after it had been kept at 180° F. for half an hour all grew up healthy.

Under this method, inasmuch as the infection is not at once extinguished, but temporized with for the benefit of the stock owner, State indemnities are not necessarily called for. Yet the State can profitably test the cattle at public expense, mark indelibly those that react, schedule them and control them, so that they will not be allowed to change hands nor to mingle with sound animals until finally butchered, dead or recovered. The State should see to the thoroughness of the seclusion, disinfection, the safe disposal of all products from milk to manure, and the testing at intervals of three or six months of both cows and calves.

Raising Healthy Offspring Without Sterilizing the Milk. In the northwest territories, cows and heifers that have reacted to tuberculin, but which otherwise appear to be in good health, are made into a herd by themselves and placed on a special range apart from all other cattle. They live in the open air, slight shelter being allowed in winter only, and their calves are allowed to suck the dams until winter. The wide range, the open air life, and the early destruction by oxygen and sunshine of the discharged bacilli, tend in the main to ward off infection, except such as comes in the milk, and the majority of the calves grow up in apparent health and are fattened for market. A small minority are born tuberculous or contract the infection from the milk, but

this does not seriously impair the financial success, and living in the open air they bring little danger to others. The loss is infinitesimal as compared with the expense of milking a large herd, sterilizing the milk and feeding it by hand. Advanced cases, with objective symptoms, should always be removed, and the cows may be tested at intervals if compatible with profit. The seclusion of the herd should be complete, by distance, by the configuration of the country, or by fence.

For this system the climate of our Southern States, where stock can remain out of doors all the year, offers a better field than the semi-arctic northwest.

Removal of all Unthrifty Animals and Those Showing Physical Symptoms of Tuberculosis. Before the days of tuberculin testing I succeeded in extinguishing tuberculosis in several herds by the prompt removal of all unthrifty animals and such as showed objective symptoms of tuberculosis, the disinfection of the buildings, the restocking from sound herds, and the strict separation of the new stock from the old. In one herd of 200 this entailed the final destruction of the whole original herd; in others the destruction was in the main, limited to particular (susceptible) families. But in these days, with the tuberculin test available, a resort to a method of this kind would produce an unnecessarily slow, uncertain and expensive result.

Removal of Animals Showing Objective Symptoms or Reaction under Tuberculin. The Bang (Danish) method is the chief example of this, and is so considerate of both state outlay and stock owner's interests that it is deserving of high praise. Under it the State usually waits for the stock owner to take the initiative, but to encourage applications from the owners, it furnishes tuberculin testing without expense, and even allows a small indemnity for animals killed because of advanced tuberculosis. In return for this the stock owner agrees to furnish separate buildings (or enclosures) yards and pasturages, new or, when necessary, disinfected, one set for the high conditioned, nonreacting, healthy herd, and a second for the animals that reacted but which show no further sign of tuberculosis, with separate attendants, utensils and other appointments for each. This reacting herd is furnished with the best of food, air, accommodation and hygiene generally, and the milk is sterilized before it is allowed to pass into consump-

tion by man or for calves or pigs or for the production of butter. The quarantined herd is marked, registered, and kept under government surveillance ; it cannot be parted with for stock uses, but it is at the disposal of the owner to keep it for milk, or fatten at once for the butcher. Finally every member of this herd is slaughtered under government inspection, and the beef put on the market or sent to the rendering works as may be decided. The system secures the hearty coöperation of dairyman, dealer and government, and while it comes short of the speed and efficiency of a generally applied method of extinction, it is accomplishing a great work for Denmark, putting an immediate stop to the advance of the disease in the worst infected herds, and placing the latent cases of such herds in a safe seclusion for the rest of their lives. At first the tested herds showed 40 per cent. affected ; now less than 20 per cent.

The feature which would be likely to work the least satisfactorily in the United States is the disposal of the sterilized milk as such. It is to be feared that this milk would find a poor market with us, and if it proved unsalable, the preservation of the reacting herd would be no longer an economic success.

In Pennsylvania where practically the same method is in force, leaving it in the option of the owner to keep the reacting latent cases and sterilize their milk, or to abandon them to the State, have them appraised and slaughtered with indemnity, the uniform practice has been to accept the latter alternative. Not a single owner, I believe, has elected to keep a herd in quarantine and sell the milk sterilized. The result has been that four times the number of applications come in that the appropriation will warrant the officials to take in hand.

A special feature of the Pennsylvania method is the provision that a stockowner can have his herd examined, and tested with tuberculin, at his expense, the State to furnish a certificate setting forth the condition of the animals. In case of infection, the owner has the option of abandoning the reacting ones to the State, to be secluded, or appraised and slaughtered, he meanwhile guaranteeing that he shall introduce no new animals into the herd except by tuberculin test under the direction of the State Board.

The usual provision is in force that no indemnity is allowed for any animal that entered the state not more than three months

before, and without the tuberculin test demanded of all such stock animals.

Indemnities are restricted to $25 per head and under for non-registered animals and grades, and $50 for registered thoroughbreds. The average appraised price has been $23.

On the whole an excellent work is being done in Pennsylvania, and herds now tested are found to contain not more than half as many infected animals as did those tested a few years ago. A better showing would doubtless have been made if the State appropriation had permitted the board to give attention to all applications made.

The good results may be attributed to the adoption of a system which secured the confidence and trust of the stockowner: he sees that the State has no desire to oppress nor injure him; he is in no dread of a high-handed confiscation of his remunerative property without indemnity; while advanced cases are destroyed he is left the option of segregating his latent cases and marketing their products under the precaution of sterilization; he can raise the progeny of these animals if he will, under similar precautions; he can sell his stock and its product under government certificate if they prove sound; he can avail of government assistance in protecting his herd against reinfection, by the testing of animals to be purchased. Under such a system there is no object in secreting infection, nor in underhanded sale of tuberculous animals, with the spread of the disease into new centres. Even the tardy or inimical stockowner is roused to action by public opinion, when he finds the market for his suspected product growing more and more uncertain, and feels the daily encreasing pressure of opinion among neighboring owners, that he is exceeding his rights in maintaining an infected herd in the very midst of their purified ones.

A most important item of the system of Pennsylvania, and of sixteen other states, is the compulsory testing of all stock cattle introduced into the State. This has the further support of the Federal Government which demands a tuberculin test of every stock bovine animal imported into the United States. This is a natural corollary of every attempt to restrict or extripate tuberculosis from a State, but when through willful blindness a great State like New York, repudiates a measure of this kind, it but

makes its dairy and breeding herds the depository of the reacting animals that other states wisely exclude. The sifting process goes on at Buffalo and other centres and the New York herds are loaded with the rejected, diseased animals. Under the operation of this, it will be wonderful, if the New York stock owner is not soon roused up from his idle dream of fancied security, by a great extension of the infection.

Generally Applicable Measures. Under any system many special rules will be demanded in individual cases, which it will require the skill of the expert sanitarian to apply, and which cannot be referred to here. The following are generally applicable whether by the owner in his particular herd, or by the State or other authority dealing with the subject.

1st. On discovery of a case of tuberculosis, test the whole herd with tuberculin. (See Tuberculin Testing.)

2d. Remove all animals that show a typical reaction.

3d. Destroy and burn, boil or deeply bury all cases of the disease, unless it is decided to form an isolated herd of latent cases which are in good condition and apparent good health.

4th. In case of doubt or disturbing influence which may have caused rise of temperature (calving, heat, exposure, concurrent disease, changes in management, etc.), keep the suspected animals apart for four weeks and test again.

5th. Repeat the test every six months, and if two successive tests show no indication of tuberculosis, the herd may be accounted sound.

6th. As soon as tuberculous animals have been removed from a stable, let it be vacated and thoroughly disinfected with chloride of lime, 4 ozs. to 1 gallon of water, and enough quick lime to make a good whitewash which will show if even a square inch is missed. When chloride of lime is objectionable because of its tainting the milk, use mercuric chloride 1 dr. to 1 gallon water, with enough sal ammoniac to make it freely soluble. The roof, walls and especially the floor, gutter and feeding trough must be first thoroughly scraped, washed and cleaned, all rotten wood work must be removed, and, in case of double boarded walls, the boards must be removed on one side to permit of a thorough application. Utensils should be scalded and dressed with carbolic

acid, 1:20. A stable incapable of disinfection should be abandoned for a length of time or burned.

7th. In making new purchases, avoid any herd in which tuberculosis has appeared, or that has had sickness or deaths in recent years.

8th. Do not purchase from city, suburban nor swill stables.

9th. Do not take a cow that is in ill health or low condition, especially one with cough, nasal discharge, foul breath, wheezing breathing, hard nodules under the skin, diseased udder, swollen bones or joints, or a tendency to scour or bloat.

10th. Test every animal with tuberculin before admitting it into the herd.

11th. Do not admit strange cattle to house, field or yard. Keep apart from the herd until tested.

12th. Keep each animal strictly to its own stall and manger.

13th. Board up between the stalls in front so that no two cows can feed from the same manger.

14th. Be especially watchful of the older cows and on the slightest sign of ill health, separate and subject to the tuberculin test.

15th. In case a herd of cattle is found to be tuberculous, subject to the tuberculin test all domestic animals that have mingled with them freely and fed from the same troughs. Remove those that show a reaction.

16th. Exterminate the vermin (rats, mice, sparrows) in a building where tuberculosis has prevailed.

17th. Let no consumptive person attend on cattle nor prepare their food.

HYGIENE OF MILK AND MEAT. SANITARY POLICE MEASURES.

This has a reference to both man and beast and involves measures of private and public hygiene alike. In man, as we have seen, milk is the source of greatest danger, being habitually taken uncooked, whereas meat has been usually subjected to a high temperature before it comes to the table.

Milk. The unsterilized milk of the tuberculous cow must always be regarded with grave suspicion. Especially is the drinking of milk warm from the cow to be strongly condemned. The feeding experiments of Vilemin, Gerlach and others, in 1866 to

1869, demonstrated the virulence on the lower animals of the milk of infected cows, and many accidental infections have shown the same for man. (See relation of the bacillus of man to that of animals). This conceded, the question arose as to the virulence of milk drawn from the sound udders of tuberculous cows. Many experiments of Nocard, Galtier, McFadyean and others seemed to decide against such infection. Others have had a different experience, and especially when the test was made by inoculation. Ernst found tubercle in 30 per cent. of the cows he examined, though no affection of the udder could be made out. Theobald Smith, Hirschberger, Bang and others found tubercle bacilli in milk from sound udders. Pearson, injecting the milk from sound udders intraperitoneally into Guinea pigs had ten in sixty-three affected. Rabinowitsch and Kemper found tubercle bacilli in milk, in ten out of fifteen cows with sound udders. While it must be allowed that often a very small proportion of tuberculous cows with sound udders pass the bacilli in the milk yet a sufficient number of exceptions are found to deter one from endorsing such milk as safe. Much more is this conclusion justified by the consideration that commencing mammary tuberculosis is in many cases unrecognizable by ordinary examination intravitam or post mortem. Again when the bacilli are circulating in the blood (generalized tuberculosis) they can escape from such a vascular tissue as the mamma without the formation of a local lesion, as they can pass through the intestinal walls or lungs and colonize the adjacent lymph glands. Nocard's injections of the bacilli into the veins seem to show that they disappear from the blood in 4 to 6 days, but with a generalization of the infection from within, the presumption is against a single isolated entrance of bacilli, and in favor of a continuous introduction. In such generalization therefore the bacilli circulate in all vascular tissues, and are liable to escape with any normal secretion, but especially with the milk or urine on account of the great vascularity of the glands.

When the udder is itself tuberculous the case is incomparably worse. The milk can scarcely fail to be infecting, and the bacilli, grown in these highly vascular tissues, apart from the air, are usually of a very virulent type. Of these Martin writes in the report of the Royal Commission (England): "The milk of cows with tuberculosis of the udder possesses a virulence which can

only be described as extraordinary. All animals inoculated showed tuberculosis in its most rapid form." Woodhead is equally positive in this position. Galtier says "we should absolutely avoid the consumption of milk from cows in which the udder is tuberculous, and boil before using, the milk of all cows affected with, or suspected of tuberculosis in any form." Finally we should never consume without boiling, milk of which we do not certainly know the origin, and especially in cities, we ought not to omit this precaution in the case of milk furnished by milkmen." Rabinowitsch and Kemper add: "Milk from cows that react to tuberculin must be suspected of being tuberculous in every case."

It may well be allowed that the mixed milk from a large dairy, containing but one or two tuberculous cows, is much less infecting than that of the tubercular cow herself, and that skim milk that has been passed through the separator has been robbed of many bacilli which have been precipitated in the albuminoid material that collects on the inner side of the bowl. But these mean dilution not purification; they reduce the danger but do not altogether remove it, and sanitary police should aim at sterilization in every case where available.

Butter, *cheese*, *whey*, and other dairy products have been proved virulent in different cases (Galtier, Heim, Lasar, Bang, Obermüller, Roth, Groëning, Gasperimi, Petri, Rabinowitsch). This was true for butter 120 days old, and cheese 330 days. In a number of cases Rabinowitsch found a bacillus, similar to the tubercle bacillus and producing tubercle-like lesions, but differing in its staining, morphology and culture, and she supposed that other observers had mistaken this for the tubercle bacillus. Petri, Morgenroth and Hormann, found the bacillus of Rabinowitsch in company with the real tubercle bacillus in butter, and Rabinowitsch, later, in fifteen samples of butter found the bacillus tuberculosis in two. Pseudo-bacilli from dairy products, if decolorized by 30% watery solution of nitric acid, may be ignored (Abbott and Guildersleeve).

Bacilli coming from the mouth, bowels, or lungs, are also liable to get into the milk, through the floating dust of the stable, or from the teats, udder, tail, etc., (Gaffky).

Oleomargarin cannot be considered as free from this indictment, for though the tuberculous glands, etc., from the mesentery, may

be sterilized in the preliminary heating process, yet the subsequent mixing with milk is liable to convey the infection.

The ideal course with milk, if the herds cannot be purified from tuberculosis, would be to compulsorily remove from the herd every cow that shows objective symptoms of tuberculosis, or any internal disease of the udder, and to subject all the milk of the remainder to Pasteurization at a temperature of 155° F. for twenty to thirty minutes. This, however, requires skilled and faithful management to avoid renewed contamination from the lips of the vessel which may have escaped the heat, or from hands, vessels and objects that were in contact with the milk before. The boiling temperature for fifteen minutes would be a safer resort, as requiring less careful handling, yet even this may be contaminated afterward under poor management. It has the further drawback of the boiled milk taste and the coagulation of the albumen. But the outlay for such careful sterilization will soon amount to more than will the tuberculin test.

Meat. For various reasons meat must be held less virulent than milk, but mainly because it is less frequently the seat of tubercle than the udder, and because it is usually cooked before being eaten. The muscular tissue of the ox appears to be unfavorable to colonization by the bacillus, and although the intermuscular lymph glands, do not partake of this inherent resistance, yet as the glands are very frequently affected from the tissues which they drain, they necessarily partake in some degree of the comparative immunity of the muscles. This immunity is, however, far from complete, as the frequent implication of the intermuscular glands (prescapular, axillary, prefemoral, etc.), sufficiently show. Again in estimating the virulence of meat we must never forget that in the great majority of cases, in ordinary infected herds, the tubercle is still essentially local; no generalization has taken place. The muscle is vascular throughout, and in cases of generalized tuberculosis is infecting, yet a single transient escape of tubercle bacilli into the blood does not ensure permanent infection of that fluid, which can usually purify itself in six days or less (Nocard). On the contrary, when the escape of bacilli into the blood is constant, of necessity the virulence of the blood is constant and a rapid generalization ensues. Such continuous escape may occur in actively advancing tuberculosis at any point, but it is more certain if the degenerating tubercle

has opened through the walls of the vessels (capillaries), so that the infecting bacilli can pass into the blood in a continuous stream. The pus and other microbes, in complex infections, hasten this degeneration and contribute to generalization of the tubercle and emaciation. The transient infection of the blood with the pure tubercle bacillus does not, however, lead to emaciation and marasmus, and hence the frequency of high condition in spite of extensive tuberculosis.

Ostertag gives the following as indicating blood infection: "When with emaciation there is evidence of recent blood infection, enlargement of spleen and all lymph glands, miliary tubercles of the lungs, liver, spleen and kidneys." In the absence of these indications, though there may be numerous old-standing tubercles, caseated, calcified or sclerosed, he considers that there is no reason to dread infection of the blood and carcass. Even a tuberculous intermuscular gland does not, in his opinion, condemn the adjacent muscle. When vomicæ (caverns) are present in the lungs and internal organs the flesh may still be used "if embolic tubercles of different ages, indicating repeated eruptions of tubercle bacilli into the blood stream, are absent from the spleen and kidneys." This meat is, however, to be sterilized before marketing (Ostertag).

The United States Bureau of Animal Industry orders the destruction of all cases of "extensive or generalized tuberculosis;" "any disease or injury causing elevation of temperature or affecting the system of the animal to a degree which would make the flesh unfit for human food;" also "any organ or part of a carcass which is * * * affected by tuberculosis * *."

In most countries of Europe even emaciated animals are used for food for animals or even man, provided the wasting is not too extreme. The carcass must, however, first be subjected to a temperature of 230° F. for a period of three hours. This is sold in a special market as low priced meat and labelled as such. Ordinary cooking does not always sterilize, as Martin and Woodhead, like Vilemin, found living bacilli in the centre of a cooked six-pound roast.

Immunization. Attempts have been made to immunize the system against tuberculosis. The most notorious is that of Von Behring who claims that in three months, by three successive

intravenous injections of a "bovovaccin", he can render the system of the calf refractory to virulent bacilli, even when inoculated. Thousands of his protected calves have been exposed to casual infection with comparative impunity, but a certain number have apparently resisted virulent inoculation as well. This is supported by experiments of Vallée and others in Europe, and of Buckley in America. Its advocates concede that the method fails with mature animals, and various experimenters have found the results inconstant. Pearson, using the avian bacillus and later a nonvirulent bacillus from man, intravenously, has also had a large measure of success. In all alike a number of check calves, under virulent inoculation, but without previous protective treatment, developed tuberculosis, mostly generalized. A measure of protection is undeniable, but cannot be safely made use of when radical, systematic work is carried on for the extinction of the germ. Von Behrings' "bovovaccin" fails with mature animals, its advocates themselves being judges: there is evidence from other sources that it fails at times in the young; for the cases in which it appears to succeed we have as yet no sufficient evidence that the protection is permanent.

In estimating its value the unequal virulence of tuberculosis at different times and places must also be considered. Sometimes whole families or herds are decimated or killed out in a few months, and at others we see tuberculous men and families live to old age, and herds, generally affected for years, with no particular indication of general ill health. In my hands a large herd has just shown 50 per cent. reactions to tuberculin, yet the general health seems good and the yield of milk generous. In these cases there is an acquired tolerance (partial immunization), yet the presence of every infected man or beast is a continual source of danger to the sound and unsound alike. The individual, partial immunity thus becomes a factor in the preservation and extension of the infection. Some day, under encreased virulence, or greater susceptibility, the disease will assume a more fatal type, and the losses will encrease in ratio with the numbers affected. Such deadly outbreaks have been noted among Indians, Esquimaux, Oregon herders, Transvaalers, Negroes, and Philippinoes, and in Jersey, Guernsey and other thoroughbred cattle, and in all cases the preservation of the

bacillus is an invitation to such mortality whenever the susceptibility and environment become specially favorable.

Again tuberculosis occurring casually does not naturally induce a marked encrease of immunity in the animal attacked. In an animal system that does not succumb readily and perish, the bacillus does not usually die, but in such tolerant system it may live for a long lifetime. It cannot, therefore, be hoped that a constant, satisfactory and lasting immunity can be educed by artificial injections of the pathogenic product. In this, tuberculosis differs widely from all acute, self-limiting diseases (lung plague, rinderpest, anthrax, blackleg, measles, scarlatina, variola, etc.) which are followed by a striking immunity, and thus offer themselves for artificial immunization.

To secure even the uncertain and imperfect immunization of cattle from tuberculosis, at $2 per head would demand $120,000,000 for our 60,000,000 cattle, and by the repetition of this at short intervals, for each crop of calves, or to replace cows that die, or are removed from the dairy, it would impose a grinding tax, with no prospect of abatement. Large as would be the outlay for a complete extinction of the bacillus of cattle by rational means, it would be but a trifle compared with a perpetual tax like this.

But if the people and their legislators decide to preserve tuberculosis in their herds indefinitely, then the owner of a valuable herd may reduce his losses by applying the confessedly imperfect immunization to the calves of his tuberculous herd. But this is a mere make shift, with no hopeful promise of complete extinction of the infection, or of the absolute protection of the human family from infected meat and dairy products.

PSEUDO-TUBERCULOSIS FROM STREPTO-BACILLUS.

As early as 1885 Malassez and Vignal recorded a form of tubercle in the Guineapig by inoculation from a subcutaneous gland of an infant which died of supposed tubercular meningitis. The microbe appearing in pure cultures was a very short bacillus

in chain form, and aggregated in the form of zoöglœa. Nocard found the same in an epizootic in *chickens*, Eberth in a *rabbit*, and Chantemesse, Charrin and Roger, Nocard and Masselin, and Pfeiffer in the Guineapig, and others in the *rabbit*, *hare*, *dog*, *cat*, *horse* and *mouse*.

The *bacillus* is 1 to 2μ long, with rounded ends, in chains and zoöglœa. Some are ovoid or round. It is ærobic, (facultative anærobic), stains readily in anilin colors and bleaches in Gram's solution. Grows readily in peptonized bouillon, gelatine or blood serum, with foul odor.

Symptoms. These were mainly a progressive emaciation with weakness, apathy, sluggish movements, breathlessness and wheezing when hurried, and in one cat (Hoen) weeks of semiconsciousness.

The *lesions*, caseated or purulent, miliary, pea-like, or even in larger conglomerate masses were mainly found in the liver, spleen, and intestine but also subcutaneously.

Though less common than tuberculosis, this may become specially virulent and widespread in the small rodents and should be extirpated by segregation and destruction of the sick, disinfection, new buildings and runs, care of water and food supplies, etc.

PSEUDO-TUBERCULOSIS IN CALF FROM DIPLO BACTERIA.

Vallée, in 1898, described a miliary tuberculosis of the liver, occurring endemically year by year in young calves in the same herd. He traced it to an ovoid bacillus, smaller than that of swine erysipelas, nearly as broad as long, nonmotile, often in pairs, but never chains, asporogenous, staining even in Gram's (I) solution, and growing freely in bouillon, glycerined potato, gelatine and gelose, forming transparent white or bluish gray colonies. It grows in acid and fails to coagulate milk, ferment sugar, or form indol. It proved pathogenic for the calf, Guinea-pig, rabbit and pigeon. Thermal death point 70°C.

There were general *symptoms* of extreme weakness and complete anorexia. *Lesions*, confined to the liver, resembled miliary tuberculosis but without implicating the lymph glands. The individual tubercle was grayish, and two or more had often coalesced. The hepatic tissue was soft and friable, with a great excess of phagocytes, and destruction of the acini, with an outer zone of congestion. The initial lesion occurs in a capillary, usually in the periphery of an acinus. The endothelium is swollen, and invested by bacilli, which block the vessel. The hepatic tissue proper is invaded later. This intravascular origin of the lesion, and its strict limitation to the liver, supports Vallée's theory of infection through the umbilicus, while the onset not before eight to fifteen days after birth, implies a tardy propagation of the germ. Intravenous injection kills the Guinea-pig in 7 or 8 days with liver and peritoneal lesions, the rabbit in 2 to 3 days with hepatic, peritoneal and even pulmonary lesions, and the dog and pigeon in 2 days with hepatic lesions. Subcutaneous injections caused a purely local lesion in Guineapigs and none in pigeons.

Prevention. Vallée succeeded by disinfecting the buildings, and boiling the milk before feeding to the calves. Antisepsis of the naval immediately after birth is another obvious precaution.

TREMBLING IN SHEEP. LOUPING-ILL. INFECTIVE MYELO-MENINGITIS. IXODIC TOXAEMIA.

Definition: infective, tick borne disease, characterized by meningo-myelitis. Animals susceptible: sheep, and possibly swine (Meek, Greig-Smith) and cattle (Williams). Known in North Britain only in spring (in Skye also in autumn), on rough pasture with much brush, and wood ticks, (Ixodes ricinus, erinaceous, marginatus or other). Experimental infection. Bacteriology: Bacterium fluorescens β and γ found in exudate and infecting. Accessory causes: dried grass of previous year, brush, low condition, cold, youth. Symptoms: incubation 10 to 30 days; impaired innervation, hyperæsthesia, timidity, excitibility, trembling, jerking, lack of coördination and balance, falling, convulsive struggling, jumping, rolling of eyes, stiffness, opisthotonos, paresis, paralysis of hind—later of fore limbs, apathy, Wry neck, arched back, stiff joints. Diagnosis from *myelo-meningitis* by its ezoötic appearance, in spring, on tick infested ground: from *paralytic rabies*, also by absence of that disease locally; from *tetanus* by its general prevalence, the absence of tonic spasm, and presence of palsy; from *braxy* by the lack of emphysematous swellings, and of speedy sepsis; from *anthrax* by usually healthy spleen and its confinement to sheep. Lesions: cerebral meningitis with encrease of subarachnoid fluid, of myelon reddened, softened, also of other serosæ, stomach, bowels, liver and kidneys. Prevention: destroy ticks in winter by burning grass and brush, by ploughing and cropping; or fence off half the pasture one year and the other half next; or lime soil; or dip repeatedly in April, May and June to keep off ticks; avoid moving sheep in these months: Give liberal feeding. Mortality 10 to 20 per cent.

Definition. An infective disease of sheep, inoculated by ticks, and producing a meningo-myelitis, with drowsiness, hyperæsthesia, irritability, paresis and other nervous disorders.

Animals susceptible. This is almost exclusively a disease of sheep, yet Meek and Greig-Smith claim to have seen it in swine that have eaten the raw carcasses of *louping-ill* sheep, or that have ranged the tick infested pastures, and in rabbits inoculated with the microbe from the wound caused by the tick. W. Williams claims to have seen well marked cases in cattle, and heard of cases in horses and swine. He speaks, however, rather obscurely of "*the tick disease*" and seemingly includes in this all affections inoculated by ticks.

Geographical Distribution. This disease has been hitherto described as existing in the northern part of Great Britain only,

but given the presence of the tick, and of the infection which it carries and the malady might easily be extended indefinitely. It is known to prevail in Northumberland north of the Tyne, in Kirkcudbright, Dumfries, Ayrshire, Lanark, Peebles, Roxburgh, Berwick, Argyle, Inverness, and Ross. In Berwick it is less prevalent than in the other countries named, while in the Western Isles it is not only widely spread rising on the hills 2000 feet or as high as the sheep range, but in Skye there are two distinct outbreaks, in spring and autumn respectively, apparently coinciding with the appearance of two successive generations of ticks. This may be due to the prevalence of warm winds from the Gulf Stream. Further investsigation will doubtless show a much wider distribution—the author has seen an affection bearing the same general characters on the spurs of the Lammermoors in East Lothian, and the supposed adaptation of the Norse word *hloupa* (staggering) suggests that it is probably not unknown in Scandinavia. W. Williams notes its prevalence on the Silurian formation, but ticks confine themselves to no geologic stratum, and the tick is the main agent in carrying infection.

Causes. 1st. Sheep Ticks. The sheep tick is not the *ked* (Melophagus ovinus) which is common on long wooled sheep everywhere and is an example of a wingless, degraded dipterous insect.

The sheep ticks, on the other hand, are true ixodes, and of the same family with our common wood tick and of the cattle tick (boophilus annulatus) of the Southern States and the West Indies. The ticks collected from the sheep by W. Williams were identified by Mr. Moore, of the British Museum, as ixodes ricinus, ixodes erinaceous and ixodes marginatus. Those obtained by Meek and Greig-Smith showed the following characters: The male is 2.48 mm. long, by 1.30 mm. broad; the female is 5 to 5.5 mm. long, by 3 mm. broad, or when gorged with blood, 10 mm. long, by 7 mm. broad. The fasting female is yellowish green, and when full of blood, blue. Following the rule of their genus, the mature tick has eight legs, the larva but six. The fasting larva has head, legs and dorsal plate (scutellum) brown, the remainder of the body yellow. Scattered hairs appear on the body, legs and maxillæ.

In all ticks the rostrum is a characteristic feature. It consists centrally and inferiorly of a dart covered below and on each side

by rows of teeth turned backward, which when, imbedded in the skin, hold so firmly that the parasite may be pulled in two in any attempt to pull it out. Above this dart and on the two sides lie the cheliferæ (horns), each furnished with three or four teeth turned outwardly and more or less recurved, by which the dart is worked into the skin. Finally, on the lateral sides of this central apparatus, are the two maxillary palpi, which are not inserted into the skin, but applied against it and operate as feelers prior to and during the insertion of the dart and cheliferæ. The maxillary palpi are club shaped and soft.

Ticks pass through three moultings before they attain to the sexually mature eight-legged form, and though the hexapod larvæ attach themselves to animals and irritate the skin by their bites, it is only the mature, impregnated egg-bearing female that lives exclusively on blood and sucks this to excess.

While given species of ticks show a preference for particular genera of animals, yet ticks generally in their vagabond life will leave the long grass and brush where they have been hatched to become temporarily the guests of any passing animal. It is, therefore, premature to seek to identify any single species of tick as the only bearer of the infection, and it is quite possible that any one of several species may contribute to its propagation.

Experimental Infection by the Tick. W. Williams muzzled four sheep from a healthy district and turned them for several hours a day on a tick-infested field, and two sickened—one on the eighth day and one on the sixteenth. Twelve ticks sent out of the district and put on a healthy sheep caused illness on the tenth day. In a second experiment with ten sheep, during a colder spring, when there were fewer ticks, no deaths occurred.

Meek and Greig-Smith turned twenty sheep on a tick-infested louping-ill pasture, six of the number wearing muzzles to prevent grazing, seven having been dressed with a mixture of sweet oil, 2 quarts; castor oil, 1 quart; train oil, 1 quart; pitch oil, 3 gills, and cade oil, 1 gill, while the remaining seven were unmuzzled and undressed. The muzzled sheep were regularly taken out and fed on food from a healthy locality. At the end of a fortnight oils were reapplied on the second lot (seven sheep). No ticks appeared on these sheep, while many were found on the undressed ones. On the fifteenth one of the unmuzzled and un-

treated sheep sickened and died with lesions of louping-ill. On the twenty-second day one of the muzzled sheep sickened, and died of louping-ill next morning. On the thirty-first day another of the muzzled sheep took ill and on the thirty-eighth was killed. It showed characteristic symptoms and lesions. The seven salved sheep, on which no ticks could at any time be found, remained healthy throughout.

Bacteriology. W. Williams describes a bacillus which he figures as forming filaments of very uneven breadth, with frequent branching (contrary to the habit of bacilli), and forming spores in clusters. These were obtained from the coagulum of the cerebro-spinal fluid and Meek and Greig-Smith conclude that the alleged mycelium was but the filaments of fibrin.

McFadyean found pus microbes.

Meek and Greig-Smith (Veterinarian, 1896-7) found in the black bloody swellings under the skin, where the ticks had inserted their proboscides, a variety of microbes which in pure cultures did not provoke louping-ill. These included staphylococcus cereus albus, sarcina lutea, bacterium putridum, bacterium coli commune, micrococcus sulphureus, micrococcus bicolor, and micrococcus candicans. He also found penicillium glaucum. Two organisms allied to the bacterium fluorescens and designated as β and γ (G) respectively, were found in these sores and produced in rabbits and sheep nervous disorders and lesions which could be fairly identified with louping-ill. These are about 1.5 to 1.7μ long by 0.7μ broad, and chromogenic with a special fluorescent appearance. Microbe β inoculated on a rabbit caused on the second day rhythmic movements of the head downward and to one side, the eyelids closing as the head dropped, as if the animal were constantly falling asleep. In another rabbit it caused stiffness of the legs only and in a third it had no effect. Microbe γ when cultivated ærobically was harmless to rabbits, but when grown anærobically on mutton bouillon to which a drop of blood had been added, it caused on the second day general paralysis of the neck and limbs, spasmodic twitching of the muscle, dyspnœa and feeble heart action. Two rabbits which survived the early effects developed large axillary and inguinal abscesses four months later.

A lamb inoculated with the bouillon culture of γ showed after thirteen days, lameness of one hind limb, with paresis, and a dis-

position to fall to one side or the other. Two months later, when it had greatly improved, a second inoculation of microbe γ grown anærobically in bouillon and blood, caused, on the third day, a severe aggravation of the lameness.

Accessory Causes. Much depends on the abundance of old dried grasses of the previous year in which the larval ticks may hibernate. Land that has been burnt over in winter, that is cleared of brush, or in which the aftermath has been killed by free salting or liming will be largely cleared of the ticks.

Whatever disturbs or undermines the general health lays the system open to the disease and many flockmasters in tick-infested districts have succeeded in greatly reducing the mortality by feeding sound hay and oats in winter. Sudden changes of weather have long been noticed to coincide with outbreaks of the disease. A change to cold and wet is especially dangerous as causing a chill and robbing the system of its tone and vigor. But a sudden access of warm spring weather, especially if at the same time moist, may have a decidedly predisposing effect by lowering the general tone. A fatal paresis common in the flocks of New York, in the absence of ticks, shows a similar tendency to select the atonic animal. This occurs mainly in spring, when the sheep have been shut up in close confinement for weeks or months, with flaccid muscles and fatty livers, and above all if they are in advanced pregnancy with twin lambs, and if their fleeces are extra heavy. In both affections the majority of the flock escape, while those that are specially predisposed succumb. Lambs suffer most, doubtless because of relative weakness, and on account of their innate and unexhausted susceptibility.

Symptoms. After an incubation varying from ten days to thirty some impairment or disorder of the innervation is shown, varying widely, however, in different cases.

The two names "trembling" and "louping-ill" long used by shepherds as characteristic of the disease indicate spasmodic disorder of a clonic kind, the paresis which is essentially passive having been very naturally overlooked, or held to be subordinate. There is hyperthermia the temperature rising at times to 105° or higher, and often marked hyperæsthesia and excitability at the outset. On approaching the patient it is very much frightened, and when caught, struggles and twitches in a remarkable manner

or trembles violently. If merely raised or disturbed the trembling or clonic spasms are very marked, the nose is jerked forward and upward from the contraction of the muscles attached to the occiput; the legs may be lifted jerkingly as in stringhalt; when raised they are moved stiffly or sway uncertainly before the foot is once more planted; or the sheep loses its balance, falls to the ground and struggles convulsively in its efforts to get up and escape. As the result, it will in certain cases jump to its feet, rising meanwhile to an undue height in the air. In other cases there is squinting or rolling of the eyes, and movements of the jaws with frothing from the mouth. Or the spasms may be tonic affecting especially the muscles of the back and loins, and causing extreme stiffness or rigidity or even oposthotonos. Lambs are unable to suck.

But whether the early spastic symptoms are well marked or not, paresis and even paralysis set in sooner or later. This usually begins as paraplegia, or exceptionally one limb only may be affected at first, causing the animal to walk on three legs. For a time the fore limbs may be free, and the patient attempts to move by dragging the hind limbs, which are extended backward. When the fore limbs become involved the animal remains down helpless and after awhile apathetic. Temperature and sensibility are both greatly lowered in the paralyzed limbs. Sometimes the spasms are lateral and the head may be drawn to one side.

In the animals that survive the early attack, there is likely to remain some lasting deformity, such as wry neck, arched back, stiff or swollen joints. Abscesses, which appeared in the inoculated rabbits after a lapse of four months, are a not uncommon sequel in sheep, the pus collecting in the neighborhood of a joint, or of the lymph glands of the axilla, inguinal region, breast or shoulder.

The succession of symptoms are in the main such as are observed in other cases of myelo-meningitis, first exalted function and later depressed and abolished.

Diagnosis. From other forms of myelo-meningitis it may be distinguished by its enzoötic occurrence on tick-infested pastures, which already have a reputation for causing this malady, by its appearance only in the season of the development of the tick, by the presence of the tick or of its sores on the skin, and by its

entire absence from adjacent fields which are free from ticks. From paralytic rabies it is differentiated by the same conditions, and as a rule by the absence of rabies from the district and of any evidence of a bite. In tetanus the tonic persistent nature of the spasms, the absence of paralysis, the marked spasms of the muscles of the eye, and the usually isolated condition of the case should prevent any confusion. Braxy is to be distinguished by its emphysematous swellings near the surface of the body, and by the comparative absence of hyperæsthesia, spasm, or paralysis. The carcass in braxy undergoes much more rapid decomposition. Anthrax is more rapidly fatal, shows no such marked nervous disorder, has a dark, nonoxygenated and often incoagulable blood, an enlarged sanguineous spleen, and the characteristic anthrax bacilli. It attacks the larger herbivora as readily as the small.

Lesions. The most constant and striking lesions are found in the nerve centres. In many cases there have been found cerebral meningitis, involving the choroid plexus (Fair, Hamilton, Klein, Murray, McFadyean) with an encrease of the ventricular and subarachnoid fluid (Murray, Hamilton, Williams). The exudate may be yellow or rosy from contained blood globules (Klein). The meninges are thickened and the seat of ramified redness. In the region of the spine inflammation is found not only in the meninges but also in the cord, which may be blood-stained, softened (Mathewson, Goodwin, Robertson), or in older cases indurated (Robertson, Hamilton, Williams, Young). In this last condition there is a sclerotic condition of the neuroglia, and it may be a distinct atrophy. The exudate is usually abundant and more or less coagulated into a soft, diffluent jelly.

In many cases there is inflammation of the serous membranes of the chest (pleura, pericardium, endocardium), and even of the lungs (Fair, Hamilton, Klein). In some instances there has been inflammation of the stomach and intestines (McFadyean), liver and kidneys (Klein), and enlargement of the spleen has been noted. The most constant lesions appear to be those of the nerve centres, but the wide variety of organs involved in different cases sufficiently accounts for the variability of symptoms.

Prevention. As the ticks are the chief media of infection, the disease may be eradicated by their destruction. The burning of all withered grass and brush during the winter months will do

much in this direction. Their destruction is rendered even more complete by ploughing and putting the land under a series of cultivated crops. By this means not only is the winter shelter of the tick removed, but the animal host which it requires for its complete development is denied it, and it must perish before the land is again seeded to grass. When the land is unsuited to cultivation, the same end may be in some measure secured by fencing off half the pasture, and leaving it unpastured for a season, meanwhile burning the dry grass or temporarily suppressing it by a liberal application of salt. The following year the pasture so treated may be restored to pasturage, and the other half subjected to the same course of treatment. In the absence of such thorough treatment, a liberal application of lime to a virgin soil will often bring a growth so fresh and appetizing that the stock keeps it closely cropped and thus removes the shelter for the offensive ticks. Finally, the ticks may be prevented from attacking the animals, by repeated use during April, May and June of a dip in which tar oil, cade oil, heavy petroleum or other odorous insecticide forms a component part (see the dip of Meek and Greig-Smith.

A very obvious precaution is to avoid the movement of sheep during April, May and June from tick-infested pastures to others which furnish rank grass, brush or other suitable shelter for the preservation of the parasite.

It has been noticed that sheep indigenous to the tick-infested and louping-ill pastures are less susceptible than those that have been introduced from outside, but, as yet, no attempt appears to have been made to secure immunity by the use of sterilized products of the microbe, nor therapy by the resort to anti-toxin. A liberal and tonic diet is an important element in prevention Grain and hay should therefore be allowed whenever necessary to bring the sheep to early summer in good condition.

Treatment can hardly be said to have been attempted, though mild cases are allowed by the shepherds to recover. According to Meek, the deaths often average 10 to 20 per cent. of the flock.

BRAXY, BRADSOT, GASTRO-MYCOSIS OVIS.

Defininion : Acute, infectious, bacteridian disease of sheep, with colic, enteritis, emphysematous swellings, dark, diffluent blood, and after death rapid putrefaction. Geographical distribution : Iceland, Norway, Faroe Islands, Scotland. Causes : inclement weather, exposed localities, low condition, winter food, chill, frosted grass. Bacteriology : Bacillus gastromycosis ovis : 2 to 6 μ by 1 μ in pairs or filaments, sporogenous, polar staining, anaerobic, liquefying, gasogenic, found in the gastro intestinal congested mucosa, serosa, liver, kidneys, blood. Pure cultures by boiling five minutes. Pathogenic to sheep, Guinea pig, mice, pigeons, hens and less certainly rabbits. Symptoms : resemble blackquarter, sudden, rapid, fatal, back arched, stiff hind parts, crepitating swellings on hind parts or elsewhere, colics, tympany, anorexia, pulse and breathing hurried, separation from flock, lying, drooping head, ears and eyelid. Usually found dead in morning when apparently well previous night. Lesions : early putrefaction, slight, transient rigor mortis, tympany, foetor, sero-sanguineous exudates under skin, on fourth stomach and bowels, and elsewhere. Contents of large intestine dry, hard. Effusions in serosa. Spleen enlarged or not. Liver and kidneys congested, softened, small, pale necrotic areas containing bacillus. Prevention : drainage and cultivation, winter feeding, abandon infected pastures in late fall and winter as pasture for young, avoid overstocking, burn old fibrous heather. Immunization by sterilized kidney extract. Treatment.

Definition. An acute infectious disease of sheep, manifested by sudden attack, colicy pains, inflammatory and sero-hæmorrhagic lesions of the bowels, and sanguino-emphysematous swellings occurring subcutaneously and especially in the hind quarters. The blood is dark, tarry and comparatively incoagulable and after death putrefaction advances with extraordinary rapidity.

Geographical Distribution. Braxy is generally prevalent in Iceland where it was described over a century ago (1778) by Ketilson as "vinstrarfár" or "vinstrarplága" ("Omasum disease"). In Norway it prevails on the whole Atlantic border to as far north apparently as the sheep industry extends (Stavanger to Tromso Amt). The Faroe Islands are said to be affected throughout. In Scotland it prevails like louping-ill along the west coast especially, and embraces Caithness, Ross, Cromarty, Banff, Inverness, Aberdeen, Argyle, Bute, Ayrshire, Lanark, Galloway, Dumfries, Peebles, Selkirk and Roxburgh.

Causes. Like louping-ill this affection is associated with inclement seasons, exposed localities, and insufficient or indigestible food, but it differs in being an affection of autumn and winter rather than spring. It is rarely seen in summer. At a time when it was looked upon as an acute indigestion, its coincidence with hard frost or deep snow, was explained on the basis that the victim had been driven to eat dry, fibrous, indigestible grasses, brakens and heather. W. Williams who formerly identified the disease with anthrax, seemed to go back to this theory of indigestion. Though that is no longer tenable, yet it would be wrong to ignore the effect of inclement weather and unwholesome food in predisposing the animal system, and robbing it of the healthy tone which would otherwise have successfully resisted the infection in many cases. The occurrence of deaths after frosty mornings more than during mild weather, suggests at once the chill effected in the animal, the chilling of the paunch by the frosted grass eaten producing a subsequent congestive reaction, and the known facility with which frozen vegetables undergo rapid fermentation.

Cowan and Borthwick, (Transactions of Highland Society 1863) agree that the disease is especially prevalent when the land has been overstocked in summer, or when there has been a drought which withered up the pastures, and later a free growth of green herbage from the autumn rains. This they attribute to the "foul and unwholesome" character of the autumn growth, but it suggests no less the low condition of the sheep on the overstocked lands and the soft, aqueous character of the herbage grown rapidly in a comparatively cold season. Cowan quotes cases in which the lambs, weaned early and put in a separate pasture (hogg hirsel), suffered a mortality of 50 per cent., while in later years when allowed to remain with the ewes until winter, the deaths were reduced to 10 or 15 per cent. Here the more rugged health and vigor were manifestly strong prophylactic factors.

Both Cowan and Borthwick incriminate the withered heather and the dry, fibrous ("tathy") and innutritious tufts of grass which make up a large proportion of many hill pastures in autumn and winter.

Cowan strongly condemns heavy smearing with tar, which he believes to encrease the mortality, by lowering the general tone of

the system: "Hoggs are very dull and listless for some time after being smeared with tar, more especially if heavily smeared; . . . . when the skin is taken off, every shed or opening in the wool where the tar has been laid on, is as distinctly visible upon the flesh as the stripes upon a piece of printed calico."

It has been further alleged that the deaths are especially common during the full of the moon, the usual explanation being that the sheep are tempted to feed in the night and overload the stomach. It may be added that at such times the grass is taken in a cold—(often frosted) condition so as to chill the stomach and cause a congestive reaction, and that short of this, under the night dews the microbian ferments are moist and ready to start into full activity, while the herbage if it has been partly frosted is particularly susceptible to bacteridian attack.

Bacteriology of Braxy. The essential cause of braxy was revealed by Ivar Nielson in 1888, who demonstrated on the local lesions of the alimentary canal and the capillaries of various internal organs a bacillus which he named *bacillus gastromycosis ovis.* This is 2 to 6μ long by 1μ broad, occurring in pairs or filaments. The organism has an eliptical form and stains deeply at the extremities, while the central, bulging portion fails to take the color, is highly refrangent, and represents the spore. The non-spore-bearing bacilli are long, uniform rods with rounded ends. The germ is anærobic, grows readily in serum glycerine agar, and is gas-producing and foul smelling. Liquefies gelatine. It is found abundantly in the congested parts of the abomasum and to a less extent in the bowels, in the mucosa, and in the submucous and subserous tissues. It is also found in the blood and in the small areas of degeneration in the liver and kidneys. Jensen obtained pure cultures through the survival of the spores when impure cultures were boiled for a few minutes.

Animals susceptible. Inoculation of the cultures subcutem produces an affection resembling malignant œdema, or braxy, in sheep, Guinea pigs, mice, pigeons and hens, and less certainly in rabbits. Inoculation of a calf had no deleterious effect, while a second succumbed in forty-eight hours. In its pathogenesis this microbe appears to be more closely allied to that of malignant œdema than that of black quarter, as the latter attacks cattle very readily and has little effect on rabbits, pigeons and chickens.

Symptoms. The more obvious manifestations of braxy bear a resemblance to those of black quarter, so that the two affections have been often confounded. In both the attack is sudden, the course rapid and fatal, the back arched, the step short, especially with the hind limbs, and there are swellings which crepitate on pressure on the hind quarters or some other part of the body. Symptoms of colic soon appear, the sheep lying down and rising frequently and moving the hind limbs uneasily, and the abdomen becomes distended, tympanitic and tender. Rumination and feeding are promptly suspended, and as a rule defecation as well. Urine may be passed frequently in small amounts and of a high color. The pulse is rapid and often irregular and the breathing hurried and labored. In some cases the abdominal pain is less acute, the animal standing apart with drooping head, ears and eyelids and frothy lips, or lying by itself at a distance from the flock. Sooner or later emphysematous swellings appear on some part of the body, but most commonly on the hind quarter, which have a soft, doughy feeling and crackle or gurgle on pressure. In the great majority of cases, however, the disease runs a rapid course and ends in death in a few hours, and as the attacks are mostly in the cold of the night, the victim, which appeared well at night, is found dead in the morning. Cowan has often noticed that those that stepped short at night were dead next morning. In certain cases the sheep stood obstinately to the end and fell dead as if shot. When death was deferred for several days a common result was shedding of the wool.

Pathological Anatomy. A marked feature of braxy is the early putrefaction of the carcass. The rigor mortis quickly passes and the carcass bloats up and exhales a putrid odor. On removing the skin one sees extensive sero-sanguineous exudations, mixed with bubbles of gas of an offensive odor. The abdomen is tense and tympanitic and the anus protrudes. Visceral lesions are especially marked in the fourth stomach, the walls of which are thickened in patches or throughout by a hæmorrhagic or sero-hæmorrhagic exudation of a dark purplish color. If killed in the early stages this may be very restricted in area, but becomes general in animals that have died of the disease. Similar exudations are found on the walls of the first three stomachs, or on the small or large intestines. The fourth stomach and duodenum are, as a

rule, empty of food, though containing a sanguineous liquid. The contents of the large intestines are usually dry and hard. Some serous exudate is usually present in the peritoneum, pleura and pericardium. The spleen may be normal or slightly enlarged. The liver is pale, soft and friable. The kidneys may be congested and swollen and usually show brownish areas of necrosis in which the bacillus is readily found. The blood is dark, and though it may be coagulated, the clot is usually soft and diffluent.

Prevention. Much may be done in the way of drainage and above all cultivation and liming of the braxy pastures, and again laying them down in grass. Cowan found that braxy diminished largely in ratio with the improvement and cultivation of the soil. A rotation of crops and the free æration of the soil tends to destroy an anærobic microbe or to render it nonvirulent. Winter feeding is another well attested source of protection. Hay with turnips, oats, linseed cake or oil cake seems to encrease the tone and vigor, and to counteract the fermentions in the digestive organs which lay the system open to attack. Wholesome and nutritious food then must be a main stay whenever the health threatens to be undermined by insufficient or unwholesome pasture, by dried or withered grass, ferns or heather, by the watery grass of recent and rapid growth, by frosted and partially decayed herbage, or by pasture exhausted by overstocking or drought.

Fields and hills known to be infecting must be abandoned especially in late fall and winter and in the case of the younger and more susceptible sheep. Understocking is always better than overstocking as the flock is kept better nourished, stronger and with a greater measure of tolerance and resistance. Upon land covered with old, fibrous, astringent heather, burning is often of great value. The new growth of young heather is much more digestible and nutritious, and destitute of injurious astringency, and maintains a stronger and healthier flock.

Finally the question of *immunization* arises. Nielsen attempted this by drying and heating to sterilization the diseased kidney and injected small quantities of this suspended in water. Sheep treated in this way in Norway and Iceland have had a circumscribed inflammatory swelling and afterward appeared to resist casual infection when placed on the braxy fields. Jensen carried

the experiment a step farther and inoculated his artificially immunized sheep with 2½ cc. of a virulent serum-gelatine-agar culture. It was rather unwell for half a day and walked lame but soon recovered. A dose of $\frac{1}{80}$ cc. of the same culture killed in fifteen hours a larger sheep that had not been artificially immunized.

Treatment. Though some cases of braxy recover yet the treatment of the disease can hardly be seriously considered as yet. A disorder which is so rapid in its progress, so early associated with such great and rapidly extending lesions of the most vital organs, and which proves fatal so early and almost invariably, leaves little room for successful treatment. In the milder cases, likely to recover of their own accord, this may be hastened by the internal use of antiseptics and purgatives, and perhaps even by antitoxic serum from the blood of an immunized animal.

SOUTH AFRICAN HORSE SICKNESS. ŒDEMA MYCOSIS. DUNPAARDZIEKTE. DIKKOPZIEKTE.

Definition: Geographical distribution: S. Africa; enzoötic or epizoötic on damp soils; damp, humid atmosphere. Pathogenesis: horses, and more mildly mules and asses, quagga; (cattle and goats?). Causes: green forage, from rich, damp lands, eaten dewy; cut and fed with dew on; dried in sun safe; so of horses stabled or coralled; in hot summer only; inoculable; penicillium; cultures cause the disease; debility. Forms: lung sickness—head sickness—blue tongue. Symptoms: incubation 8 days. Fulminant form asphyxiates in an hour. Acute lung form has rigor; remittent hyperthermia; prostration; dyspnœa; cyanosis; serous nasal discharge; frothing; gurgling breathing; cough; death in three or four days. Head œdema; general swelling and oozing of serum. Blue tongue: great lingual swelling; cyanosis; coldness; projection from mouth: salivation; stertor; asphyxia. Three forms combined. Mortality. Lesions: serous exudate coagulates with heat or blood; intermuscular exudates; whole head densely infiltrated; excessive bloody pleural effusion; punctiform petechiæ; lungs pale, yellow, great interlobular infiltration; or, if worked, hepatization; dark, congested mucosæ; blood diffluent; heart pale; spleen enlarged, blood-gorged; kidneys infiltrated; gastro-intestinal congestions; cerebro-spinal effusion. Prevention: keep indoors during summer and autumn; allow no fresh damp forage; or cut only after dew is off; check rein or muzzle; pastured horses must be stabled at night or in damp weather. Immunization: by protective inoculations; recovered horse is re-inoculated until a high grade of resistance is secured and his blood used to immunize.

Definition. An ectogenous, infective disease of solipeds in South Africa, characterized by intense vascular congestions, destructive changes in the blood and the profuse exudation of liquor sanguinis into the tissues of the affected parts.

Geographical Distribution. The affection is not known out of South Africa, where it has been observed since 1780. It appears yearly in certain areas in the Transvaal, Orange River Colony, Natal, and adjacent States, but only in certain years in Cape Colony and especially in its southern portion. In some years it makes wide extensions so that it has appeared to become epizoötic instead of enzoötic. The habitual enzoötic prevalence is in the areas that are relatively lower, damper and richer than the surrounding country, where the vegetation is luxuriant and the surface of the ground moist. Thus it is a disease of low meadows, basins, river bottoms, drying marshes or ponds, and the Boers have been in the habit of protecting their horses by sending them to high, dry tablelands, from the first appearance of the disease until the first frost. Yet elevation in itself is no protection, thus Johannisburg, 6000 feet above the sea, is habitually ravaged and Rhodesia loses 90 per cent. annually. A humid atmosphere, mist, or rain with a high temperature are directly connected with the outbreaks.

Animals Susceptible. Horses take the disease in its most fatal form. Mules suffer like horses, while in asses and guaggas the malady is relatively somewhat more benign, and the virus after having passed through the ass, has lost part of its potency. It does not attack cattle nor goats. (Theiler. See Heart water.)

Causes. The affection has been traced to the green forage, grown in damp, hot seasons on the rich moist bottom lands, in basins, gullies, etc., and which has been consumed while damp with night dews or fog. Few suffer that are only turned out to pasture after the sun has dried up the dew, and that are shut in the stable or kraal before sunset. Coley who witnessed a loss of 60 per cent of the stabled horses at Eshowe, Zululand, found that the deaths were among horses that had been allowed to eat their fodder wet. The Guinea or Ubaaba grass and Indian corn were cut at night and fed to the horses next day. The horses that ate this wet from the bundles were attacked, while those that had it only after it had been opened and dried in the sun escaped.

Race horses that receive no green fodder very rarely suffer. Horses that are corralled (in kraal) at night escape.

The hot season is the season of greatest prevalence, the disease beginning in November and proving especially fatal from the end of December to the first of March. It appears in a modified form until May when the first frosts appear.

Though the disease can be fatally inoculated by transferring the blood from one horse to another, it is the rarest possible occurrence to have it propagated in this way. It can be absolutely prevented therefore by attention to the diet.

The alleged cause of *horse sickness* is an ultra-microscopic organism which enters the system with the moist, dewy food. Edington, who discovered a cryptogam, has found it in the blood in all his necropsies of horses dying of horse sickness. Why this should be no longer infecting when dried does not clearly appear. It has been alleged that the disease has gradually extended to the higher grounds which were formerly free from it, and the introduction of diseased or infected horses has been advanced as the cause, but in the unfenced state of the veldt and the former abundance of wild animals this should have ensured such extension long ago, if it is really a permanent one. The deadly prevalence of the malady in particular areas in given years, and its entire absence from such localties in others may explain the instances of apparent extension. The dryness and cold of winter is the factor which usually extinguishes the poison in a given district. We have as yet no absolute proof of a progressive acclimatisation of the germ in a colder and drier region. Wittshire observes that it will prevail on one side of a narrow river, while the other at an equal elevation is practically free from it. There is no mention of shade which might have explained such a difference in the growth of microbes on the right and left banks.

Such limitations, together with the activity of the infection in damp seasons, and during damp hours of the day, and its inactivity in dry air and vegetation, would strongly suggest a microbe which is conveyed in the body of some invertebrate, but this appears to be nonessential because Edington has cultivated his virus in vitro and inoculated its products on horses so as to secure immunity.

Debility doubtless renders an animal more susceptible, yet the disease usually kills nearly all horses attacked, excepting such as have been immunized. This is the same for casual and inoculated cases. When, however, the virulence has been lessened by culture or by passing through the body of an unsuitable animal the results are very uncertain; some horses it will kill, while in others it produces a slight and harmless fever.

Forms. Two leading forms of the disease are known: 1st, the *lung sickness* (*Dunpaardzeickte*), and 2d, the *head sickness* (*Dikkopzeickte*). A variety of this last is *blue tongue* (Blautong), which has been confounded with glossanthrax.

Symptoms. The *lung sickness* may appear as a fulminant affection following on the usual *incubation* of about eight days. Suddenly, in the midst of apparently full health and vigor, the breathing becomes accelerated and dyspnœic; this encreases for about an hour, then the patient staggers, falls, ejects a mass of white froth from mouth and nostrils and dies. When death is more delayed, there may be noted a rigor, and in the evening a rise of temperature to 103° F., lowering a little next morning and rising again toward night, yet making an encrease day by day until near the end, when it becomes sub-normal. Death in such cases occurs on the third or fourth day, preceded by great prostration, hurried, labored breathing, dark red or cyanosed mucosæ, loud rattling over the large bronchia or lower end of the trachea, coughing and dropping of a serous fluid from the nose, or accumulation of white froth around nostrils and mouth. The froth soon condenses in part into a straw-colored liquid, which collects in considerable quantity. The abundance of froth blocking the air passages produces death by suffocation.

In the *head œdema*, the muzzle, lips, head and neck become the seat of excessive exudation, the swelling of the face drawing back the lips so as to expose the teeth of the lower jaw. The skin is rendered tense and exudes the straw-colored serum, as do also the buccal and pituitary mucosæ.

In *blue tongue* the exudate is concentrated in the lingual organ, which swells to an enormous size, forcing the jaws and lips apart, and hanging out as a dark blue, cold mass. A foul, liquid mixture of saliva and exudate drivels from the mouth. The pressure on the larynx may cause marked stertor, advancing to asphyxia.

While these three types may seem to be distinct and uncompli-

cated in some cases, more commonly the exudation appears to a slight extent in all three situations, and it is only the predominance of the symptoms in one particular part that assigns the attack to one type rather than another. All are very fatal, but the lung sickness is preëminently so, very few surviving.

Lesions. There is usually a mass of white froth around the mouth and nostrils. The serous exudate coagulates readily in the presence of minute traces of blood, and forms a solid mass of clot when heated. A yellow gelatinoid exudate is found in streaks or patches, subcutaneously and between the muscles, but especially along the jugular furrow. In the *head sickness* the whole subcutaneous and intermuscular tissue in the head and neck are infiltrated, and the straw-colored liquid escapes abundantly when the part is scarified. The same is true of the tongue, which is stained throughout with blood that has gravitated into it.

The pleuræ contain an abundant exudate more or less deeply stained with blood. The same is true of the pericardium. In the latter Edington has found 140 fluid ounces. On the surface of the lungs and pericardium are extensive yellowish exudates. They are covered with petechiæ mostly small or punctiform. If the horse has stood at rest throughout the illness the lungs seem pale, yellowish, yet swollen and indisposed to collapse. The interlobular tissue especially is infiltrated with serum so that toward the free margin the lobules may be separated by intervals of half an inch in breadth as in lung plague of cattle. In horses that have been worked during the illness the whole organ is congested and firm, resembling the condition of croupous pneumonia. The trachea and bronchia show dark congestion of the mucosæ and a mixture of froth and serous exudate. The large blood vessels contain diffluent blood clots of an intensely dark color. In the vascular furrows of the heart and along the large vessels are yellow exudates or blood extravasations. The muscular tissue of the heart appears normal or rather pale, and under the microscope the striæ are found to be obscured by cloudy swelling, and minute blood extravasations and hæmatin are met with. The endocardium is cloudy, with blood extravasations, and exceptionally ante-mortem clots are found. The spleen is usually swollen, very dark, blood gorged and covered with petechiæ. On section there are found extensive extravasations, with masses of blood pigment and crystals. The kidneys are enlarged, the capsule

easily detached, the epithelium of the glomeruli and convoluted tubes swollen and their nuclei multiplied. A gelatinoid exudate is usually present in the renal pelvis. Congestions have been found in the right gastric *cul-de-sac* and less frequently in the intestines. Exudations have also been found in the cerebro-spinal nervous system, the laryngeal mucosa and the conjunctiva. The latter is usually cyanosed.

Prevention. The first consideration is to keep work horses indoors or in a kraal during the summer or sickly season. Here they must be fed on dry hay and grain only, grass being strictly withheld. If it becomes absolutely necessary to feed green fodder of any kind it should not be cut until all dew or rain has completely dried off in the heat of the sun, and if kept over night should be kept under cover and again dried before feeding. When taken out to work the animal should wear a check rein or muzzle so that he cannot by any chance reach the green vegetation. This rule must be most strictly adhered to at night or during damp weather.

For horses turned to pasture a fair amount of protection may be secured by shutting them in a stable or kraal before sundown, and until the vegetation has been thoroughly dried by the sun the next morning.

Immunization. A horse that has recovered from the *sickness* has been long held to be immune and will bring from six to ten times its former price. As any disease is liable to be called the sickness, this enhanced value is too often insubstantial. Wiltshire even says that all "salted" horses eventually die of *horse sickness* if allowed to live long enough. Be this as it may Edington appears to have established a reasonable measure of immunity by his protective inoculations. He takes a recovered ('salted') animal and reinoculates it at intervals with encreased doses of virulent blood. After the last of these inoculations the subject is allowed to rest for a long period of time, and is then reinoculated with a small dose of virulent blood. A definite amount of this horse's virulent blood is mixed with 50 cc. of serum and injected subcutaneously; some days later 30 cc. of the same serum with the same dose of blood is injected; at a later date the procedure is repeated, with a reduced dose of serum, and fourteen days later pure virulent blood is injected." The result was satisfactory.

Koch, experimenting later, found a great difference in the virulence of the blood of successive sick animals, so that with each new animal furnishing the virus the dose must be carefully determined by experiment before it can be safely used for immunization. The following gives, not a safe dose for every case but an approximation to the average doses, to be tested in the case of each animal furnishing the virulent blood:

Inoculation	*Virulent Blood*	*Interval*	*Serum of Immune Horse*	*Interval*
1.	0.01cc.	4 Days.	100cc.	12 Days.
2.	0.05cc.	4 Days.	50cc.	12 Days.
3.	0.2 cc.	4 Days.	50cc	12 Days.
4.	0.5 cc.	12 Days.		
5.	1.0 cc.	12 Days.		
6.	2.0 cc.	12 Days.		
7.	5 0 cc.			

The virulent blood is drawn by cannula from the jugular of a pronounced case of the disease. It is defibrinated, strained through wet, sterilized muslin, then mixed with an equal amount each of sterile glycerine and sterilized water having an admixture of 1 part of carbolic acid to every 1000, and finally placed in bottles, well filled, stoppered and preserved in an ice chest, in the dark for 2 or 3 weeks. The globules are dissolved and their solids should be separated by a second filtration through wet sterilized muslin. The liquid is preserved in sterilized bottles, well filled, and stoppered and kept in an ice chest. For inoculation the required dose is diluted in enough sterilized sodium chloride solution (0.85 per cent.) to make 5cc. The injection is made beneath the skin of the neck high enough up to allow of the injection of the immunizing serum below it, 4 days later.

To prepare the immunizing serum a recovered horse is given intravenously, 2 quarts of the warm, fresh, defibrinated blood of an animal in the last stages of horsesickness, the blood having been first passed through a sterilized wet muslin filter. Four such injections are given at intervals of not less than 14 days. Fourteen days after the last injection 4 or 5 quarts may be drawn and used for immunizing serum, and the same amount on four successive occasions at intervals of 7 days. After some months rest the horse may be again fortified and its serum used anew.

DOURINE.

Synonyms. Definition : Contagious disease of breeding solipeds, with special lesions in generative and nervous system, and skin, and caused by a trypanosoma. Susceptible animals : horse, dog, rabbit, rat, mouse, ass. History : in Europe and America. Causes : contagion ; coition ; microbiology ; trypanosoma equiperdum. Its successive stages : 1. granules and refrangent spherules ; 2. chromatin bodies with two prolongations ; 3. fusiform body with nucleus and neucleolus and two flagella ; 4. pyriform bodies with flagella ; 5. fusiform body with nucleus, nucleolus and undulating membrane : in blood, sperm, milk, vaginal mucus, sores ; disappears from blood in intermissions and rapidly after death. Lesions : phlegmons of generative organs, papules, vesicles, mottling, swollen inguinal glands, penis, testicles, caseation or atrophy, thickened lymphatics, nervous lesions, friable bones, arthritis, liver, spleen, kidneys, lungs, lack of red globules, anæmia, muscular atrophy ; in mare, in dog. Symptoms : *Horse:* incubation 11 days, variable ; preputial, scrotal, sub-abdominal swelling, catarrh of genital mucosa, vesicles, sores, swollen penis, semi-erections ; paraphymosis, slight fever, tender loins, frequent micturition, paresis, swollen joints, tendons, emaciation, marked anæmia, apathy, dementia, nasal ulcers, submaxillary swellings. *Mare :* vulvar swelling, distortion, leucorrhœa, eruption, spots of depigmentatior, erection of clitoris, urine in driblets often, inguinal swellings, sterility or abortion, lameness, trembling, great anæmia and emaciation. Diagnosis : from urticaria : from glanders generative lesions, no response to mallein, paresis : from chronic paraplegia, by its coition cause, and in horses only ; from vesicular exanthema by gravity and nervous phenomena. Prognosis : Mortality 70 per cent. and upwards. Symptoms in ass slight : in dog severe and fatal ; in rabbits severe. Treatment : prevent copulation, castration, local antiseptics early. Prevention : prevent copulation of infected and suspected animals, castrate, import only on certificate and on long quarantine.

Synonyms. Venereal Disease of Solipeds ; Equine Syphilis ; Maladie du Coit ; Chancrous Epizootic ; Breeding Paralysis ; Epizootic Paraplegia.

Definition. A contagious remittent affection of solipeds, transmitted by copulation, and attended by specific lesions in the generative organs and nervous system, such as local venereal swellings, chancrous ulcers and cicatrices, dementia and paralysis.

Susceptibility. As occurring casually the disease is essentially an equine one, yet the following species are susceptible to experimental inoculation in the order named : dogs, rabbits, rats,

mice, asses. While the horse shows the greatest susceptibility, the ass is comparatively very resistant to the poison.

History. The malady has probably long prevailed in the east, yet it was first clearly distinguished in 1796 when described by Ammon as prevailing in the royal stud at Trakehnen in Northern Prussia. We have later descriptions of the disease in the same locality in 1801 (Hertwig), and 1807 (Ammon). It was found in Bromberg in 1817 to 1820 (Waltersdorf), also in Hannover (Haveman), in Austria and Bohemia in 1821–8 (Fischer), in Styria in 1821, in Switzerland in 1830, in France in 1830–2 (Lautour), in Silesia and Pomerania in 1833–40 (Fischer), in Italy in 1836, in Russia in 1843, (Fischer), in Silesia and Poland in 1830-40 (Freidberger and Frohner), in Algiers in 1847–55 and in Syria and Asia generally and perennially (Daumas and Signol).

It appeared at Wapella, Ill., in 1882, the first affected animal being a brown stallion that had been imported from France and which bore on his neck a brand like the letter D. In this locality it extended to a considerable number of breeding mares and stallions, and having been recognized by Dr. W. L. Williams, was largely stamped out by a rigid quarantine of diseased and exposed animals. Some exposed animals had, however, left the district, and isolated centres of infection have been since found in Nebraska and elsewhere in the United States and N. W. Territories.

It is not known to have invaded Belgium, Scandinavia, England, South America nor Australasia.

All indications point to Asia or North Africa as the primal home of the disease, as they still prove its perennial one.

Causes. The disease is transmitted by contagion and almost exclusively in the act of coition. Hayne has seen the affection in geldings, and Haxthausen in mares that had never been served. Schneider, Buffard, Nocard, Blaise and others have transmitted it freely by inoculation of blood and nervous matter, so that the possibility of infection through other channels than the generative organs must be admitted. But such irregular means of casual infection are so rare, and the probability of transmission of the virus from a non-breeding animal is so remote that in the practical measures of sanitary police, the breeding horses alone need be taken into account.

The extra vascularity of the male and female generative organs

at the period of coition undoubtedly favors infection, as a latent or apparently recovered case will relapse under its frequent repetition. The abrasion of the epithelial surface also forms infection atria and favors new multiple points of infection.

The disease has occurred congenitally (Rodolff, Jessen). It has been claimed that the higher bred horses with lighter frames are the more susceptible (Fischer), but this is probably a delusion, the disease having been often introduced by the Barb and Arabian and propagated among their grade descendants. The heavy Percheron shows a very ready susceptibility and a virulent and fatal form of the affection.

Microbiology. That the affection was due to a microbe was clearly shown by its absence from every part of the world into which infected horses had not been brought. The secluded countries, Belgium, England, Scandinavia, that breed their own horses, the distant Australia, New Zealand and South America remained free in face of the constant presence of the infection in different parts of Central and Southern Europe, in Africa and Asia. The horses of America and South Africa showed a ready susceptibility to the virus brought by infected horses, and rigorous sanitary police control speedily cleared a district of the trouble.

Thanhoffer found in the blood, vaginal mucous, testicle, semen, spinal fluid, myelon, and roots of the dorsal and lumbar nerves cocci, especially streptococci and less constantly bacilli, to which he attributed the malady. More recently Schneider and Buffard have demonstrated that an infusorian, the *trypanosoma of dourine*, is the essential pathogenic agent.

The *Trypanosoma Equiperdum* varies greatly in form at different stages of its growth or in different media. In the exudate of the slight early tumefaction, without as yet other symptoms, it is found as minute granules in groups, as larger spherical very refrangent bodies like very large cocci, each having a strongly staining nucleus, and as larger bodies in which a delicate membranous covering encloses one, two or three masses of chromatin and extends to form one or more points (club-shaped or fusiform). Each chromatin mass had a nucleolus on its outer surface or slightly apart from it. Twenty-four hours later there may be added: First, short, thick, chromatin bodies, with two slightly undulating, pointed, membranous prolongations. Second, more delicate, fusiform bodies, each with one chromatin nucleus, a de-

tached nucleolus, and the membrane prolonged into two actively moving flagella. Third, larger pyriform bodies with chromatin nuclei and nucleoli and the membrane prolonged into one or several flagella. Fourth, fusiform bodies, thick or delicate, each having a chromatin nucleus and nucleolus, and arranged singly or in groups of two, four, six or more, united together at one end and diverging at the other to form a stellate mass. These last, 20 to 30μ long by 1.5 to 2μ broad, may perhaps be the adult form of the parasite from which the small granular or spore forms found in the most recent lesions are derived. The fusiform outline, the deep staining central mass, with its adjacent nucleolus, and the pointed or flagellate membranous prolongations, more or less motile or undulating, are characteristic features.

In these morphology and evolutionary forms the trypanosoma of dourine has not been shown to differ from that of surra, nor the nagana or Tsétsé disease, the granule form, the spherical, the club-shaped or pyriform, the fusiform with more or less stellate grouping, are characteristic of all (Lydia Rabinowitsch, Kempner, Schneider, Buffard). The distinction is found in the pathogenesis of the two diseases.

With active cutaneous or mucous lesions, the parasite is usually found abundantly in the blood, sperm, milk, vaginal secretions, and the erosions of the vaginal mucosa or penis. During intermissions, however, and in the absence of local lesions, examination of the blood may fail to detect it, yet its inoculation on a dog will usually produce the affection. It disappears from the blood and tissues with great rapidity after death, so that, to prove successful, inoculations should be made before death or immediately after. They are ineffective after 48 hours.

Schneider and Buffard, Nocard and others found the tympanosoma in the blood and exudates of horses, asses and dogs, suffering from dourine, and failed to find it, in the same localities, in animals of the same species which were free from dourine. The infected blood, preserved for 24 hours in sealed glass tubes, and then inoculated on two dogs produced characteristic dourine, with the extensive production of trypanosoma in the blood, the destruction of blood globules, and the pathognomonic local lesions. Inoculations of two other dogs, with the same material, at the end of 48 hours produced a slight transient hyperthermia only,

without local lesions or propagation of the parasite in the blood. The same blood inoculated after 15 days produced neither local lesion nor fever.

Lesions. Horse. In the early stages are found a phlegmonous or œdematous swelling of the *sheath*, *scrotum*, *penis* and *inguinal glands* and a yellowish liquid effusion into the scrotal cavity. The skin covering these parts may show a papular or vesicular eruption or if this has passed, a mottling with white spots shows where the lesions have been. Later still the *inguinal glands* are shrunken and have undergone fibroid degeneration and induration and the testicles, swollen or shrunken, contain centres of caseation. The connective tissue of the epididymus and cord is the seat of a gelatinoid exudation. The *walls of the scrotum* may be greatly thickened and the seat of abscess or of caseous degenerated hyperplasia. In advanced cases the *testicles* are usually abnormally small even if the scrotal mass is enormously distended. The *sheath* and *penis* may be the seat of more or less numerous ulcers, and swellings, contractions and distortions of the latter organ are not uncommon. The penis may, however, retain its normal dimensions. The walls of the *lymphatics* in the inguinal region may be the seat of hyperplasia, the thickening causing them to stand out like cords as in glanders. In the advanced stages the *muscles*, especially those of the hind limbs, become pale and atrophied.

The *nerve centres* undergo profound changes which have been studied by Thanhoffer. The *pia mater* in the affected parts of the spinal cord is the seat of active congestion and thickening. The central canal of the cord is dilated (syringomyelia) more at one point than another, contains more than the normal amount of liquid, and the neuroglia around it is thickened and fibrous (sclerosed). The *substance of the cord*, both white and gray, shows congestion, blood staining, at points foci of softening, and at others induration (hyperplasia of the neuroglia). The *nerve cells* are modified in various ways, some being granular, some discolored by fine granular pigment, some having enlarged and multiplied nuclei, and some show vacuoles. The *nerve filaments* often show a granular degeneration extending from the nerve cell into the axis cylinder, and the latter is liable to be varicose or uneven in size. In the affected portion of the cord leucocytes

are numerous and hyperplasia is often present, The neuroglia especially tends to encrease, and apart from the foci of softening tends to give a special firmness to the substance. At intervals, in the perivascular spaces, may often be found minute (microscopic or macroscopic) blood clots. The subarachnoid and subdural fluid is encreased and may be pinkish. At the roots of spinal nerves, especially in the dorsal and lumbar regions, a gelatinoid exudate may invest the nerve, distending the connective tissue beneath the neurilemma and even occupying the interval between the nerve filaments. Sometimes large corpuscular bodies are found between the nerve fibres.

The *cerebral meninges*, especially the *pia mater* are congested and opaque. Foci of softening are by no means uncommon and the cerebral ventricles contain an abnormal quantity of fluid.

The *bony tissue* generally has lost its consistency and the medullary matter may be unduly reddened. The large joints contain an excess of synovia somewhat pinkish in color, and the ligaments of the hip joints are often congested, thickened and softened. The articular cartilage may even show patches of blood staining.

The *intestines* are usually nearly empty, soft, pale and flaccid, and Ruthe has in one case observed rounded ulcers on the mucosa. Fibroid thickening on the peritoneal surface may indicate a previous exudate.

The *mesentery* is thickened, with infiltration and has a yellowish discoloration and the *mesenteric glands* are usually enlarged, softened and friable, though sometimes firm and contracted. The *lymph glands* adjoining the generative organs are often swollen, pigmented and studded with foci of caseation, varying in size from a pea upward.

The *liver* is softened and congested or fatty. The *spleen* is small.

The *kidneys* are usually large, but flaccid, pale and bleached.

The *thoracic organs* may show little change, though hypostatic inflammation and foci of caseation or suppuration may be present.

The *blood* is pale and watery and forms a loose, pale, diffluent clot, while there is an extraordinary diminution of red globules and a relatively great encrease of leucocytes eosinophiles.

In advanced stages the *muscles* are pale, anæmic and shrunken especially those of the hind limbs.

Mare. In the mare in addition to the lesions of the internal organs and blood, the following may be noted in connection with the generative system. Phlegmonous or œdematous swellings, sorès or ulcers on the lips of the vulva, and on the vulvar and vaginal mucosæ. The parts become variously distorted, and the vulva, habitually open may expose the swollen and ulcerated clitoris. A crop of papules or vesicles, running into sores or ulcers may appear on the urethral orifice, the vulva and adjacent skin, and even though overlooked, their seats are marked later by loss of cutaneous pigment and the formation of small white spots. The mammæ are sometimes inflamed, œdematous and tender, with suppurative or necrotic foci and the adjacent lymph glands are enlarged by infiltration or contracted by sclerosis.

Dog. These resemble those in horses. In the bitch genito-urinary congestion, inflammation and catarrh, subcutaneous infiltration under the belly and inside the thighs, swelling of the inguinal lymph glands, emaciation, and pallor and atrophy of the voluntary muscles are marked features.

Symptoms. Horse. Incubation may last from one to eight weeks (Maresch), but is usually 11 days from the infecting service. It may be abridged by a special susceptibility, and by repeated infections and hence the more acute cases are especially seen in the male in daily service. In some such subjects local genital infiltrations are speedily followed by paralysis or vertigo which cuts off the patient in a few days.

More commonly the malady is *chronic* though varying in different countries, epizoötics, or even in particular cases.

The first *Symptom* is slight swelling of the anterior border and raphe of the sheath, which gradually extends backward to the scrotum and inguinal region and forward on the abdomen it may be as far as the brisket. This may be hot and painful, but is usually œdematous, cool and painless. The infiltration affects the end of the penis, the meatus may be red and angry with a slight muco-purulent discharge, and red spots, vesicles and ulcers may stand out clearly on the pigmented surface. Williams never saw such eruptions in the American cases and doubts their existence except as the result of injuries. There are frequent semi-erections and service is still possible. Later the engorgement extends to the specially pendent testicles, inguinal glands and others

adjacent. Paraphymosis is occasionally seen or more commonly, the penis hangs out of the sheath soft and flaccid and erection seems impossible. The local swelling may become excessive, pressing the testicles up against the inguinal ring, or suppuration and extensive abscess may follow. Appetite is retained and the temperature remains moderate (100° to 102° F.).

The local swellings may almost completely subside, except a slight tumefaction of the end of the penis, hence some (Fischer) have held that the cutaneous lesions are the primary ones, yet the start of infection at the generative organs and the fact that a stallion often infects a number of mares before there is any suspicion of his own infirmity is evidence enough of the genital seat of the earliest lesions.

The cutaneous lesions, which are essentially secondary, but highly characteristic, appear from forty to sixty days after the infection (Schneider and Buffard). They are from a quarter of a dollar and upward in size (in some old horses much smaller), and rise abruptly from the healthy skin, becoming the more marked that the hairs upon them stand erect. These may arise suddenly and subside again in one day, or give out a serous exudate which mats the hairs into a tuft. They may, however, last four or five days. These are more patent after a full drink of water, or after sprinkling or sponging with water. Even when they have been overlooked these results may often be seen later on colored skins, in the presence of many circular white spots entirely devoid of pigment. The most common seat of these cutaneous lesions is the hind parts (anus, tail, croup, quarters), but they may appear on the sides, neck, shoulder or thighs.

Tenderness of the loins may now be shown, with frequent, painful micturition, but the penis becomes more and more paretic, so that coition becomes encreasingly difficult or impossible. Paresis also shows in the hind limbs, the animal remains recumbent a great part of the time, rises with difficulty, starts suddenly forward at the fetlock, and drags the toe on the ground in walking. Swelling of the joints and tendinous sheaths, with attendant lameness, is not uncommon. The appetite remains good, yet emaciation and weakness make marked progress.

The advanced stages are characterised by marked anæmia, paraplegia and dementia. The visible mucosæ are pale, emaciation

advanced, the patient stubs his toes and sways in walking, and finally drops unable to rise ; or he stands with hind legs straddled and semi-bent, and largely oblivious of all around him. He may be too stupid to eat unless the food is placed in his mouth, and yet the neigh of another horse or a mare may draw forth a feeble retort, as if the deranged generic instincts remained. Swelling, or even abscess of the lymph glands, axillary, submaxillary or inguinal, is usually present, and mucopurulent discharge with ulceration of the nose or eyes is not uncommon. The joints crack when moved and fractures are common.

The duration of the disease is two or three months, the progress being more rapid in winter.

Mare. Vulvar swelling eight or nine days after service, with ejection of urine in jets, contraction of the vulva, redness, swelling and erection of the clitoris, and a mucopurulent discharge are marked symptoms. European observers note nodules, vesicles and ulcers on the mucosa and adjacent skin, but these were not observed in the American cases (Williams). Yet later they showed the characteristic white spots. Switching of the tail, stamping of the hind feet and painful straining to urinate are followed in certain cases by ejection of the urine forcibly in jets or in small dribblets. The catarrhal discharge is at first watery, but later becomes viscid, sticky and white, yellow or grayish, or even red. It mats together the hair of the tail and thighs, and putrifies, exhaling a repulsive odor. The swelling involves the space between the thighs and often implicates the mammary glands and even the floor of the abdomen. The inguinal glands are often involved, and hyperplasias with degenerations and even abscesses may appear in this region. As in the stallion the local lesions may have periods of advance and subsidence, and in favorable cases, that are not again served, there may seem to be a temporary recovery. The mare, however, remains infecting, and if served the local disease is at once roused into activity. The button-like skin eruption appears on the quarter as already noticed in the horse and in grave cases may caseate or suppurate and burst, forming an indolent and intractable sore. The dark skin of the vulva and perineum becomes marked by white spots or by irregular patches caused by the confluence of several such spots, which for ordinary breeds of horses are highly characteristic of

this disease. Many Arabian horses naturally acquire such white spots on the dark skin, and in pure Arabs and grades this appearance need not be held as evidence of dourine.

Infected mares rarely conceive, and any that do so are likely to abort before the sixth month.

The systemic symptoms, nervous, paralytic, tremulous, dyspeptic, atrophic, cerebral and cachectic follow a similar course as in the stallion.

In grave and progressive cases the lesions of the generative organs become very marked. The lips of the vulva become rigid and distorted so that it remains constantly open and the erect clitoris continually exposed. The skin of the vulva is tense, dry and shining. Lameness is shown in one or both hind limbs, knuckling over at the fetlock, shortening of the step, planting the toe first and the heel later with a jerk, lack of balance, paresis and even inability to rise.

A disposition to trembling is common to both sexes, as is also an intolerable itching of the skin which may make the animals tear the lower parts of the limbs with the teeth. In either there may be a local paralysis of a lip, an ear, an eyelid, or some other part of the body. In both sexes the disease tends to extreme anæmia, debility and emaciation, and to infective internal inflammations (lungs, bronchia) or septic or purulent infections.

Diagnosis. From *urticaria* this disease is to be distinguished by the absence of lesions of the generative organs in the former, by its association with change of food and digestive disorder and by the absence of all evidence of contagion.

From *glanders* it is to be distinguished in the same way by its casual transmission by sexual connection only, by its restriction to breeding animals, and by the irresponsiveness of the victim to the mallein test. The progressive paresis and hebetude are valuable diagnostic phenomena. From *surra*, by its avoidance of non-breeding animals, its transmission by coitus, the immunity of cattle, and the absence of marked regular febrile reactions.

From *chronic paraplegia* it is distinguished by the same prevalence in breeding solipeds only, and by its mode of transmission.

The greatest difficulty is experienced with the slight and comparatively occult cases, and in some of these the history of the infection of a number of breeding animals, which have been

served by the same horse may be the one guiding point for a number of the cases. For sanitary purposes it is well to treat as suspicious animals all mares that have been served by a stallion which is in the line of infection. This suspicion would attach also to any stallion that had served a mare which had been to a diseased or suspected stallion, and to all mares that have been served by the suspected stallion after he had been to the suspected mare.

From *simple vesicular exanthema* dourine is to be distinguished by its comparatively slow progress, and by the uniformity of the lesions of the generative organs and nervous system, which are lacking in the vesicular affection.

Prognosis. A certain number of animals recover from the milder attacks which have not become complicated by the grave trophic and nervous lesions, but as in glanders, recovery from severe attacks and in the advanced stages is practically unknown. The mortality is set at 70 per cent. and upward.

Symptoms in the Ass and Mule. The disease usually remains discrete, the lesions in the generative organs or other seat of infection being the marked symptoms, and recovery the rule. Œdema of the end of the penis, obliteration of the folds around the orifice of the urethra and eversion of the urethral mucosa are the most constant features, distinct even in semi-erection. Œdema of the sheath and skin eruption may follow. Exceptionally a mule proves as susceptible as the horse, and shows the disease with the same fatal severity.

Symptoms in dog. A bitch, 15 days after vaginal injection of the infected equine blood, showed severe vaginitis and hyperthermia (100°). On the 23d day she aborted, followed by muco-purulent discharge, vulvar œdema, pallor of the mucosæ, anæmia, rapid emaciation, paresis and occasional convulsions. Blood drawn from the vulva contained the trypanosoma. She died on the 66th day. The temperature rose to 103°.

Two male dogs that lined the above infected bitch showed after 12 days, engorgement of the sheath and scrotum, muco-purulent discharge, and abundance of the trypanosoma in the blood of the affected parts. Cutaneous lesions on the loins, thighs, sides or forehead, showed erection of the hair, with infiltration, or shrivelling and bloodlessness.

Other dogs showed arthritis, corneal opacities and ulcers,

hypopion, cataract, nasal discharges, facial periostitis, dyspnœa and syncope.

Symptoms in Rabbits. Rouget found in inoculated rabbits, extensive gangrene of the skin, involving even the cranial bones, ulcerative keratitis, hypopion, panophthalmia, following on the earlier genital troubles of vaginitis, swelling and discharge.

Treatment. Some cases recover spontaneously a few may be aborted in the earliest stage of the disease, but cases that have advanced to any extent and assumed a grave character are practically hopeless.

An important element in treatment is to do away as far as possible with the cause of generative excitement since the disease is aggravated and more rapidly advanced by frequent copulation. Rodloff from a very wide experience speaks highly of castration of the stallion. Castration of the mare has not been specially advocated and the absence of marked lesions in the ovaries, may deter the veterinarian, yet whatever promises to lessen in any degree the genesic excitement is not to be despised, and the measure has besides a sanitary value for other animals.

Early local treatment is the most promising, and especially if it can be applied to exposed animals during apparent incubation. Inject the urethra, sheath, vulva, vagina and uterus with antiseptic lotions, and apply them to the external sores. Mercuric chloride 1:2000, carbolic acid 2:100, silver nitrate 1:250, calcium chloride 1:100, or chlorine water may be taken as examples. When local swellings have supervened it would be entirely appropriate to incise them freely or even to excise them and cauterize thoroughly with stronger agents.

Internal treatment by mercuric chloride 1½ to 3 grains, potassium iodide 2 to 5 drs., arsenic 7 to 14 grains, has been widely used but to little purpose. The same may be said of Rodloff's treatment by tonics and carminatives (sal ammoniac, camphor, iron, angelica, gentian, ginger and valerian).

Prevention. The one thing to be secured is the prevention of copulation with any animal that has been affected, or exposed to this disease in the past three years.

1st. Any stallion or mare once affected must be excluded from breeding for at least three years after apparent recovery.

2d. Every such animal that has been exposed by copulation

with a suspected animal must be excluded from breeding for the same period even if no active symptoms have been shown.

3d. Every affected or exposed animal, should be quarantined in the hands of his or her owner and placed on an official register and the sale, gift or loan of such an animal, or its movement to a new place where it might propagate the disease should be made a misdemeanor.

4th. In any country or district in which the disease exists all stallions and mares should be registered and none should be allowed to be used for breeding purposes without an official certificate showing that each of the animals mated is free from all suspicion of having been exposed to this infection.

5th. If any stallion or mare is imported from a country in which dourine exists it should be accompanied by an official certificate showing that it has not in the past three years been exposed to the possibility of infection with dourine.

6th. In the absence of such certificate the imported animal (capable of breeding) should be kept in strict quarantine for the period of three years.

7th. A much more radical measure, which may be made to supersede all of the above, would be to castrate every soliped (stallion or mare) which has suffered from the disease in the past three years, and every such animal that has by coition been exposed during that length of time to even a remote opportunity for contagion. This would embrace all apparently sound mares that had been served by a stallion which had shown slight symptoms of the disease, or by a stallion which had not himself shown such symptoms, but which had served a mare that had shown such symptoms; or one that had served a mare that had not shown such symptoms, but that had been previously served by a stallion which had shown such symptoms, or that previously covered a mare that had shown such symptoms. In such cases the State might well afford to indemnify the owner for any reduction in value of the castrated stallion from that borne by the animal as a prospective breeder, the breeding for which indemnity is sought being understood to count only from a date of three years after the sanitary castration. A mare once attacked should be remorselessly castrated or killed. A perfect recovery in a horse can be better attested; that of a mare is always uncertain, and most

secondary outbreaks after the apparent extinction of the poison have come from breeding mares that have apparently recovered, or that after exposure have shown no appreciable symptoms.

When a State is so lost to all sanitary considerations as to abandon an affection of this kind to take its course, the owners of stallions and mares cannot be too careful to avoid the exposure of their valuable breeders to the risk of infection. Each mare brought for service should be admitted only when accompanied by a certificate showing all previous services in the last three years, with the identification of the stallion, and this irrespective of whether the service has been fruitful or not. In such certificate the owner of the mare should bind himself to make good all damage or loss that may accrue from his failure to set forth in the certificate every such service and every symptom of illness affecting the generative organs from which the mare has suffered in the three years antecedent.

The owner of the stallion should give a similar guarantee that the horse has in the past three years served no mare that was in any way open to suspicion, and that the animal has not suffered from any affection of the generative organs which had any of the characteristics of dourine.

No mare should be served which shows swelling, nodules, distortion, or gaping vulva, a muco-purulent discharge, or too frequent or two prolonged *heats*.

No stallion in such locality should be allowed to serve which shows pasty swelling of the sheath, swelling, shrinking or distortion of the penis, red, angry, tender meatus urinarius, or a muco-purulent discharge.

Unless in case of Arabian horses the appearance of white spots on the dark ground of the sheath, penis, vulva, or perineum, should be ground for debarring from service until an absolutely stainless record covering a number of years has been shown.

MAL DE CADERAS.

Synomyms. Quebra Bunda, Broken Buttock; Disease of the Rump; Infectious Paraplegia; Paraplegic Trypanosomiasis.

Definition. An acute trypanosomiasis of solipeds characterized by fever, rapid emaciation, extreme anæmia, debility, dropsies, advancing paraplegia, and almost always a fatal result.

Historic Notes. Geographical distribution. Long familiar in equatorial America, it was recognized in 1830 in the island of Maraja as a sequel of the ruthless destruction of 60,000 horses and the exposure of the carcasses without burial (Calendrini). As a result the horses of the island were virtually exterminated. At first the affection proved fatal in a few hours, but after a year's prevalence, when the more susceptible animals had been killed off, death was usually deferred as far as the 8th or 15th day, and now it is from two or five months. A septic complication doubtless existed at first. At the present time the malady prevails from Corrientes in the Argentine Republic on the south, to Bolivia on the north, following especially the damp river bottoms and other wet grounds. The whole basin of the Amazon suffers, and especially Parù, Maranhò, Ccarù, Goya, Matto-Grosso, and above all Maraja. In Argentina, Paraguay, Uruguay and Bolivia the marshy regions are severely ravaged.

Animals Susceptible. Of farm animals the *horse* and *dog*, suffer most severely, the *ass* and *mule* much less, while *cattle*, *sheep*, *goats* and *swine* seem to resist casual infection, and give but a mild reaction when inoculated. The *cat* is less susceptible than the dog. The wild animals suffer about as follows: *White* and *gray mice* and *rats*, *Cabiai* (*Hydrochœrus capybara*), *monkey*, *coati*, *rabbit*, and *Guineapig*. It exterminated the cabiai in Marajo in 1830. Birds appear to be immune, though Voges claims the infection of chickens, turkeys and ducks.

Microbiology. The essential cause is the *Trypanosoma Equinum* discovered by Elmassian in 1901 and confirmed by Voges. It is 24 to 26 μ long, by 1 to 2 μ broad, with a flagellum as long as the body, extending back from the head, in the undulating membrane, for two-thirds of the body length. A distinctive feature of this trypanosoma is the lighter coloration of the

centrosome, situated posteriorly. It multiplies by longitudinal division. The microbe is found in the blood, exudates of the serosæ, synovia and urine.

Accessory Causes. As in *surra* and *nagana* the most important is the presence of blood-sucking flies, tabanus, mosquito and the mosca bravá (Stomoxys calcitrans) are especially blamed. All blood-sucking parasites are to be dreaded. Conditions favorable to the encrease of the insect bearers contribute. The vicinity of barns with exposed manure and vegetable rubbish, exposed carcasses of the dead horses and other genera, low damp localities and rainy seasons, abundance of undergrowth, damp soils abounding in organic matter, and a high temperature are to be noted. Calendrini found the greatest fatality in stables surrounded by manure and decomposing carcasses, while Voges claims for to-day a lessened mortality in *dry*, *elevated* (and presumably *cleanly*) stables, as compared with marshy pastures.

Symptoms. As in surra and nagana there are a series of attacks of hyperthermia separated by complete intermissions. The trypanosoma is found in abundance in the blood during the febrile access, and are absent during the non-febrile period. Slight or premonitory symtoms may appear for 7 to 14 days, dulness, sluggishness, hurried breathing under exertion, weakness and emaciation in spite of good appetite, generous feeding and ample rest. Hyperthermia advances slowly to 102° to 105° F. and after some days suddenly drops to the normal, to rise again in the same manner after the intermission.

One of the most constant symptoms is the loss of power of the hind limbs which sway and stagger, the femurs turning inward, as if dislocated, and on uneven ground the animal can not walk without falling. When down, he cannot rise without assistance. In the earlier experience of the malady (1835) only 8 to 16 per cent. failed to show these paraplegic symptoms, whereas in recent years 50 per cent. or more escape them.. In these cases the emaciation alone, goes on gradually encreasing until the patient appears like a living skeleton. Some retain an appearance of liveliness, yet all stand on three limbs, and change from one hind limb to the other every six or eight seconds.

The retraction of the abdomen is a marked feature, yet expulsive contraction is defective, the patient fails to put himself in

the position for urination or defecation, and there is more or less detention of urine or fæces, the latter being dry, moulded, covered with mucus and of a reddish yellow color. The sphincter ani is dilated. After a time the urine escapes in fine jets, so small that in the absence of stretching to urinate, or raising of the tail, they are easily overlooked.

The urine may be normally clear, or dark colored, albuminous or bloody. The penis hangs out of the sheath several inches farther than in health.

Appetite is sometimes impaired, or completely lost, though usually the patient eats and drinks to the last, but without proper digestion or assimilation. It does not check the advance of marasmus. Thirst often becomes excessive and in such cases, there is diuresis together with frequent and excessive rumbling of the bowels. The loins are very sensitive to pinching.

About sixty to seventy per cent. in different outbreaks show œdema, in the epigastric region it may be six inches in diameter, or it may extend from the sternum back so as to include the abdomen, sheath, or mammæ, and perineum, and even the hind limbs. Considerable serous oozing takes place from this for four or five days, after which it dries up.

In about seven to fourteen per cent. the head became œdematous and swollen, with an abundant fœtid purulent discharge from the nose and eyes, and extensive ulceration of the pituita and conjunctiva. Such cases became completely blind prior to death. In other cases extensive ulceration of the skin sets in with the formation of most repulsive sores.

The nervous symptoms assume various forms ; in some there is stupor with head resting on the ground ; in others extreme debility and paraplegia with phenomenally rapid emaciation ; in a few hemiplegia, or even delirium is shown ; in all there is a marked paresis of the digestive organs and especially impairment of peristalsis. The blood is deficient in red globules and rich in leucocytes (lymphocytes and eosinophiles.

The most constant symptoms appear to be dyspnœa under exercise, paresis of the hind limbs and intestines, genital atony, a wonderfully rapid and extreme emaciation, œdema, and a tendency to impaired nutrition or ulcerous degenerations of the pituita, conjunctiva or skin.

Lesions. The pale coloress blood is often characteristic. The normally light colored tissues (serosæ, mucosæ, adipose tissue, etc., are abnormally white or yellow. The muscles are pale and atrophied especially in the hind quarters. The intermuscular tissue often shows a gelatinoid exudation, and in the gluteal region points of hæmorrhage may be found. There is enlargement of the liver, spleen and lymph glands, and pallor of the kidneys with points of congestion. The heart is soft, flabby and on the endocardium petechiated. A slight serous effusion may be present in the chest, abdomen or pericardium.

Diagnosis must be based on the very characteristic symptoms and on the lesions, but above all on the presence of the hæmatozoon during and immediately after each access of fever. Or inoculation of a dog or Guineapig may be resorted to.

Treatment proved a continuous failure. Quinine and cacodylates by the mouth and subcutem were equally ineffective.

Prevention. Calendrini claimed excellent results from the killing and burial of the diseased, the separation of all sound horses from the places where they had been, and the thorough disinfection of all harness, utensils, wagons, etc. For a number of years his district (Soure) was, by this means, kept free from the plague. The precautions recommended under *Surra* would be equally applicable here. Above all remove all manure and rubbish heaps, or seclude them in close dark pits where they can be disinfected, apply to the skins of the animals preparations of naphthalin, tar or other agent obnoxious to the insects, and screen the stables against their invasion. Fly covers should be worn when abroad. Petroleum may be sprinkled on all stagnant water, devoid of fishes and frogs, to kill the mosquitoes. Stable vermin, caviai, dogs, water hogs and Guineapigs should be subjected to the measures for suppression. Agglutination of the hæmatozoa is determined by the serum of strongly immunized cattle, sheep and swine, but attempts to avail of this in the immunization or therapeutics of the horse have not proved satisfactory.

PROTOZOAN CATTLE FEVER. TEXAS FEVER. PALUDISM OF CATTLE.

Symptoms. Definition : protozoan, tick-borne, febrile, affection, of wild damp lands, and warm seasons, with enlarged spleen and liver and hæmolysis. Historic Notes ; Old World ; Australia ; tropical and subtropical America. Causes : contact of cattle from salubrious districts with the insalubrious or with cattle from such ; Piroplasma bigeminum ; a bovine parasite, reducing red globules by ¼ths. ; successive forms of piroplasma ; the cattle tick, boophilis annulatus, bearer of piroplasma ; demonstration of the tick agency ; toxic saliva of tick ; toxic property in blood ; question of identity of infection-bearing ticks. Lesions : putrefaction rapid, icterus, ticks, blood oozing in skin, hydræmia hæmoglobinæmia, few red globules, small petechiæ, slight serous exudates and effusions : congestion, petechiation, sloughing, perforation of gastric mucosa, congestion of intestinal mucosa, in rectum like port wine ; liver enlarged, congested, biliary radicles in acini gorged with bile ; spleen enlarged, engorged : kidneys œdematous, blood-stained ; bladder petechiated ; urine opaque or red, in convalesence watery ; womb ; fœtus. Incubation three to ten days ; delays due to hatching of ticks. Symptoms : Acute case : anamuesis ; hot season ; hyperthermia 104° to 109° F. ; hurried breathing and pulse ; anorexia ; dulness ; costiveness ; icterus ; prostration ; weakness ; delirium ; urine turbid, red ; blood hydræmic ; diarrhœa ; emaciation. Duration one to seven days. Fatal (90%) to exotic cattle ; mild in indigenous, or cool season. Mild case ; temperature 103°, anorexia, dulness, costiveness, enuresis, albuminuria, pallid mucosæ, emaciation, round protozoön in globules, ticks, oligocythemia. Differential diagnosis, from anthrax. Treatment : laxative : antiseptic ; mucilaginious food ; picking off ticks ; anti-ixodic lotion ; tick free pasture or place. Prevention : destruction of ticks ; picking ; dipping or smearing with tick killing preparation, paraffine or extradynamo oil and sulphur, danger with shipping ; dressing of all cattle at intervals during warm reason ; cultivation of tick-infected land ; exclusion of cattle for one summer and two winters ; soil cattle for three weeks in each of two tick free pens, to let ticks drop ; danger of nonimmunized cattle in infested area ; suggestions for extinction by States. Immunization : Infection of sucking calf ; infected by a few ticks : by graduated injections of piroplasma blood ; technique ; injection of blood from body of tick. Limited value of artificial tolerance. Marketing of the beef. Federal restrictions.

Synonyms. Splenic fever ; Spanish fever ; Mexican fever ; Southern cattle fever ; Australian tick fever ; Tristeza ; Red water ; Black water ; Bovine periodic fever ; Bovine yellow fever ; Maladie du bois ; Holzkrankheit ; Moor evil ; Wood-ill ; Ixodic Anæmia ; Roumanian hæmoglobinuria. Piroplasmosis.

Definition. A specific fever of cattle, enzoötic during the warm seasons in the low, malarious grounds and wooded or uncultivated districts of different countries, caused by a protozoön in the blood and red globules, which is conveyed from animal to animal by ticks, and leading to engorgement of the spleen and liver, destruction of the red globules, hæmoglobinuria, and oligocythæmia.

Historic Notes. This malady has doubtless existed from time immemorial in different malaria districts of the Old World, where the wood and moor ill is now coming to be recognized as a protozoan tick-borne disease. The malady exists in Roumania (Starcovici, Babes, Gavrilescu) Turkey (Nicolle, Adil-Bey), Sardinia (San Felici, Loi), Southern France (Lignieres), Italy (Celli, Santori), Algiers, Tunis (Lignieres), Finland (Krogins, Von Hollens), West Indies, Mexico, Nicaragua, United States of Columbia, South America as far south as the Argentine Republic, German East Africa (Koch), Transvaal (Theiler), S. Australia (Pound). In Australia imported European cattle found the infection waiting for them in the uncultivated bottoms. In America it doubtless prevailed on the seaboard and islands of the Gulf of Mexico from the time of the importation of Spanish cattle, but for the first definite account of it we are indebted to Dr. James Pease, who records the widespread destruction of the native herds in Lancaster Co., Penn., in connection with the introduction of cattle from the south. None of the southern cattle died, but wherever they traveled, the native stock perished all but universally. Other droves from South Carolina were equally destructive to all cattle along their track. The recorded symptoms of anorexia, great weakness, often inability to stand, trembling, groaning, bloody urine, bleeding from the nose, costiveness, congested kidneys, and decomposed, incoagulable blood serve to identify the disease.

Later, whenever southern cattle were moved north, the disease followed their trail. Florida cattle left infection along their route until they reached the border of Virginia, where it usually ceased. When taken from the Georgia mountains to the lowlands, they died without infecting the native stock, and, when such native stock of the lowlands were moved to the hills or the north, they conveyed the fever to the stock among which they came, though themselves well and improving all the time

(Wilkinson). Similar experiences were had in all the middle states up to the war of 1861, but, in too many cases, the real source of infection was overlooked. It was observed that the disease was confined to the vicinity of the main highways and drove roads running north, and spared the lands lying somewhat back of these routes. Attention was drawn to the Texas cattle in 1853 when a herd of 450 which had wintered in Jasper Co., Mo. moved north passing through Vernon Co. in June, and causing losses of 50 to 90 per cent. of the native cattle along their course, and only along that line. Such invasions occurred yearly, and in 1858 $200,000 worth of native cattle perished from this cause in Vernon Co. alone (A. Badger). During the war (1861-64) the cattle, in Texas especially, encreased without meeting with an adequate market, and, on the opening of the trade once more, they were sent north in large numbers carrying infection with them. When Forts Smith and Gibson had been occupied by the Union soldiers, the southern cattle poured in along the military road and the Kansas farmers along this route suffered severe losses, as well as those to whom the southern cattle were finally distributed (Bray).

Causes. Up to 1889 the true cause of Texas fever was unknown. It was well established that cattle brought from the lowlands of the southern states, during the warm season, though themselves in apparently the best of health, proved deadly to northern cattle with which they came in contact, to those that followed them in the same pasture during the same warm season, and even in many cases to the mountain cattle of the south. In the same way northern cattle, removed to the infected regions in the south, contracted the fever and almost all perished. This was equally true of cattle taken from the northern states to Jamaica or other islands in the Gulf. In the winter season, after the first severe frosts of autumn and before the last keen frosts of spring, the southern cattle could be safely introduced into the northern states and on this a *modus vivendi*, for a trade in southern cattle in the winter only, was based.

Microbiology. Piroplasma bigeminum: Apiosoma bigeminum. (Apios pear, geminus twin). In 1888 Starcovici discovered pyriform organisms (*Babesia bigeminum*) in the red blood-globules of Roumanian cattle suffering from hæmoglobinuria, and Babes,

after a study of the organisms, named them *Hæmatococcus*. The following year Theobald Smith found them in the Texas fever blood, and recognized them as protozoa (*Pirosoma bigeminum*). Wadoleck proposed *Apiosoma*, Bonome *Amœbosporidia*, and Patton, *Piroplasma*. The latter pointed out that *Pirosoma* was already in use for another organism. Th. Smith's discovery identified Texas fever with the Roumanian hæmoglobinuria, and stimulated the Bureau of Animal Industry to an extended research which, in the main, elucidated the true nature of the disease. In a long series of experiments the observers produced the disease in healthy susceptible cattle, by injecting them, in the warm season with the blood of sick animals, and as constantly failed in the experimental inoculation of similar blood on non-bovine animals such as sheep, rabbits, Guineapigs and pigeons. In Australia, Pound had violent fever in two injected sheep but no pyroplasma, and their blood injected on the ox, had no affect. In none of these latter were the blood-globules invaded by the parasite, nor were the corpuscles lessened in number. In the affected cattle, the red cells were reduced from the normal 7,000,000 per cubic mm. to 1,800,000 and even lower in some cases.

The *Piroplasma Bigeminum* passes through a series of forms in the blood. Theobald Smith found in the red globule and attached to its margin a *pale round body* 0.5μ in diameter, and staining freely in *alkaline methylene blue* and other basic anilin dyes and in *hæmatoxylin*, but not in acid coloring fluids. These he found in the red globules in acute cases, often in company with the pear-shaped bodies, and usually in the absence of the piriform bodies in chronic cases, in non-fatal relapses, in cases occurring in cooler weather, (late autumn or early winter) and in immune southern cattle. The red cells containing these rounded organisms were not crenated nor distorted, though 50 per cent. of them might contain the parasite. He looked on these as the earlier stage of the organism which later developed into the *piriform body*, by segmentation of its substance. The *piriform* or *spindle-shaped bodies* were usually found in pairs connected at their pointed ends by a filament and extending across nearly the whole breadth of the red globule. *Free microörganisms*, pear-shaped or round, he failed to find in the blood of the large vessels, but saw them

only in the cardiac capillaries and especially in the kidneys. In some cases the dim remnant of the disintegrated blood globule could still be detected around the parasite.

Laveran and Nicolle, examining the blood of Italian cases by fixing and staining, found the two forms, round or oval, and piriform, and claimed that the first passed into the second by segmentation.

Lignieres working in Buenos Ayres with the most ample opportunity as regards fresh material and authority to use it, watched the successive changes in the living organisms, and reached further conclusions. He diluted the blood with a 7 per cent. salt solution, or with ox serum or both, until the globules stood apart in the field. The blood can be kept under observation for days under a cover-glass luted with sterile paraffin, and the changes clearly traced. Securing the blood from a subject having a great abundance of infected globules (usually at the height of the hæmoglobinuria) he found mainly the *piriform parasite* intra-globular and free, and in the latter an active whirling motion was kept up by means of the flagellum at its pointed end. As usually arranged in pairs (gemina), whether inside or outside the globule, they are connected by the flagellum attached to their pointed ends. Careful observation enables one to detect in the pyriform mass a small brightly refrangent point like a nucleus. In this form the piroplasma is 3 to 4μ in length.

After 4 or 5 hours, and on toward the 8th, the piroplasma has assumed the *round* or *oval form* with a small linear prolongation (flagellum) and shrunken to 1 to 1½μ in diameter. All the piriform bodies pass into the rounded so that this last is the second stage of their development and not the first as was formerly supposed. The round forms are always present in great numbers in the cortex of the kidney in the second stage of the disease (toward the subsidence of the hæmoglobinuria). The refrangent nucleus is no longer to be seen.

After one, two or more days there appears in the round parasite a *chromatine mass*, which breaks up into 2, 3, 4 or 5 smaller *chromatic bodies*, which Lignieres considers as germs. He has seen no division of the protoplasm, but on the contrary the germs escape, yet remain for a time attached to the outer surface of the parent organism. They show rapid jerking movements.

Lignieres claims to have followed all these changes in the blood kept in a sterilized glass cup at room temperature or in the thermostat, and in the stomach of the tick, as well as on the warm stage of the microscope.

He claims to have made a further success in cultivating the parasite in ox-blood serum highly charged with hæmoglobin. It was only occasionally, and by the use of blood extraordinarily rich in the parasites, that success was obtained. In one such case he produced five successive cultures, the product being the rounded forms only and within these the germs. There were no piriform bodies. These are not formed outside of the red globules. The third successive culture in this medium grew with great readiness, producing larger parasites with less disposition to contract, but the fourth and fifth cultures were encreasingly poor. Inoculation with these cultures failed to produce the disease. To explain this the doctrine of passive germs, strong for survival, but weak pathogenically, is hazarded.

To summarize, the successive stages of the piroplasma are: 1st. The intraglobular *pear-shaped bodies* with flagellum often connecting two bodies. 2nd. The *rounded bodies with refrangent nucleus*—intraglobular or extraglobular. 3d. The *free round bodies* with the nucleus divided into 2 to 5 *chromatin masses*. 4th. The *free chromatin masses*, large, *active*, *infecting germs*, and small, *passive*, *non-infecting germs*. The insuccess of inoculations of cattle with the last named bodies throws an air of doubt upon them as links in the pathogenic chain. Definite information on the antecedents, environment, food, etc., of the cattle unsuccessfully inoculated, including the season, shelter and meteorological conditions might have brought us a step nearer to the full life history of the piroplasma.

The Cattle Tick: Boöphilus Annulatus: Boöphilus Bovis: Ixodus Bovis: I. Dugesii: The Invertebrate Host of the Texas Fever Organism. As early as 1868 shrewd observers had noticed that in all outbreaks of Texas fever the affected animals were covered with ticks, and drew the natural inference that the disease was due to the bites of these insects. But the prevalence of ticks in localities where the disease was unknown served to draw attention away from the important fact that was suggestive of the true explanation of the disease. The truth, however, con-

stantly obtruded itself that casual cases were never found in the absence of the tick. Finally, in 1889, Kilborne conceived the idea of putting the matter to the test, and with the approval of the Chief of the Bureau of Animal Industry, set aside special paddocks for this purpose. Five native cattle were placed, at midsummer, with three South Carolina cattle, from which all ticks had been carefully picked, and they completely escaped infection.

After seventy-one days, on September 6th, when the hottest weather had passed, two were turned into a lot with four South Carolina cattle of the original herd which had stocked the pasture with ticks. Of the two, one died of Texas fever, September 20th, and the other sickened in the last week of September, and had a relapse in October, but finally recovered. Of eleven other native cattle placed in this tick-infested field up to September 30th, ten sickened and one escaped. One animal placed in the field October 19th escaped.

Again three North Carolina cattle and three natives were placed in a field September 14th and 15th. The new generation of ticks was retarded by the cool season, so that few larvæ appeared on the native stock yet one of the three sickened.

These results were confirmed by a series of other similar experiments.

In a further experiment, September 13th, thousands of ticks, mostly mature, from North Carolina, were scattered over a sound lot and four native cattle turned into it next day. Three sickened and one, to outward appearance, escaped.

These results were corroborated by experiments made in succeeding years. In addition the disease was produced regularly in native stock by placing on their skin the six-legged larvæ of boophilus bovis, which had been hatched in glass vessels in the laboratory. It was also shown that the Washington winter destroyed the ticks in infected pastures so that native cattle could be safely turned on them the following spring or summer.

To summarize :—

1. The blood of southern cattle containing the piroplasma produced the disease when injected into a healthy susceptible animal.

2. The animal with piroplasma in its blood, did not convey the

disease, in the absence of ticks, to a susceptible animal kept with it.

3. The animal with piroplasma in its blood and covered with ticks conveyed the disease to a susceptible animal kept with it.

4. The ticks hatched and raised in glass vessels in the laboratory, when put on susceptible animals, infected them.

5. Ticks taken from cattle harboring the piroplasma, and put on the skin of susceptible animals, or on their pastures in the warm season, infected the exposed stock.

6. The six-legged larvæ developed in the laboratory from the eggs of mature ticks, taken from cattle having the piroplasma, conveyed the disease.

7. On bare pastures as far south as Washington the winter frosts destroyed the ticks so as to render the pastures safe on the following season.

8. Ticks artificially raised in a warm laboratory, produced the disease when placed on susceptible cattle in a warmed stable (65° to 80° F.) in winter.

9. In the Gulf states, in stables which the cattle occupy constantly or enter twice daily for milking or feeding, the ticks may live through the entire winter. The same has occurred in the warm swill stables in the north.

10. When taken into a new locality, it is rarely the mature ovigerous ticks that bite and infect the native cattle of the place, but the next generation of larvæ, so that time must be allowed for the laying and hatching of ova.

11. Ovipositing usually occupies about a week, while hatching varies with the temperature from two to six weeks.

12. Cases can be adduced in which native cattle followed, on the same pasture, the tick-bearing infecting cattle, and remained for a week or more, and yet escaped, the larvæ being as yet unhatched from the ova. Other native cattle, following these two or three weeks later, perished almost without exception.

13. This delay in the hatching may be indefinitely prolonged, and thus in the southern states, the winter may be tided over, without the loss of vitality in the ova, especially if it is covered by leaves, moss, wood, or decaying vegetable matter.

14. When dealing with lung plague in Chicago in 1888, I noted the facts that every cow that entered a city stable through

the stock yards during the dry, hot, midsummer weather died of Texas fever within a month, while those that passed through the same yards during a particular rainy week, all escaped. Berkau has shown that, in the absence of the coating of the glutinous saliva, the eggs do not hatch, and here we may assume that this covering was washed off by the rains and the eggs perished.

15. It has long been noticed that the ticks are scarcely at all dangerous to young calves living on milk. This applies not only to calves born of cows native to infected localities, and therefore possibly having a congenital immunity, but also to the calves of northern and susceptible cows, and which were exposed simultaneously with their dams. It suggests a special defensive power in even the bovine system when sustained on animal food. In the Bureau of Animal Industry experiments, calves of four months, already using vegetable food freely sickened, but still as a rule, recovered.

16. The Bureau fed three cattle with adult live ticks (2000 to an animal) but no infection resulted.

17. Four cattle were injected intravenously with the liquid charged by crushing ticks in a mortar with distilled water. In some cases the liquid was put through a Pasteur filter, in others only through two thicknesses of filter paper. No infection ensued.

18. Lignieres injected, subcutem, in different animals the pulp of the ticks at all stages of life, ground in a mortar with distilled water, but found in no case tristeza as the result nor any destruction of red globules.

The apparent paradox involved in the last three items probably finds its explanation in the statement of Nicolle and Adil-Bey that, in biting, the tick instils into the wound a venomous saliva which causes local congestion and infiltration and presumably operates on the blood globules as well. Curtice describes the two racemose glands situated under the head shield, the secretions of which are pressed out by the movements of the mouth ring and appendages. How much of this irritant and toxic action is inherent in the saliva, and how much due to the protozoan contained in it has not been shown. Nicolle, Adil-Bey and, later, Lignieres showed a similar toxic property in the blood. Three

to five cc. of blood taken from an acute case at the crisis and injected into the marginal vein of the ear in a rabbit, killed the subject in a few seconds. A similar amount thrown into the peritoneum of a Guineapig destroyed life in a few minutes. It is probable that the dilution of the venom in the mass of tick pulp and distilled water reduced its toxic quality to such a low ebb that the red globules were comparatively unaffected by it and successfully resisted the attacks of the microbe.

The name *Boöphilus bovis* was given to the bearer of the piroplasma by Cooper Curtice who made a special study of the tick, and its development. For the description see *Parasites, Ixodes*. Among the most marked and distinctive features of the female are the extreme shortness and relative breadth of the rostrum, the slender palpi, the eight rows of spines on the lower surface of the labium, the smooth mandibles with terminal hooks, the limbs long, slender, in seven segments, and each furnished with a terminal pad (pulvillus) and one hook (fore limbs) or two hooks (hind). Curtice has identified the ticks of hæmoglobinuria in various other countries with the boöphilus. The *Garrapata* of Mexico and the West Indies, the Hæmaphysalis rosea of Cuba (Koch), the Ixodes Annulata of Florida (Say), the Ixodes Dugesii of Italy (Nequin), the Ixodes Algeriensis and the Ixodes Egypti he found to be identical. There may be some doubt as to the Rhipicephalus Annulatus Microplus of Buenos Ayres, which agrees with the boöphilus in size, in the thickness of its rostrum, in the eight rows of hooks on the lower surface of the labium, in its host and habits, in the fact that it transfers the piroplasma to cattle, but the male has a distinct horny tail like the B. Australis with which it appears identical. Curtice holds that it was originally a North African tick, which was carried by the Spaniards to their American colonies. Rhipicephalus Annulatus of Roumania is probably the same, together with the ticks that convey the piroplasma in the other countries of Europe. There remain the B. Decoloratus of South Africa and the "Scrub-tick" of Australia both distinguished by the presence of a tail in the male. The life-history of the Queensland "Scrub" tick coincides with that of the boöphilus of America (Pound). In Rhodesia the brown tick R. Appendiculatus, carries the germ of the Coast fever.

Lesions. If the course of the disease has been short, followed by an early death, the carcass may be full and rounded, but if the animal has been sick for five or six days there is marked loss of condition and weight—emaciation. As after any other affection occurring during very hot weather, decomposition sets in early, though not quite so speedily as in anthrax, in which the subject dies full of rich blood. Something, too, depends on the condition at death, putrefaction being manifestly slower in protracted and debilitated cases. The color of the skin, the mucosæ and normally white tissues varies in the same way. As it has been largely seen in our northern States (and Australia) in fat cattle, which contracted the disease in railway cars, cattle markets, or dealers' or butchers' parks, etc., the deep orange hue of the white tissues is one of the most marked features, and even the muscles have a deep mahogany yellow hue. In poor milch cows and stock cattle in the South, on the other hand, the icteric hue is often conspicuous by its absence. Cattle killed early for experimental purposes may also show less icterus. The color appears to be influenced largely by the abundance of red globules in the blood when the animal was attacked, by the rapid destruction of these globules, and the saturation of the blood and tissues with hæmoglobin in solution. The presence of ticks on the skin, especially along the ventral aspect, inside of the thighs, on the scrotum, udder or perineum, sufficiently explains the number of minute infiltrations into the derma, the oozing of blood or serum, and the matting of the hairs into little tufts.

The *pale, watery condition of the blood* was recognized as one of the most constant features in 1868, together with the disappearance of the red glubules. The clot is remarkably soft and, at the crisis of the disease, the serum is of a reddish hue by reason of the hæmogoblin in solution. When, however, the urine is no longer stained, the hæmoglobin having been eliminated, the serum assumes its normal pale amber hue. For the first counting of the red globules in this disease we are indebted to the Bureau of Animal Industry. The average count in healthy cattle approximated to 6,000,000 per mm. of blood, and in three days this would descend to 4,000,000, 3,000,000, 2,000,000 or even 1,183,000. The rates of decrease was $\frac{1}{8}$ to $\frac{1}{6}$ of the entire number in one day. In case of recovery the repair of the red globules was

slow, from one to two months being required to bring them up to the normal standard. Lignieres claims recoveries after the count had gone as low as 300,000 per mm., and in fatal cases, a few hours before death, it may be but 31,000 per mm.

In high conditioned animals, with high fever, often aggravated by travel, the muscles may be dark and firm, but in those out of condition and in the advanced anæmic stages of the disease the muscles are pale, and there may be sub-cutaneous œdema below the chest and belly. These last features are especially noted by Smith and Kilborne.

The *lungs* are usually normal. Sometimes limited congestions, punctiform petechiæ, emphysema and small areas of œdema or hepatization are noticed (Smith and Kilborne).

The *pericardium* contains a little bloody serum and is marked by petechiæ.

The *left heart* is usually empty, but the *right heart* full of fluid, or later, of clotted blood, in the latter case without buffy coat. The endocardium, and especially on the musculi papillares, is marked by petechiæ, punctate or in considerable patches. The cardiac capillaries are full of blood, with numerous piroplasmata.

The *peritoneum* often contains a little reddish serosity, and a slight gelatinoid exudation is sometimes found around the kidneys or elsewhere in the abdomen. Petechiæ are frequent.

The *stomachs* usually show petechiated spots on the mucous-membranes, and more or less diffuse congestion. Sloughing of the mucosa at such points is not uncommon, and even perforation of the folds of the third and fourth stomachs. The Bureau of Animal Industry and Lignieres both found these stomach lesions very inconsiderable. The smaller pin-head erosions described by Gamgee were identified by the Bureau of Animal Industry as bites of the strongylus convolutus. The *small intestines* are usually moderately congested.

The *caecum* and *colon* show more congestion, becoming at times of a deep red or almost black hue, and considerable extravasation of blood may take place. This is especially marked in the rectum, which may be of a port wine hue, comparable to that seen in rinderpest or hæmorrhoidal anthrax. The fæces are often dry and massed in balls in cæcum and rectum, while if diarrhœa has set in, the discharge may be colored with blood or

blood elements. Yet in the cases reported by the Bureau serious lesions of the intestines were rather the exception, and some subjects showed scarcely any lesion.

The *liver* is usually enlarged, averaging three to five pounds heavier than in a healthy ox of the same weight. In these enlarged and congested cases it is of a deep yellowish brown color, and often shows yellow spots on the darker ground. Microscopically each acinus has a bright yellow centre from which yellow radiating canals diverge to join the peripheral gall duct. In the superficial or portal portion of the acinus, the hepatic cells are granular from fatty change, yet the nucleus is usually still recognizable. Toward the central zone it may have disappeared. The further this has advanced, the softer, the more easily pitted and the more friable the liver. The congestion of these radical gall ducts with the dense colored bile, displays the structure of the acini in a clear and beautiful way, which no injection can accomplish. When the affected tissue is teased out and placed under the microscope the inspissated contents of the bile canaliculi may be seen as yellow cylindroid casts sometimes bifurcated to represent the union of the two canals. If stained in Ehrlich's acid hæmatoxylin, the necrotic elements refuse to take the stain so that the contrast between the dead and the living tissues is enhanced. Fatty degeneration is common in the liver of healthy beef cattle so that this is less significant than the congestion of the acini, and the phenomenal distension of the radical gall ducts with inspissated bile.

The *gall bladder* is usually full (½ pint to 1 quart or more), and its mucous membrane congested and sometimes petechiated. The *bile* is thick and viscid, like tar, it may be yellowish green, darkening on exposure and contains hæmatoidin crystals and abundance of flocculi showing bright yellow or orange by transmitted light and reddish brown by reflected light.

The *spleen* is always enlarged, often enormously so. From an average weight of 1.5 lb. to 1.7 lb. for a 1000 lbs. ox, it will rise to 2, 7 or even 10 lbs. One measured 27 inches long by 7½ inches wide and in the centre 3 inches thick (Rauch). Even in apparent health the Gulf coast cattle have spleens averaging about 2½ lbs.

The spleen is gorged with blood which appears purple as seen

through the stretched and attenuated capsule, and darker petechial spots are found at intervals. When cut into, the pulp alone appears dark, brownish red, grumous, and showing under the microscope many red blood cells, larger cells granular and undergoing fatty degeneration, yellow flocculi, crystals of hæmatoidin, and granules of black pigment. It is the excess rather than the nature of these agents that is significant. The pulp may be pressed or washed out, bringing the trabeculæ and Malphigian bodies into view.

The *kidneys* are most seriously affected in acute and rapidly fatal cases. There may be œdema, with blood staining and even extravasation on their lower surface and in the adipose tissue. The gland may be enlarged and the cortical substance congested of a dark brownish red or black. Its capillaries are gorged with red globules in which the piroplasmata are very numerous. The medullary portion is much paler, and with fatty granules in the epithelium, and oil globules in the tubules. The renal pelvis is more or less petechiated and marked by extravasations.

The *bladder* is marked by petechiæ and usually contains some quarts of urine more or less deeply stained with hæmoglobin. The depth of color is in exact ratio with the extent and rapidity of the destruction of red globules, and of the elimination of their coloring matter. When the destruction is proceeding rapidly the urine may be as dark as port wine ; when their disintegration has lessened it may be pale though the temperature is still high (105° F). In slight and tardy cases there is reason to believe that the redness of the urine may be omitted altogether as is the icteric discoloration of the mucosæ, and hence cases seen in animals indigenous to the protozoan fever districts, have been described as a distinct disease. In these mild cases and advanced stages there is usually a certain amount of albuminuria remaining. In the early stages the urine is strongly alkaline, effervesces with acids, and has a high specific gravity (1030-1040); later when abstinence and suspended digestion and assimilation cause the patient to subsist on its own tissues the reaction may become distintly acid and the specific gravity reduced (1010-1020). It no longer effervesces. During convalescence while there is a great deficiency of red globules and other blood solids, the urine tends to become pale and watery, of a low specific gravity, and lacking in even its normal pigments.

The *womb* will at times show petechiæ and in pregnant cows the fœtus will show sero-sanguineous effusions or even extravasations in the chest or abdomen, and hæmoglobinuria (Lignieres).

Incubation. Outbreaks occurring in the North, in herds into which southern infected cattle have been brought, were at first held to indicate an incubation of thirty or forty days (or even sometimes sixty-five), but this is now explained by the time required for the laying and hatching of the eggs of the mature ticks and the evolution of infecting young larval or seed ticks. The actual incubation, as shown by the subcutaneous or intravenous injection of the blood of an infected ox, extends from three to ten days. The hyperthermia is usually shown on the third day, and the more manifest outward symptoms on the sixth. Extreme heat of the weather, a special susceptibility of the animal infected, and especially a large dose of the blood and protozoa will hasten somewhat the onset, but three to six days may be set down as the rule after the ticks have introduced the parasite into their victim. Cattle taken from the northern states and placed on southern pastures, or passing over trails already well stocked with the ticks, are infected at once and sicken in from three to ten days. Cattle in their northern home placed on a previously uninfested field with southern cattle just arrived, do not suffer for thirty, forty, sixty, and in exceptional cases, even ninety days. The paradox is explained by the time wanted for the laying of the eggs and the hatching of the tick larvæ. The female tick does not lay eggs until she is fully mature, and if the ticks on a southern ox are still immature there is a variable period of delay until the eggs are mature enough to be deposited. Then the ovigerous tick drops off her host and spends one week in laying her eggs. In warm weather these eggs take three to four weeks to hatch, so that usually five weeks elapse before the young (seed ticks) can climb upon the ox and infect him. Add three to six days more for the actual incubation and we account for about six weeks of delay in the appearance of the disease in northern cattle. If we consider further that a wet season occurring after the eggs have been laid and before they are hatched tends to divest them of their protective covering and to expose them to destruction, and that, in any case, a cold season will delay the hatching until the recurrence of warm weather,

and that the absence of bovine victims will doom the new-born larva to an arrest of development, so that further indefinite delay may be entailed, we have abundant explanation of the frequently delayed evolution of symptoms. Yet in general terms the apparent prolongation of incubation is due to fortuitous circumstances which delay the infection, and not to any actual extension of the incubation itself.

Symptoms of Acute Type. Cattle infected outside the area of habitual prevalence and stock from non-infected districts, conveyed into the infected ones in hot weather, usually contract the disease in its acute and fatal form. The period of the year is often significant, a number of animals being attacked at once in the hot dry period of late summer or autumn—July to September in North America, February to May in Argentina.

The first symptom is a rise of temperatnre, and this may last two or even three days before other morbid phenomena are noticed. It may rise to 104° F. in the first day and later to 107°, 108° or 109°. The more acute the case and the hotter the weather the greater the rise. The highest records are obtained late in the day, the lowest in the morning. The temperature often rises for two to four days, and then suddenly drops with the occurrence of collapse and imminent death. While the thermometer is of the highest value in taking the temperature, yet the extraordinary hyperthermia is easily detected by grasping the root of the horn or ear, or by feeling the nose, feet, anus or lips of the vulva.

After 2 or 3 days the respirations become accelerated to 60 to 100 per minute, and the pulse to 90 to 100 or more. There is complete loss of appetite and rumination after the development of these symptoms, the mouth is hot and it may be dry, the muzzle dry, the head pendant, the eyes dull or semiclosed and congested (usually icteric), the bowels confined, to be relaxed again as the fever subsides. A disposition to stand or lie down in water has been frequently noted. Nervous symptoms are usually present. The extreme dulness, languor, and apathy, the drooping head and ears, the unsteadiness of the support the animal staggering or propping himself up by spreading all four limbs, and the tendency to assume and retain a recumbent position, are marked phenomena in our domesticated northern cattle. The paresis

may absolutely incapacitate the animal from getting up. In our wilder range cattle it may show itself in active delirium and Lignieres notes the same of the Pampas cattle in Argentina. The animal lying dull and apathetic (triste), on being approached may raise his head, open his eyes and glare threateningly at the intruder. Sometimes when trembling violently, and swaying ready to fall, he will marshal all his remaining energy to plunge at a man on foot or mounted. Some have become blind and unconsciously walked against obstacles, others have been noticed to run in wide circles.

The milk secretion is suppressed, any little that can be drawn in the advanced stages having a thick, creamy appearance. Abortion is common in the pregnant cow.

The condition of the urine has, however, always drawn especial attention and the names *red-water* and *hæmoglobinuria* have accordingly been largely applied to the disease. When, in infected areas, the milder types of the disease have failed to show *red-water* (Jamaica), the identity of the affection with Texas fever has even been denied. Shortly after the rise of temperature, the urine becomes turbid, and this gradually encreases to a more or less deep red. It assumes its darkest hue when the destruction of red globules is most active and during convalescence it disappears. The suppression of urinary secretion may account in some cases for the absence of this symptom even at the crisis of the fever, yet, as a rule, it is present at such time, and, even though it may have escaped notice during life, the *red-water* is found in the bladder at the necropsy. It may be of all grades, from the merest tinge of redness to a reddish brown, coffee-grounds, or blackish aspect. The coloration is not due to red globules, but to the hæmoglobin which has escaped from the disintegrating globules, and been eliminated by the kidneys. It is always associated with albumen, and, in the advanced stages and during convalescence, when the elimination of hæmoglobin has ceased, that of albumen continues in small amount for weeks.

The thin, watery appearance of the blood when the disease has reached its height, is constant and even more characteristic than the *red-water*. A single drop drawn from the skin will show to the naked eye the pale, thin, transparent appearance, but examination under the microscope will confirm this. It remains,

too, for a length of time, being recognizable for a month in cases of recovery. In connection with the watery blood, the mucosæ and the muzzle (if naturally white) assume a pallid aspect. This is best marked in the absence of icterus, yet even with the yellow discoloration, the absence of ramifying red vessels is very characteristic.

The bowels are at first constipated and the fæces passed in small, hard balls. Later they may assume a reddish brown or chocolate color, and a covering of mucus and fine blood clots. Diarrhœa supervenes in some cases. In passing the fæces, the everted mucosa usually shows a dark red color.

Course and Duration. Acute cases, above all if traveled or otherwise excited, may terminate in death in 24 to 48 hours. More commonly death will take place in 4 to 7 days. Some patients survive longer, but owing to the extraordinary loss of blood globules and the lesions of important solid tissues they are unable to rally, become steadily weaker and perish in from two weeks to three months. Such animals are pale and bloodless, weak on their limbs, careless of food, and encreasingly emaciated. The pulse is weak and irritable and the eyes sunken. The temperature becomes normal or nearly so, soon after the suspension of the hæmoglobinuria. In cases of recovery there remains for a month or more an unnatural pallor, with marked loss of condition and weakness which are only gradually overcome. Convalescent animals are liable to die of indigestion when overfed.

The *mortality* averages not less than 90 per cent. in susceptible mature cattle from a healthy district in the hot season. Later, from October onward, the tendency is to a milder type of disease and a greater ratio of recoveries.

Symptoms of the Mild Type. This is seen mainly in cattle indigenous to the Texas fever district, in sucking calves, and in mature cattle from healthy districts but attacked during the cool or winter season. It can be produced at will by placing a limited number of ticks (5 to 20) on the skin of susceptible cattle, especially in the cool season. Again, it occurs as a relapse in cattle that have survived an attack earlier in the season.

Though there are all gradations from the violent type, yet we may set down as mild all cases in which the temperature does not rise above 105° F., running frequently about 103° F. There is

loss of appetite, dulness, languor, costiveness, scanty urine, albuminous but not hæmoglobinuric, pallor of the mucosæ, and marked loss of condition. Examination of the blood shows the presence of the parasite in the red globules but usually in the coccus or round form only, and the destruction and disappearance of the globules is much less marked so that, though the blood is anæmic and watery, it is not nearly so much so as in the violent and fatal cases. Without the examination of the blood it may be impossible to distinguish these cases from other febrile affections, yet occurring as they do in the infected district in a number of animals at once, in the cooler season, and showing albuminuria, and marked anæmic symptoms, they should lead to suspicion and a search for the boöphilus on the skin, and the oligocythemia and the protozoa in the blood.

Differential Diagnosis from Anthrax. As anthrax is the one disease with which Texas fever is most likely to be confounded, it may be profitable to collect in tabular form their differential features.

Protozoan Cattle Fever.	**Anthrax.**
A disease of given districts, wooded, swampy, uncultivated, and places where cattle from these districts are taken.	Prevails in rich, swampy impermeable soils: not permanently implanted on open well drained lands.
Always shows Boöphilus, minute or mature, on the skin of the patient.	Has no essential connection with the Boöphilus: latter usually absent.
Extends in warm season, as the Boöphilus from an infected district is introduced.	Practically never conveyed by the Boöphilus.
Can be carried by the Boöphilus apart from cattle.	
Infection destroyed by winter frost above 35° of N. latitude.	Infection not destroyed by winter frosts
Attacks bovine animals only.	Attacks mammals, generally, especially herbivora.
Lapse of 5 to 6 weeks after introduction, for tick development, precedes outbreak.	Outbreak may occur at once after introduction of infection.
Confined mainly to summer season	Occurs at any season: most prevalent in summer.
Sucking calves nearly immune.	Sucking calves susceptible.
Mucosæ become increasingly pale; icteric in violent attacks.	Mucosæ dusky brownish red, not pallid, nor icteric.
Urine red or blackish in acute cases at height of the disease. Hæmoglobin. Albumen.	Urine exceptionally red: blood globules, in some cases.
Blood becomes increasingly thin and watery: Oligocythemia.	Blood becomes thick, tarry, incoagulable; not watery nor oligocythemic.
Blood reddens more or less in air: then becomes darker.	Blood reddens little, or not at all on exposure.
Spleen greatly enlarged, purplish, often firm: pulp reddens slightly in air.	Spleen greatly enlarged; pulp very dark; does not redden in air.
Liver enlarged, congested, often yellow, with fatty and necrotic areas: remarkable yellow injection of radical bile ducts.	Liver enlarged, congested, dark red or violet.
Bile abundant, thick, tarry.	Bile fluid.
Kidneys pale, or congested, black.	Kidneys congested.
Lymph glands slightly enlarged, petechiated or (rarely) hæmorrhagic.	Lymph glands enlarged; often hæmorrhagic, black; peripheral œdema.
Pallor in violent cases, in plethoric, icterus of tissues, especially of white ones.	Tissues generally reddish, blood-vessels dark, prominent.
Muscles in acute cases mahogany colored.	Muscles pale, grayish, unless they are seat of congestion or extravasation.
Blood and its red globules show the pyroplasma in different forms.	Blood shows bacillus anthracis, but no pyroplasma.

Treatment. Up to the present medical treatment has been essentially unsatisfactory. Lignieres gave quinia sulphate in large doses by the mouth, and in doses of 2½ drams subcutem

daily, before and during infection without any visible effect on the progress of the disease. Methylene blue (¾ to 1¼ dr.), salicylate of soda (7½ drs. daily), arsenious acid (1¼ dr. daily), cacodylate of soda (7½ grs. subcutem) were also tried with no good result. The Metropolitan Board of Health, New York, claimed a succession of recoveries under the use of carbolic acid in the drinking water and sprinkled on the ground so that the animals inhaled it. The cases were, however, the survivors after the first and more acute cases had perished, and the results no doubt depended largely on the mildness of the attacks. The same agent in other hands has not been equally successful. A large number of other agents have been used in vain.

Among the most important measures are a laxative food, like flax seed gruel, a careful picking of all ticks from the surface, the washing of the skin with a 5 per cent solution of creolin, and the removal of the animals to a tick-free pasture, lot, or building. This at once arrests the introduction into the blood of fresh and continuous accessions of the pyroplasma and, if begun early enough, will determine a mild and nonfatal case.

Prevention. The prevention of the protozoan cattle fever is based on the life history of the parasite, and may be directed (1st) to the *destruction of the Boöphilus Annulatus;* or (2nd) to *encreasing the resisting power of the exposed animal, to the Piroplasma Bigeminum. 1st. Destruction of the Ticks. (a) On the cattle.* The *picking of ticks from the skin* is effective if the object is to make the animal safe for a few days only as the boöphilus habitually clings to the skin of the one ox from the stage of seed tick to that of ovigerous female, ready to drop off and lay its eggs. An animal going direct to slaughter may therefore be sent through an uninfested district, even in the hot season with a fair amount of safety, after the careful gleaning of the ticks. The greatest care, however, must be taken to manipulate thoroughly all parts of the skin and above all, the ventral aspect, the inner sides of the limbs, the scrotum, udder and perineum. The animals must be shipped at once after such gleaning of ticks with no further opportunity of taking on a new supply, the cars and other conveyances must be cleaned and treated with acaricides and the litter burned as soon as they are vacated, and the cattle must be passed over no loading banks, chutes nor yards that may by any possibility be used for other cattle unless these are going into a slaughter house.

The picking will safely remove all the larger larvæ, and the mature ticks which are ready to lay their eggs, but it cannot be implicitly trusted to remove also the all but invisible embryos or seed ticks, and if the host is preserved these grow up and mature, while if they are accidentally dropped or brushed from the surface, they climb upon the first available ox and mature on that. By passing from ox to ox they may be kept alive for a time in the pens adjoining the slaughter house, but fortunately they do not travel over a few feet and if no cattle escape from such pens there is small risk of their preservation.

Dipping or smearing to destroy the seed ticks on the skin becomes an essential adjunct to, or substitute for, picking. The Bureau of Animal Industry has experimented largely on dips with most important and valuable results, even if they have proved only in a measure successful and desirable. Aqueous dips they early discarded. Poisonous agents like corrosive sublimate and arsenic are liable to poison through absorption and licking, with the added drawback that neither these nor calcium sulphide are at all effective in destroying the ticks. Proprietary sheep dips were abandoned on similar grounds. Baths of cotton seed oil were introduced by Francis, but proved not quite effective even when phenic acid, benzine, gasoline, or different mineral oils were added. *Paraffin oil* gave the best results, and later a staple sold as *extra dynamo oil*, which in combination with *sulphur* (1 : 100) proved most destructive to the ticks, was adopted. But in the hot season, when such dipping is required, any one of these baths produced *heating*, and illness in the cattle, and together with the exertion and excitement served to rouse into dangerous activity the germs already present in the blood. Ophthalmia, too, was a very frequent result. If the cattle could be kept on their native pastures the dipping might be permissible, but this was to lose the object aimed at—the wholesomeness of these cattle on uninfected ranges. When shipped north in the hot weather the losses were so great as to be prohibitory.

If, however, it could be reserved for use on the southern pastures, to prevent the maturing of the ticks and the laying of eggs for a future generation, it might be employed to rid the infested pastures of the boöphilus, and consequently of infection. The question, then, is reduced to the comparative advantage of the

destruction of the ticks, on the one hand, and the cost of frequent dipping throughout the warm season, on the other. The following season there ought to be no ticks left.

Cooper Curtice advocates kerosene 1 gallon in combination with an equal amount of lard, 1 lb. sulphur, and 2 lbs. pine tar. Melt the lard, add the sulphur and tar, bring to the boiling point, cool, add the kerosene with stirring. Rub daily with a brush on the whole skin but especially inside the arms and thighs. On tick-infested pastures it must be continued through the season, and if thoroughly done will leave the fields tick-free the following year. Like oil dipping it would manifestly be incompatible with immediate shipment on a long railway journey, but Curtice vouches for its efficacy as a means of eliminating ticks from southern pastures. The main question is the expense. What would be perfectly adapted to small herds of very domesticated cattle in North Carolina would be a herculean and expensive task in the large herds of Texas. Curtice mentions cotton seed oil, fish oil and even a small proportion of linseed oil as good substitutes for the lard.

Destruction of Ticks on Pastures. Fields, farms and larger areas can be freed from the boöphilus by the thorough application to the cattle pastured on them of one of the above-described methods, provided that no strange cattle are admitted on the land. The ticks are sluggish and, unless carried on the bodies of animals, do not crawl many feet from where they drop. If cattle are kept in the next lot, they should not be allowed to come in contact with the treated or protected stock, but a double fence with an interval of five or six feet, will prove a sufficient barrier to the advances of the tick, apart from its bovine host.

Cultivation of a tick-infested soil for one year or more, with complete exclusion of cattle from November or December until March or April of the second year thereafter, will exterminate the ticks. During the intervening summer there may be plenty of young live ticks on this land, but, in the absence of the bovine host, and blood, these cannot reach maturity, lay their eggs and thus leave new generations. In the course of the second winter therefore they are exterminated. In restocking such land, it is all important to see that the cattle placed upon it do not introduce any ticks on their bodies. Equally essential is it, to see that

cattle are excluded from the cultivated land in winter as well as in summer. During warm days ovigerous female ticks, dropped from the skins of such cattle, may produce eggs and larvæ to start a new crop in the coming summer. But as has happened to the wood ticks of the North, so in the South, cultivation of the soil and the exclusion of cattle for a length of time, will exterminate the race of ixodes.

Exclusion of cattle for two winters and the intervening summer will eradicate the ticks even in the absence of cultivated crops. To reach full maturity and propagate its kind, the tick must have bovine blood. If therefore the ticks of a whole season (spring, summer and autumn) are denied bovine victims, and thus cut short in their development, no crop is left for the succeeding spring. If then a cattle pasture is divided in two parts by a double fence with an intervening space of 5 or 6 feet, and if the cattle are confined to one of these parts for a whole year and are transferred to the second half in January after dipping or smearing with ixodicide oil they may be kept entirely free from ticks thereafter. That half of the pasture which is abandoned in the second year, will be tick-free and salubrious in the third year.

Another resort, advised by Curtice when a large tick-free pasture is available, is to place the infected cattle in a pen, and *soil* them for three weeks, no longer. Then transfer them to another clean pen and *soil* them there for three weeks more. Then examine closely, and if entirely free from ticks they can be put in the large clean pasture. Should they still carry a few ticks they should be placed in a third clean pen for two weeks more, when they will be tick-free and may be turned into the large pasture without the formality of examination. This is substantially based on the period of parasitism of the tick on the skin of the ox, and its development from the newly hatched larva (seed tick), to the ovigerous female. This period is from three to four weeks. The greater number of the ticks are therefore dropped off as mature ticks to lay their eggs in the first pen, while the remainder are similarly left in the second pen. As the stock leaves the two pens in succession long before the deposited eggs have had time to hatch out, they can take on no more ticks and emerge from the second pen clean and safe. The same pens cannot be

used repeatedly, as the eggs develop into seed ticks in 15 days in hot weather, and at once attack cattle.

A stockowner who, independently of his fellows, adopts one of the above expedients in an infested district, is however confronted by the risk of the infection of his herd, by the accidental or careless contact of his cattle with outside ones, and especially with the places where they have been. A broken fence and the entrance of tick infested cattle, or the escape of his tick-free cattle into infested lands, will be the death warrant of all that have not been previously exposed to the disease. Another consideration is that this rigid seclusion of the protected herd must be continued indefinitely so long as ticks are maintained anywhere in the district. The protected animals cannot be driven over a highway without exposing them to almost certain death. Even if a group of adjacent stockowners agree to purify their respective farms, they cannot debar their less careful neighbors from using the highways for tick-infected stock, nor from turning such out on adjoining fields. The veriest scrubs, admitted to the highways, woods and unfenced grounds, keep up the general diffusion of the fever germ and its tick bearer and undo the best directed efforts of any combination of owners of high class and valuable stock.

Well directed legislation, excluding cattle for one or two years, from all woods and unenclosed lands, and enforcing some one of the available methods for the clearing of fenced and stocked lands (cultivation, pasturage by cattle on alternate years, frequent dipping or smearing, passing the stock through a succession of pens), could be made to put an end for all time to the obnoxious tick. If even some other than bovine animals should be discovered to harbor the boöphilus and pyroplasma it could be included in the prohibition and the work made complete. The results would far more than compensate for any necessary outlay. Illinois, with 55,414 square miles of area has over 3,000,000 cattle. The coast states from Virginia to Texas, with Arkansas, Indian Territory and Oklahoma, amount to 767,215 square miles, and in the same ratio should sustain 43,340,040 head. A stock of 25,000,000 at $20 per head would amount to a capital of $500,000,000. Immune from the pyroplasma these cattle would draw freely on the best blood of the north and under the milder skies would compete

with the northern cattle on more than equal terms. Living in the open air, they would in the main escape tuberculosis and the other stable-propagated diseases of the north, and their dairy and beef products would enter the market free from suspicion, and command a readier sale, if not a higher price. The stock themselves could be moved to northern markets at all seasons without restriction, and escape the serious losses that now come from a sudden transfer, while pyroplasma-infected and susceptible, to the violent excitement of travel, and the frost-bound destination. Their owners could watch the markets and sell in the best, in place of being compelled, as at present, to hurry them in during November and December, and to sell often at a ruinous sacrifice. With the extinction of the boöphilus the present unrestricted pasturage and all other privileges now enjoyed would return, freighted with a value never borne before, and the free Southern cattle and cattle products need fear no competition in the markets of the world, and could no longer be justly subjected to any restriction.

There would remain the constant danger of the introduction of the boöphilus anew from Mexico, the West Indian Islands and the Central and South American States, where in the absence of frosts the boöphilus cannot be extirpated in the same way, and here accordingly all importation must be forbidden. Cattle from Southern Florida and from islands on our Southern Coast may demand a similar exclusion. There is too much at stake to permit any laxity, and no infected area should be allowed to send out its cattle until it has been abundantly well proved that such district or State is absolutely tick-free.

2d. *Immunization : Encreasing the Resistance to the Piroplasma Bigeminum.* That cattle can be fortified to resist the attacks of the piroplasma is shown in the immunity possessed by the indigenous herds generally, in the regions infested with this parasite and the boöphilus. To begin with, there may be a survival of the fittest, the more susceptible strains of blood having been long ago cut off. But the immune southern cattle if kept for years outside of the infested area and then returned to it, suffer a mortality about as great as that of northern cattle in the same circumstances. Their earlier immunity, therefore, is not merely a racial difference, but must be due in greatest part to an acquired

resistance, and further, this resistance is not permanent but must be renewed at short intervals. The immunity may be in part acquired in the womb of the infested dam, in the last months of gestation, but it is chiefly post-natal through the attacks of the ticks.

a. Infection of sucking calf. The indigenous cattle acquire immunity mainly through the attacks of the boöphilus, in the first month or two when they are still on an exclusively milk diet, that renders the piroplasma practically harmless. Following a parallel method, calves, living on milk alone, can be taken into the infected regions and exposed to the attacks of the ticks with safety, and with the result of protection for the future. Escaping the first invasion, they continue to harbor as many ticks summer after summer, as will reinforce yearly their acquired power of resistance, so that they continue measurably safe though spending the life in the area of the infection.

Francis and Connoway applied this to Jersey calves of two to six weeks old applying to each 25 to 50 ticks. It led to slight hyperthermia, some dulness and inappetence, but on recovery they all gained flesh and condition. Two died from exposure but necropsy showed no sign of Texas fever. The following summer all were infested with 200 to 500 ticks apiece but not one sickened in consequence. These were Jersey calves (the least susceptible breed) and the experiments were made in cool weather in autumn. The limitation of the practice to the cool fall or winter months renders the operation much more safe.

b. Infecting Older Animals by a limited number of Ticks. Yearling Jerseys, Holsteins and Shorthorns were subjected to 25 to 50 ticks in July, they showed only slight rise of temperature, and later resisted the free exposure to tick infestation. It must be recognized that these were still young animals, with presumably greater resisting power than the mature, but on the other hand they were of the susceptible northern herds, they were first infested in the hottest season, and the acquired resistance appears to have been perfect throughout the succeeding summer. The added precaution of subjecting them, in late autumn or winter only, to the ticks raised in a warm room or thermostat, would add greatly to the safety of the operation. After recovery from the effects of the first crop of ticks, a second crop of 50 to 100

should be placed on the skin so that the system may be thoroughly habituated to them and the measure of resistance correspondingly strengthened.

This measure may be advantageously applied to valuable cattle that are to be moved into the infecting territory, but it has serious drawbacks. The relative strength of the poison introduced by the ticks to the susceptibility of the animals on which they are placed, can never be perfectly gauged, and a certain small but appreciable number of deaths result from this first infesting. This has been observed in North and South America, Australia, Roumania and Turkey. Again, the plan entails the necessity for clean, noninfected premises (lots or buildings) for each fresh lot of cattle, as the places previously used are left in a tick-infested condition, and are likely to furnish a dangerous excess of ticks to any susceptible animal. The buildings could, of course, be disinfected and purified, but this entails considerable expense.

c. Infection by Graduated Injections of Blood Containing the Pyroplasma. Up to the present this is the most promising method of securing resistance to the pyroplasma. It is advised to take the blood from an immunized northern animal or from one indigenous to the infected district. Such an animal is not, however, strictly speaking *immunized.* It has acquired a *tolerance* so that it is no longer in much danger of succumbing to the pyroplasma, but it does not exclude the pyroplasma from its system. The *micro-parasite* is still found in the blood, though mainly in the coccus-like form in the interior of the red globules. The animal to be fortified against the disease is therefore inoculated with the germ of the disease itself, though it may be, at the time, in a somewhat inactive form. If, however, the inoculated animal is specially susceptible, or if the dose is excessive, the disease is produced in deadly form. The virulence is less in the case of blood drawn from a northern animal just recovered from the disease, than from an animal indigenous to the infected district, and which harbors the pyroplasma, it may be in spore form (Lignieres), without showing obvious disease. The former source of the blood is therefore the more desirable, while the latter is the more easily obtained. The precaution, however, should be adopted of reducing the dose when taken from an in-

digenous animal. Another important precaution is to select the winter or cooler season for the operation rather than the summer.

The animal which is to furnish the blood may be fixed in stocks, or held with a bull ring, or it may be cast so that it can be kept still. The hair is clipped or shaved from over the jugular vein in the upper third of the neck and the surface is washed with soap and water and with a five per cent. aqueous solution of carbolic acid. A thick cord (¼ inch) is tied tightly around the back part of the neck so as to compress and raise the jugulars. With a sharp pointed bistuory sterilized by boiling, a small incision is made through the skin, directly over the centre of the jugular and a cannula and trochar $\frac{1}{10}$th inch in diameter and sterilized by boiling, is passed obliquely upward through the coats of the vein and the trochar withdrawn. The blood flows through the cannula and is received in a sterilized (scalded) glass beaker. The blood is stirred slowly with a sterilized glass rod until all the fibrine has coagulated when the latter is lifted out and the remaining liquid blood is ready for use. The blood is injected with a hypodermic syringe which, with its nozzle, has been thoroughly sterilized by boiling. The point selected for injection is usually back of the scapula on the middle of the chest. The skin is clipped or shaved, washed with soap, soaked in a five per cent. carbolic acid solution, then pinched up, perforated with the point of the bistuory, and with the nozzle of the syringe passed through this wound the blood is injected into the subcutaneous connective tissue. The slight wound may then be covered with tar or collodion or merely left undressed. The mass of blood in the connective tissue may be diffused through its meshes by rubbing so as to favor absorption.

The dose of defibrinated blood employed is 5 cc. if from an *immunized* northern ox, or 3 cc. or even 2½ cc. if from an indigenous animal. The animal operated on should be in good health and condition, well fed, and kept if possible in the shade, in a cool stable, or under trees.

In some respects it is preferable to operate on the animals before they are moved from the north or other noninfected territory, but as there is danger of infection in preserving and carrying the blood, the treatment is more conveniently deferred until the animal reaches the infected region where the blood can be had fresh. In

such cases the animals should be shipped in carefully disinfected cars, and before leaving they should be liberally oiled or larded so that the ticks will not climb upon them, in being led to their stable. They must be kept stabled until the febrile effects of the injection have entirely passed, usually a month or more.

d. Injection of Blood from Bodies of the Ticks. In view of the difficulty of shipping infected blood without danger of contamination or sepsis, and the occasional accidents that happen to animals injected with such blood outside of the infected area, attempts have been made with dried blood, or that charged with antiseptics (calcium oxalate), or that had been frozen, but in every instance the virulence of the pyroplasma was destroyed. Dalrymple and Dodson availed of the blood drawn by mature ticks, which, in their blood-gorged condition, were shipped to the points where the injections were to be made. The mature ticks charged with blood were taken from infected indigenous cattle, and at once shipped. On their arrival they were washed externally with a mercuric chloride solution (1:1000) to destroy any adherent saprophytic or other bacteria, mashed in a sterilized mortar, with a few cubic centimeters of boiled water and the fluid portion drawn off and injected subcutem, into the animal to be protected. From 3 to 12 mature female ticks were used for each animal. The results were the same, only milder than when the blood of the indigenous animal was used direct, and the subsequent tolerance of the pyroplasma proved satisfactory.

It is difficult to explain the moderate effect of the considerable mass of blood injected in such cases, as compared with the deadly effect of the small amount that could come from the insertion of the rostra of even 50 or 100 ticks. But perhaps the venomous saliva instilled in concentrated form into the bites, protects the pyroplasma in the very limited area, until it gains sufficiently in numbers and force to hold its own even in the circulating blood.

Limited Value of Artificially Induced Tolerance. It must be added that all these measures for securing a partial immunity in the individual animal, and which enable us to safely introduce previously susceptible cattle into an infected district, virtually imply the continuance of the infection and infection bearer (boöphilus) for all time. They give no promise of the extinction of the bovine infection at even a remote future time, nor the

abolition of the taxes for prevention, which must oppress the southern cattle owner so long as the disease continues. They are most valuable measures truly, but mere temporizing ones at the best, and they could just as well give place to the more sanitary, economical and statesmanlike measure for its radical extinction.

Marketing of the Beef. The piroplasma is not communicable to man, so that the carcasses of well conditioned cattle, which bear the infection need not be rejected as human food. It is only in severe and advanced cases in which anæmia, emaciation and pallid innutritious muscles are marked features, that the flesh is objectionable, and then only as being somewhat lacking in nutriment and digestibility,—not because of poisonous qualities. Danger of infection to cattle might be apprehended, but, if used outside the infected area, the second condition of the disease—the boöphilus—is lacking, while within the existing area of prevalance of the fever, the propagation from the carcass to the animal is infinitely less likely than from one live animal to its fellow.

Federal Restrictions on Cattle within Infected Areas. The orders of the Secretary of Agriculture prohibit the removal of cattle from the following states and territory into any states that extend northward of the line indicated: California, Oklahoma, Indian Territory, Arkansas, Tennessee, Virginia and the states south of these to the Gulf of Mexico. Exceptions are made in the case of fat cattle, sent out of an infected area, for immediate slaughter at the point of destination; conveyed in cars or boats placarded as containing Southern cattle and receiving no other; fed and watered enroute in yards that admit no local or other cattle and which can be reached without passing over any highway or unfenced open ground; and unshipped at their destination directly into yards reserved for Southern cattle only and within the same enclosure as the slaughter house. If reshipped, the cars used must be subjected to the same restrictions. The cars, boats, shutes, alleyways, pens and troughs are to be disinfected by thorough cleaning; by saturation of all wood work, etc., with a mixture of 1½ lbs. lime, ¼ lb. phenic acid and 1 gallon of water, or ¼ lb. chloride of lime in a gallon of water, or a jet of steam under a pressure of 30lb. to the square inch. The manure and litter must be mixed with quicklime, or saturated with a 5 per cent. solution of

carbolic acid, or secluded in a well fenced enclosure from February 1st to November 15th of each year. This is made the duty of the stock yard companies.

Cattle may be freely moved north from the infected area at any time from November 1st to December 31st, if inspected by an officer of the U. S. Dept. of Agriculture and found free from infection.

Provision is also made for sending infected cattle northward at any season, if they have been first dipped and pronounced free from the disease by an inspector of the department.

Cattle from Mexico are admitted under analogous rules.

AFRICAN COAST PIROPLASMOSIS OF CATTLE.

Synonyms: Rhodesian fever. African fever. East Coast fever. Tropical piroplasmosis.

Definition. A fatal piroplasmosis of cattle in Portuguese and British E. Africa, resembling Texas Fever, but not communicable by one transfer of blood, not causative of red water, sometimes complicated by pulmonary lesions, and very fatal (90%).

Geographical Distribution. This affection was recognized in German E. Africa and Rhodesia for many years, and in Portuguese E. Africa in 1896, the time of the Mashona rebellion, among transport cattle placed on low ground near Beira. A similar affection is alleged to exist in Asia Minor, so that importation appears reasonable, yet if imported cattle were the first victims at Beira this would argue rather an infection waiting and ready to attack them at that point. From Beira it spread inland to Mashona, Bulawayo, Transvaal and Orange River Colony, gaining a free extension during the Boer war.

Microbiology. The protozoön found in the red globules is smaller than the piroplasma bigeminum of Texas fever and shows a rod shaped or round form. The rod is sometimes enlarged at one end where it contains the karyosome, thus approximating to the Texas fever parasite. The round form resembles an oval or elongated nucleated ring giving the differential characters according to Koch, but Theiler alleges that

these rings can be found at will by inoculating a calf with the blood of a recovered Texas fever animal and examining the blood daily. Moreover Cape Colony cattle having such ring shaped bodies in their blood die promptly when exposed to the *Coast fever*. Robertson, Bowhill and Dixon agree with Theiler. The parasite is best stained in eosin and azure II, having been fixed in alcohol and ether and then immersed in the staining mixture (freshly mixed) and left in an incubator at 30° F. for two hours.

Tick bearers. In the infected areas Theiler found the *blue tick* (Rhipicephalus decoloratus) the most abundant. Lounsbury, in a series of experiments, was especially successful with the mature *brown tick* (R. Appendiculatus), the larva of which had fed on *Coast fever cattle*. A single tick produced the disease. But the larval *brown ticks* hatched from eggs laid by mature females taken from *Coast fever* cattle proved noninfecting. Larval *blue ticks* also failed. The *brown tick* nymphæ on the other hand which as larvæ had fed on *Coast fever* cattle proved infecting. He notes further that the *blue tick* clings to the same host from the larva just hatched to maturity, whereas the *brown tick* drops off before each moulting and thus the same tick may infect at least two cattle in succession. In a series of careful experiments, the *blue tick* failed to convey the disease (Lounsbury, Robertson).

Nature of disease. This is in the main the same as in its near ally, Texas fever, with which it is often confounded and even complicated. Like that it is not communicated directly, except by the (repeated) transfer of blood, and when conveyed by ticks, in pastures, roads and buildings there is the delay of a fortnight for moulting, between the introduction of the sick ox and the infection of the susceptible animal. A week more is required for *incubation*. Koch says the sick ox may be stabled with the susceptible without transmitting the disease but this can only be true when the former has been divested of its ticks. The disease differs from Texas fever in this that there is no such destruction of red globules, nor staining of the urine by freed hæmoglobin. An attack of *Rinderpest* or of *Coast fever* in an ox recovered from Texas fever (salted) determines a relapse of the latter (Robertson).

Symptoms. After an incubation of six to twelve days, there

is a sudden extreme elevation of temperature (106° F.) with at first little change in appetite, rumination, breathing or secretions. Slight salivation, glazing of the fæces, dulness, pain and stupor come on by degrees. Resting of the chin on the manger, or, during the prolonged recumbency, on the ground, glazing of the eye, weakness and staggering, intermaxillary œdema, fœtid, even bloody, diarrhœa, cough, dyspnœa, flatness on percussion over the lungs, with abnormal murmurs, and at times crepitating subcutaneous swellings are to be noted.

Lesions. Swelling and hæmorrhagic condition of the lymph glands is a prominent lesion. Congestion of tonsils, larynx and trachea; œdema of the lungs, pleural liquid effusion; congestion and ulceration of the abomasum, with dropsical condition of the folds; congestion hæmorrhagic effusion and even necrotic changes of the intestinal mucosa, especially of the cæcum, colon and rectum; infarcts of the kidneys; and extensive ecchymosis. Unlike Texas fever the spleen is comparatively normal and there is no dearth of erythrocytes.

Prevention. As treatment is as fruitless as in Texas fever attention has been mainly directed to prophylaxis. Koch advises immunization by the subcutaneous injection of 5 cc. defibrinated blood, from the jugular of a recovered ox, to be repeated every two weeks for five months. His previous inoculations of 5 cc. and 10 cc., extended over five months, failed in the hands of other veterinarians, including those working under his own direction and failed to secure the confidence of the stock-owners. It is claimed that the inoculation is harmless, and cannot contribute to extend the infection, and yet we are told that the inoculated animal continues to bear the live ring-like parasites in its blood, and becomes susceptible.

PROTOZOAN ICTERO-HÆMATURIA IN SHEEP. PALUDISM OF SHEEP. CARCEAG. PIROPLASMOSIS.

This is described by Babes and Starcovici as prevailing among sheep in the delta of the Danube, and held by them to be identical with the Roumanian Hæmoglobinuria of cattle (compt. rend. de l' Acad. des Sciences, 1892). Its essential cause is a piroplasma affecting the red blood globules, and very analogous to that of the protozoön of Texas fever, but its especial election for the sheep shows a specific difference, inasmuch as the Texas cattle fever does not attack sheep. Not only the parasite, but the symptoms and lesions as well, furnish a close counterpart to those of the cattle infection. It remains to be seen whether the pathogenic difference is due to a distinction in the piroplasma or to the absence from the Southern States of America of the particular tick or other insect which attacks the Danubian sheep.

Bonome (1895, Virchow's Archives) describes the same disease as prevailing in Italy, describing the parasite and lesions at great length.

Finally my colleague Dr. W. L. Williams, and later Dr. Knowles, have identified the disease in the upper part of Deer Lodge Valley and the lower part of Silver Bow Valley in Montana, prevailing among sheep only, extending year by year, and proving disastrous to the sheep husbandry. Sheep were introduced into these valleys as early as 1875, but it was only in 1891 that the flock masters recognized the existence of this disease. By 1895 it prevailed over an area of 300 square miles. It made its advent in 1891 in four or five large flocks (2,000 to 10,000 head each) on land which they had occupied for nine years, and so disastrously that several sheep ranchers, after an experience of a year or two, sold out to the butcher and abandoned the sheep industry.

All or nearly all cases seen in 1896 were in parturient ewes, (4 to 6 days after parturition), the constitutional condition attending on lambing proving a most potent factor in causation.

The protozoön repeated the characters of that found in the sick sheep in Italy and the Danubian delta, and the conditions of

the blood and the structural lesions supported the idea of identity.

Altitude seems to have little or no effect as a causative factor, as the disease is domiciled alike on the low alluvium of the Danube and the Deer Lodge Valley of Montana over 5,000 feet above the ocean. In both regions there is the common condition of inundation or its equivalent irrigation, for the Montana range is dry and arid, interspersed with alkaline bogs inimical to vegetation, but prolific and fruitful under irrigation. The Montana disease has been attributed to mineral poisons carried on the winds from the extensive copper smelters in Butte and Anaconda, but the smelters had been in existence for eight or ten years before this disease was observed, and from its appearance the infection has gradually extended, attacking sheep only, and sparing other domestic animals, which would have suffered as well from a mere mineral poison on the vegetation. The doctrine of a mineral poison is equally contradicted by the habitual prevalence of the disease in spring and autumn, while it is dormant in winter and summer. In winter the flocks eat hay cut from the richer valley lands and meadows, while in summer they are pastured on the foothills and mountains, and drink from the mountain springs surrounded by alkaline bogs. The autumn outbreak occurs long after the mountain grasses have dried up, when the flocks are thrown back on the supplies obtained from the alkaline bogs and the valley pastures. In late winter and early spring the growth naturally starts first in the same boggy and valley areas, and both facts suggest a microbian infection—protozoan or bacterian. A vegetable poison would better accord with the conditions (See Cirrhosis of the Liver from Senecio. Vol. II.) If an intermediate host or bearer—insect or other invertebrate—is to be assumed it implies two generations of these, a spring and an autumn one, in the same season, or otherwise a restriction of such invertebrate to the low valley pastures and the alkaline bogs on the higher levels, and that they disappear from the drier, arid areas in summer. It cannot be an obligate parasite like a louse or melophagus which would be constantly present, nor a musquito absent in early spring. But up to the present no invertebrate host or intermediate bearer has been identified. Cases were at first reported in the Angora goat, but this animal is now known to be immune.

Microbiology. The parasite is formed in the red globules and blood serum of the affected sheep and closely resembles the microbe of Texas fever. In the blood globules the parasite is seen in different forms, round, oval, oblong or curved and from one-tenth to one-sixth the diameter of the red globule. A single red globule may show from one to four of the microörganisms. They may at times show indications of division, and at others, automatic amœboid movements, from one portion of the blood globule to another, or from the periphery toward the centre. The affected blood globules are usually enlarged, having lost their biconcave outline, and become biconvex or spherical, with irregular crenated surface, and a dull, lustreless appearance instead of a clear red or yellow aspect. The protozoön stains readily in anilin red or methylene blue.

Lesions. The condition of the carcass was good or even high, in sheep attacked when in good flesh, and in which the affection ran a rapid and fatal course. In sheep attacked while in low condition on the other hand, the case tended to be milder and more prolonged, and the body was emaciated and anæmic. Dropsical swellings were common on the ears and sides of the head and neck.

The skin, connective tissue, fat, and other normally white tissues were usually of a yellow color, varying from sulphur to lemon color. The muscles were pale and soft with a yellowish tinge.

The blood was pale, thin and watery, especially in protracted cases, formed a loose coagulum, or remained fluid with a grayish red color. There was marked leucocytosis (1:4 or 5).

The stomach and intestines were more or less icteric, and contained little ingesta.

The liver was congested, softened, shrunken in protracted cases, colored of a deep yellow especially in the interior, and with gorged biliary radicles so that the acini stood out very prominently. The gall bladder was usually well filled with a thick, flocculent bile, yellowish green, blackish green or chocolate color.

The spleen appeared shrunken, somewhat spherical, 2 to 3 ozs., firm, and with a dark, reddish brown pulp.

The kidneys were greatly enlarged, weighing 12 to 16 ozs.,

dark red or bluish black, friable, and on section exuding freely a bloody or chocolate-colored liquid. The capsule was easily detached. The bladder contained a bloody or chocolate-colored urine, but was sometimes empty.

The heart cavities were empty or contained small diffluent blood clots. Petechiæ were common on pericardium and endocardium.

Yellowish or yellowish green gelatinoid effusion was often present, not only on the head and neck, but also on the inner side of the thighs, and in one or other of the serous cavities.

Symptoms.. The first indications noticed are dulness, listlessness, a dragging behind the flock, ceasing to graze, arched back, and stiff or unsteady movements. There is moderate fever, yellowness of the visible mucosæ and skin, and rosy, bloody, or reddish brown urine.

Puffy dropsical swellings are noticed, especially on the ears, sides of the face, on the neck or thighs, and the patient lies down most of the time.

The patient usually dies in a state of collapse which has lasted for several hours, yet in certain cases it is preceded by a convulsive agony.

It is certain that the affection may appear in a mild form, as sheep slaughtered in apparent health are found to show the general icterus, the congested liver, and the leucocytosis with crenation of the red globules which characterize the disease. The icteric carcasses are said to be recognized and condemned at Kansas City and Omaha.

The *duration* of the affection is from 1 to 5 days, though it may last longer, and severe attacks usually end in death.

The *Prevention* of this disease can, as yet, be based only on the same principles that guide us in the case of Texas fever. Search should be made for an invertebrate host of the protozoön, by the extinction of which infection may be stopped. The spring outbreak could be opposed by feeding hay on safe ground until the higher pastures furnish sufficient vegetation. To counteract the autumn attack, the sheep might be fenced out from the alkaline bogs, and the forage supplied in the form of hay or soiling crops. If it should appear that any wild animals harbor or transmit the parasite, a campaign of extermination upon them would be in

order. If, as seems to have been the case in the early nineties, the movement of sheep from the infected flocks and pastures tends to cause the disease, this should be legally interdicted. Finally, the complete extermination of the sheep on infected areas could be practiced, and their places supplied by the immune Angora goat.

PALUDISM IN DOGS. MALIGNANT PROTOZOAN JAUNDICE. PIROPLASMOSIS.

Distribution: Senegal, Lyons, E. Africa, Paris, Pas de Calais, Cape Colony. Microbiology: piroplasma, differentiation from that of Texas fever, pathogenesis, tick-borne. Symptoms: incubation 3 to 5 days, dulness, prostration, apathy, drowsiness, anorexia, thirst, hyperthermia, icterus, hæmoglobinuria, offensive odor, emaciation, protozoön in globules, loss of globules. Death in collapse. Lesions: body shrunken, emaciated, fœtid; dark tissues, mahogany yellow, petechiæ, enlarged congested liver and spleen; muco-enteritis; bloody urine. Treatment unsatisfactory. Prevention: keep from tick infested land; clear and cultivate land; smear dog with insecticide ointment when hunting.

In certain malarial districts dogs suffer severely and even fatally from a febrile affection in which violent shivering is followed by great hyperthermia and yellowish or brownish red discoloration of the visible mucosæ. It was frequently attributed to malaria, and even sought to be identified with intermittent fever in man. Marchoux in 1899 studied this disease on the malarious seaboard of Senegal, and recognized the existence of hæmoglobinuria and the presence in the red globules of a hæmatozoön. Leblanc, about the same date, found a protozoön in the blood of dogs at Lyons, suffering from "red water," and Koch later found a double piroplasma in the red globules of suffering dogs in East Africa, both considering the disease analogous to Texas fever in cattle. Nocard and Almy in 1901, at Charenton, Paris, met with a similar affection in a dog which had just returned from a fox hunt, and was covered with ticks. Its urine was brownish red, like coffee grounds, and highly charged with albumen and hæmoglobin. Many of the red globules were affected and contained minute, spherical refrangent bodies which, when stained with carbolized thionine, appeared like those of

Texas fever. There were but 2,800,000 red globules in a cubic millimetre of blood.

The injection of 5 grammes of the blood into the jugular of an aged bitch caused in the 3d day hyperthermia (105° F.) and an extensive invasion of the red globules which still counted 6,100,000 per mm. On the 4th day the animal was thoroughly prostrated, refused to eat, had hæmoglobinuria, and many individual red globules enclosed from 4 to 8 hæmatozoa each. On the 5th day the count of the red globules was about 4,400,000 per mm. By the 6th day the urine was nearly normal, and appetite returned, but the red globules counted but 3,500,000 per mm. Menveux went to Pas de Calais with an Irish setter to hunt rabbits. The dog came in every night covered with ticks. In 5 days he sickened, with extreme prostration, diarrhœa and red urine and died on the 25th day.

Wm. Robertson describes the disease in Cape Colony. The piroplasma was first identified at Grahamstown by Purves and successfully inoculated by Spreul. By intravenous inoculation symptoms appeared on the fourth day and death followed about the sixth, while by subcutaneous inoculation the course was slower and the animal died about the eleventh day. Robertson inoculating from animal to animal carried it through a succession of thirteen dogs, all of which perished. He found the blood to be infecting on the third day after inoculation subcutem, and the globules invaded on the fourth, They were especially numerous in blood taken from the spleen.

Microbiology. The protozoön resembles that of Texas fever, but is larger, longer, less pyriform, and more like an oat seed, staining slightly in the centre which shows one clear spot, and more deeply at two or three places in the margin. It stains well with carbol thionin blue or methylene blue, but not readily with hæmatoxylin. The dog's blood, containing abundance of parasites, failed to infect horse, sheep, ox, cat, fowl, Guinea pig, rabbit, rat or mouse (Robertson).

Robertson found the ticks on every affected dog which had contracted the disease by simple exposure and which came under his observation. Specimens sent to Neuman were identified as Hæmaphysalis Leachi. Mature ticks from an infected dog were

preserved and their eggs hatched in an incubator. The resultant six legged larvæ, placed on susceptible puppies a month old proved harmless and no protozoa appeared in their blood. This was repeated when the same generation of ticks had reached the stage of nympha, and it was after two months from the hatching that the ticks, now mature, when placed on the same puppies conveyed the disease. On the thirteenth day the temperature reached 105° F., and the red globules swarmed with parasites, many single globules containing no fewer than eight. They were of various shapes, spherical, pyriform or cloveshaped many tapering finely toward the ends like an oat. The puppies died respectively 14 and 18 days after infection.

Experiments made on other dogs with the larval and mature ticks, fully confirmed the conclusion that the immature insect was harmless. A 14 days old puppy infected by mature ticks died on the 11th day so that the immunity of the other puppies cannot be due to a milk diet, as in the case of calves and Texas fever. Intravenous inoculation with the infected blood invariably conveyed the disease. Even mature ticks grown from eggs of those fed on infected and sick dogs are not always infecting. All the eggs do not receive the piroplasma. Jackals are exempt. (Lounsbury).

Symptoms. On the third day after inoculation the dog is dull, prostrate, apathetic and drowsy, refuses food and shows thirst. Temperature may reach 103° to 106° F. On the fourth day the mucosæ assume a yellowish tinge, and by the fifth this has encreased to a deep chrome yellow, which involves any white portions of the skin as well. Hæmoglobinuria is now well developed, the liquid being often as dark as claret, and the patient may lie perfectly prostrate, giving off an offensive odor from the skin, lungs, and especially from the mouth. The tongue is furred, the teeth dirty, and the gums may be congested or even ulcerated. Emaciation advances rapidly. The temperature may oscillate from day to day or it may rise steadily to a climax, and then descend suddenly when collapse occurs. In all cases the protozoon is found in the red globules, or free in the blood. In the worst cases the red globules may be so reduced in number that they can scarcely be found. Death comes usually by collapse. In some instances the hæmoglobinuria may be absent yet the disease advances to a fatal result.

Lesions. The carcass is usually shrunken and emaciated and exhales a fœtid odor. The mucosæ, white skin, and all naturally white structures (fat, connective tissue, fascia, tendons, ligaments, brain, spinal cord, etc.), are stained of a deep yellow. The muscles, liver and other darker tissues are of a mahogany yellow; petchiæ appear on the heart and serosæ; the liver is greatly enlarged and friable (10 lbs.) the spleen is swollen, gorged with blood and a soft, black, bloody pulp; the stomach and small intestines are empty, yellow and sometimes congested. The large intestines show mucoenteritis throughout with an abundant rusty red exudate. The kidneys are yellowish with cortex somewhat pale. The bladder, also yellow, contains dark colored urine. The red blood globules are greatly diminished in number, many are crenated, broken-up and distorted and they contain the piroplasma in large numbers.

No system of *treatment* has proved successful. Essays would naturally be made with quinia and other antiperiodics.

Prevention would naturally be sought in keeping dogs off from the uncultivated land and brush during the tick season (summer, autumn), in clearing and cultivating the tick infested pastures, in drainage, or in smearing the coat of the dog with the oil of tar liniment or other insecticide when he goes hunting.

PALUDISM IN HORSES: PIROPLASMOSIS EQUI.

Alleged identity with ague. Geographical distribution. Points of difference from ague. Causes: low, damp, undrained, inundated localities, hot seasons; inoculation, congenital. Symptoms: res less, drowsy, stiff, shivering, hyperthermia, tremors, cough, frothy, rusty expectoration, excited breathing and pulse, anorexia; puffy, petechiated eyelids; epiphora; dyspnœa; albuminous, yellow or red urine with casts: hæmoglobinæmia: colics, constipation, fœtid diarrhœa Death in a few hours to 6 days, or months. Lesions: reduction in blood globules, crenation, watery blood: petechiæ: enlarged, blood-gorged liver and spleen; congested, swollen, softened, kidneys; congested lungs with extravasations: in chronic—anæmia, dropsies, lung hepitization and suppuration. Prevention: keep susceptible horses from low, infected lands from June to November and from their water: protect from insect enemies. Treatment: quinia sulphate or bromide, hot baths, etc.

Cadeac and others describe an intermittent or remittent febrile affection of the horse, as identical with ague, and due, they mistakenly allege, to the presence in the blood of the *plasmodium malariæ* of Laveran.

Geographical Distribution. It has been observed on the low marshy grounds of Sicily, the Danube bottom lands, Algiers, S. Africa, Tonkin, Madagascar, Soudan, Senegal and Cochin China (Dupuy Lenoir, Pierre, Colin), Punjaub (Eassie,) Philippines (Gelston.)

Microbiology. The protozoan microbe, *Piroplasma equi* (Laveran) is more closely related to the *P. bigeminum* of Texas fever than to the Plasmodium malariæ of man. It is conveyed by ticks from one equine animal to another, rather than by anopheles. It is round or crescent shaped at different stages, 0.5 to 2μ, and is found in abundance in the red globules during the access of hyperthermia, and disappears in great measure during a remission. Yet the blood remains infecting even in the recovered ("salted") animals.

Accessory Causes. Like malarial diseases in man, this is largely confined to low, damp, undrained and inundated localities or those covered by trees or brush, and is most prevalent in the hot season, though it is not, like *horse sickness*, arrested by frost. The elevated plains and tablelands which are habitually dry, or well drained are exempt, and their soliped herds susceptible, while those of the lowlands are mostly immune. The mortality in Algerian horses taken to the Soudan is 90 per cent., while but 25 to 35 of the native horses of the Soudan or Senegal suffer. In S. Africa the coast horses generally bore the parasite, and were not successfully inoculated even with the blood of the sick injected intravenously, while those from the Karoo nearly all proved free from the piroplasma and susceptible to it. Injection into the veins or subcutem, of Algerian, American, or Karoo horses, of 20cc. of the blood from a sick horse, ass or mule, produces in 24 hours hyperthermia and parasites in the blood which last for 3 days (Edington), till fever subsides. It has even been claimed that it has been transmitted to the fœtus in utero.

Exposure in the hot sunshine on the open *veldt* aggravates an attack and even rouses a latent case to a dangerous or fatal form, while shelter in an open shed leads to immediate improvement (Eassie). Relapses or second attacks are often due to heat.

Symptoms. Premonitory symptoms of restlessness, drowsiness, or stiffness are followed by violent shivering, elevation of temperature (104° or 108° F.), anorexia, muscular tremors, rapid breathing, hacking cough with expectoration of frothy mucus, tumultuous heart beats, and small irritable pulse. There is complete anorexia, an opaque, infiltrated, petechiated, icteric or mahogany-colored conjunctiva, epiphora, and sometimes blood extravasations into the vitreous. The lungs may become intensely congested, with rapid, panting breathing, dyspnœa, a frothy, rusty expectoration, and extended head and limbs. This may prove fatal in a few hours. Otherwise there may be remisssions of the fever and dyspnœa at somewhat irregular intervals. Sooner or later are observed urinary changes, the liquid becomes albuminous, yellow, amber colored or red, or it shows distinct casts. These indicate the destruction of the red globules and the escape of hæmaglobin. In other cases there are slight colics and constipation alternating with a greenish yellow fœtid diarrhœa. The early nervous prostration and drowsiness may merge into vertigo, coma or paralysis. Vertigo is a very prominent feature in the Philippine cases (Gelston). The skin which, at first, may often be pricked without response, sometimes becomes tender, itchy and congested, with the erection of the hairs and the formation of pustules, or small abscesses llke hazlenuts. There may be œdematous swellings on the sides of the neck or in the sapraorbital cavities.

Course. Duration. In very acute cases death may take place in a few hours. More commonly illness lasts from three to six days. In certain instances it becomes chronic and may last two or even three months, the early congestion of the mucosæ giving place to pallor and anæmia with advancing emaciation, dropsies and finally marasmus and death, or recovery with immunity.

Lesions. These are mainly in the blood, red globules being distorted, crenated, massed in clusters and greatly diminished in numbers from (7,000,000 to 2,300,000) so that the liquid appears thin and watery. The mucosæ, internal organs and serosæ are petechiated and the serous cavities contain a yellowish serum. The muscles are mahogany colored. A yellowish tint pervades the white tissues generally. The softened, swollen, œdematous condition of the lymph gland is characteristic. The liver is

congested, virtually gorged with blood, enlarged and yellow or yellowish brown. The spleen is greatly enlarged, blood gorged, and shows irregular, rounded swellings indicating the seats of extravasation of blood. In some instances rupture has taken place. The kidneys are congested, enlarged, softened and of a brownish red or black color, with circumscribed extravasations especially in the cortical area. The lungs are violently congested, with many areas of blood extravasation, and they do not collapse when the chest is opened. The bronchia are filled with a white froth. The heart is petechiated, with a parboiled aspect and shows areas of commencing necrosis or fatty degeneration.

In the chronic form the watery condition of the blood is remarkable, the serous cavities (peritoneum, pleuræ, pericardium, arachnoid) contain considerable effusion, dropsical conditions of the neck, head, limbs and dependent parts of the body are common, the lungs show hepatization and minute centres of suppuration, and other viscera may show fibroid degeneration.

Prevention. It is advisable to keep susceptible horses from the low marshy and tick-infested lands from June to November and to avoid especially water that is drawn from such lands. It is not needful to take the stock to any very marked elevation provided the land is dry and free from wet or swampy areas and ticks. The native horses or those that have been long in the marshy district and have thus secured a partial immunity may profitably replace the more recently imported and susceptible horses during the dangerous summer months.

Edington by injecting, intravenously, the blood of an immune horse or preferably ass, produced in 27 out of 50 the characteristic fever, and on recovery they proved immune. A 2nd inoculation produced the reaction in cases where the 1st failed. This is inapplicable to horses already affected (even recovered—*salted*—horses), and those under treatment should be sheltered from the summer sun. Any intercurrent disease like *horse sickness* should also forbid immunization, as either or both may be aggravated.

To destroy ticks use similar measures to those advised for Texas fever.

Treatment. Pierre employed quinia sulphate or bromide 45 grs. in distilled water 1 oz., intravenously, Gelston, creolin, subcutem.

Hot baths, 95° to 100° F. are claimed to lower the temperature and contribute to the arrest of an attack. Other complications are treated according to indications.

SURRA : ROT.

Synonyms. Definition : Acute protozoan remittent fever, of rainy season or after, with destruction of red globules, anæmia, emaciation, dropsy, icterus and cutaneous or mucous eruption or discharge. Distribution : India, Burma, Cochin China, Persia, Philippines, etc. Trypanosoma Evansi: a fusiform flagellate infusorian with undulating membrane, attacking the red globules : agency of flies as carriers, infection of dogs ; of cows : feeding, licking ; rats ; open sores; stables ; yards ; pastures ; pickets ; stagnant water ; manure or rubbish heaps ; abattoirs. Lesions : anæmia ; trypanosoma swarms during relapses ; leucocytosis ; œdemas ; effusions ; blood extravasations ; splenic enlargement ; emaciation ; gastric and intestinal congestions : gastric ulcers ; kidneys petechiated, swollen : brain effusion. Incubation, 2 to 8 days. Symptoms : local swelling, 2 to 4 inches by 4th day ; decreases to 14th day ; the general symptoms : hyperthermia 102° to 104° F., night highest ; dulness ; sluggishness ; inappetence ; icterus ; cutaneous eruption ; remission in 1 or 2 days ; exacerbation after 3 to 10 days ; catarrhs ; petechiæ ; stocked legs ; pectoral swelling ; encreasing anæmia and emaciation ; ulcers on mucosa or skin ; generative excitement ; fœtid diarrhœa ; urine profuse, bilious, albuminous or with casts ; thirst ; intestinal rumbling ; debility ; marasmus ; hair erect ; skin dry, rigid. Duration variable. Diagnosis : based on above, confirmed by finding blood parasites, constantly fatal in horses. Surra in camel, ox, buffalo, rabbits, rats, mice, dogs, cats, apes. Treatment : unpromising ; mercurials, iodides, chromates, arsenates, terebinthinate, phenic acid, santonin. Dry, clean, airy stable, dry grain, tonics. Prevention : keep horse and mule from infecting locality ; use oxen rather : stables, pickets, etc., apart from marshes, pools, manure and rubbish heaps ; disinfectants, insecticides, seclude all surra-affected animals : smudges. Sanitary police.

Synonyms. Sar, Zahrbad, Gumzahrbad, Kushkzaharbad, Sokra, Sokhra, Tap, Tapdik (Punjab) : Phitgya, Purana (Meerut) : Berbag (Bombay) : Tarai, Tebersa, Wabai-ki-bokhar, Pernicious Anæmia, Trypanosomosis, Relapsing Fever, etc.

Definition. An acute, relapsing, protozoan fever of equines, camels and elephants, inoculable on other animals, occurring during or after the rainy season, and characterized by hyperthermia which is liable to be intermittent, remittent or relapsing,

anasarcous, swellings, petechiæ of the mucosæ, icterus, cutaneous eruption, nasal, ophthalmic, vaginal and other mucous discharges, rapidly advancing anæmia, emaciation and debility, and above all, by the presence in the blood, at intervals from one to six days, of swarms of protozoa, analogous to those found in dourine or nagana.

Geographical Distribution. Surra has long been known to the English veterinarians in India, occurring during or just after the rainy season, and especially on the low flooded lands, along canals, rivers, lakes, etc., and later in Burma, Cochin China, the Persian Gulf, Persia. Lingard claims its existence in East Africa, North and South America, Australia and Southern Europe, but he has evidantly confounded it with nagana and other affections. The discovery of the disease by Dr. Slee in 1901, among American, Australian and Chinese ponies in the Philippines is suggestive of a very wide diffusion of the infection in Southern Asia and adjacent islands, with which an American work on veterinary medicine must deal.

Cause. Parasite. Trypanosoma Evansi. The essential cause of the disease, Trypanosoma Evansi, discovered by Dr. Griffith Evans, Inspecting Veterinary Surgeon, British Army, in 1880, is a flagellate infusorian, pointed at one end, near which is a dark centrosome, and from this a flagellum running along the free border of the broad undulating membrane to the extreme opposite end of the parasite and extended beyond this as a waving lash. The length of the Trypanosoma Evansi is 20 to 50μ, (10 to 14μ, Smith and Kinyoun, Manila), its breadth 1 to 1.5μ. By reason of its large size and active motion it is easily detected in a film of fresh blood under $\frac{1}{6}$th inch objective, and no less easily when dried and stained on a cover glass. It must be borne in mind that the mature parasite appears in the blood at intervals in swarms, so that examination at one time of day, or on a particular day, may fail to detect it, while examinations made earlier or later are successful. The general structure and successive stages of growth of the parasite appear to be the same as described for the Trypanosoma Equiperdum of Dourine, to which accordingly the reader may turn for description. The distinction from that parasite is to be found mainly in the pathogenesis. In this respect it should be noted that the parasites are strongly at-

tracted by the red globules, upon which they fasten themselves by the blunt ends, shaking the cell in the most vigorous manner and even breaking pieces off and carrying them away. They are most strongly attracted by the concave part of the disc, and when there are rouleaux they will bore between the globules and even push them apart (Evans).

The appearance of the disease at the conclusion of the rainy season when the waters dry up and become foul, has led to the idea that the parasite lives in waters, but as this is also the time of the great swarming and activity of flies, and as the trypanosoma is found in the bodies of tabanidæ and hippoboscidæ that have bitten affected animals (Lingard), and as horses crowded together so that the fly with piercing apparatus still wet can pass from horse to horse (Evans), the opinion has grown that it is a compulsory parasite which is transmitted through the bites of insects. In 1880, Griffith Evans found that the native Hindoos attributed the disease to the bites of a very large brown fly which was active in July (probably a tabanus), in 1897, Pease identified the incriminated fly as the tabanus tropicus. Finally, Rodgers, in 1901, took flies that had been on surra horses, kept them 4 days or longer and found that their bites failed to produce surra; whereas those that were allowed to go directly from the sick to the healthy animal produced the disease in the latter. The direct experimental inoculation from horse to horse infallibly conveys the disease so that the flies are not needed to pass the parasite through an intermediate stage of its existence, but merely to carry it. It follows that no particular fly is the bearer but any insect may carry the infection from a bite or sore to inoculate it on a sore or by a bite on a fresh animal. Different observers have noticed the tendency to the infection of dogs and other animals that fed from rubbish heaps, or upon the carcasses of animals dead of surra, suggesting at once the intervention of the swarms of flies that congregate at such places. This is probably another example of the shrewd insight of the common mind, as in the case of the tick-borne Texas fever.

Lingard finds another bearer in the crow which sits on the backs of affected horses, pecking at the wounds, and passes at once to other healthy horses to peck their sores.

Experiments in feeding the infested blood to sound animals,

have apparently succeeded, and the observation that dogs and cats suffer from eating the carcasses is in favor of this view. Horses that lick the infested sores, or the blood drawn by the flies may readily infect themselves, and especially if the mouth bears scratches caused by fibrous food, leech bites, or sores from bits, or if the pharynx or stomach has been wounded by bots or spiroptera.

The water and food are blamed by the natives in some quarters, but Pease's observations on the Bombay tramway horses, which all perished though kept on boiled water and carefully picked fodder from sound regions, would suggest that this if a channel of infection at all, is not the main one.

The bowel excretions of rats harboring trypanosoma, when mixed experimentally with the food of the horse, have been charged with causing surra, but there are objections to the acceptance of this as a common cause. The alleged period of incubation in the horse in such cases was 40 days in place of the usual 7 or 8 days; when inoculated from a horse first affected in this experiment on a second the usual incubation of 7 or 8 days was shown, and though the horse fed on rat's dung in the infected region of Bombay contracted the disease, the experiment failed when the same dung was fed in a high dry region unaffected by surra. The natural inference is that Bombay experimental horses contracted the affection in the usual way, probably through insects.

In the rainy season the Indian rats swarm with the Trypanosoma Lewisi, an entirely different species, and though they can be successfully inoculated with the T. Evansi of Surra, the T. Equiperdum of Dourine, the T. Brucei of Nagana, and it is alleged the T. Equinum of Mal de Caderas, yet these are not their common parasite. The presumption is that the rat affected with Trypanosoma Evansi could transmit the disease to the horse through one of the many possible insect channels or otherwise.

Neither condition, sex nor age appears to affect receptivity. Open sores especially open the way for infection.

The position of stables, yards, pastures or picket grounds near stagnant water, manure or rubbish heaps, abattoirs or other places that breed or attract flies, is a much more important consideration. Foul stables, or those having light from both sides are more exposed to flies.

Pathology and Lesions. The pathology of the disease consists in the rapid destruction of the red blood globules by the trypanosomata. It is a form of rapidly advancing pernicious anæmia due to the great and active voracity of the trypanosoma. The swarming of the trypanosoma in the blood at the period of the relapse and the absence of the mature form in the intervals is remarkable. The trembling movement in the blood at the period of swarming results from their prodigiously active movements. The red globules may assume various forms, crenate, echinated, (Ranking), and more or less broken up or disintegrated, their numbers steadily decrease, leaving the blood thin and watery with rusty serum and yellow (icteric) staining of the white tissues, even the bones. Encrease of white globules, actual or relative, has been a marked feature (always present in typical cases, Burke).

Petechiæ are especially common on the conjunctiva, vaginal mucosa, endocardium, and less marked in the nose, mouth, and serosæ.

Œdemas are common, yellowish, gelatinoid exudates at the base of the heart, subcutem, between the muscles of the limbs or elsewhere, and as effusions into the pleuræ pericardium or peritoneum.

Distinct blood extravasations have been noted beneath the endocardium.

The spleen is often enlarged, excessively so if death occurred during a paroxysm. The lymph glands are swollen and appear dropsical.

The whole body is emaciated and shrunken, the visible (unpetechiated) mucosæ are pale and bloodless, often yellow, yet rigor mortis is well marked.

Gastric ulcers are common (Steel, Burke, G. W. Evans) apparently preceded in many cases by capillary embolism congestion and degeneration. Intestinal congestions are frequent (Steele, Burke), but ulcers are rare (Geo. H. Evans).

The liver may be normal (Griffith Evans), or especially during a paroxysm (Lingard) congested, inflamed and enlarged. The pancreas is usually normal.

The kidneys are petechiated, congested, œdematous, or the seat of blood extravasations.

Instances have been noted of cerebral and meningeal œdema

(Steel, Lingard), encrease of fluid in the arachnoid or lateral ventricles, or in the spinal cord, with gelatinoid exudation in the lumbar portion (Lingard), Like other tissues, in advanced stages the nerve centres are usually anæmic (Geo. H. Evans).

Ineubation. As made out by inoculation cases this extends from two to eight days, according to the dose—five to seven days being the most common. Infection by inoculation subcutem in mules showed a hyperthermia on the fifth day; or by ingestion (feeding the virus) on the sixth day (Steel). Longer (alleged) incubation depends mainly on the first slight paroxysms having been overlooked, or set down for the frequent bilious or icteric condition which is common in mules and horses in India (Steel), or to a later infection by insects or otherwise.

Symptoms. In experimental cases a small raised swelling in the seat of the inoculation, appears in 24 hours, encreasing to 2 to 4 inches in diameter, and 1 to 1½ inch high by the 4th day, and loosely connected with the parts beneath. From the 4th to the 14th day it decreases in size and softens and general symptoms set in.

In casual cases these general symptoms are the first to be observed. There is transient fever 102° to 104°, highest toward night and without preliminary chill, hot mouth and skin, dulness, sluggishness, inappetence, yellowness of the mucosæ, petechiæ on conjunctiva or vulva, and sometimes nodules like those of urticaria on the skin. After a day or two these symptoms subside, the temperature is 101° F., or below, the mucosæ clear and pale, and spirit and appetite nearly normal. These slight first paroxysms are rarely seen by the veterinarian, having been looked upon as one of the oft-recurring bilious attacks of a hot climate. The remission lasts for 3 to 10 days when the second paroxysm sets in, like the first but often more marked; temperature 102° to 104°, eyes especially the membrana nictitans petechiated, epiphora, slight catarrh from nose or vulva, it may be stocking of the legs, or pitting swelling under the breast bone, or abdomen, or in the sheath. Like the first, the second paroxysm subsides, and after another interval a third sets in, to be followed in like manner by a fourth, a fifth and so on, if the patient survives. With each the symptoms become more pronounced, the mucosæ are left more pale and bloodless, debility and weakness

are greater, emaciation is more marked, œdema of the limbs or body more extensive, hyperthermia may reach 105° or more, the pulse is weaker and the heart more liable to palpitation, and the respirations may reach 50 or 60 per minute. Yet in sparely built animals dropsy may be entirely absent (Steel). Steel often found superficial, circumscribed ulcers on the tongue, inner sides of the lips, nose, eye, or vulva, beginning as epithelial degeneration, followed by superficial erosion and early healing. Sometimes, similar erosions appeared on the skin. Generative excitement may be present, the mare appears to be in heat, while the horse has erections, which are supposed to depend directly on the implication of the generative centre in the lumbar myelon. The submaxillary glands sometimes swell and even suppurate and discharge a gluey pus (Griffith Evans). The bowels are usually costive at first, the fæces may be glazed, but in advanced stages they become soft, pultaceous and fœtid. The urine at first normal in amount, becomes later abundant or even profuse (Griffith Evans, Ranking, Nariman and Vaz, Lingard). It is at first yellow and turbid, later of a dingy green or greenish yellow. Sometimes it diminishes as the disease advances. It may contain bile, albumen, or even casts (G. H. Evans), though the latter appear to be exceptional. The reaction varies, sugar is absent, and the parasite has not been found in it. Appetite though interfered with during the paroxysms, remains fair or even voracious in the intervals and the animals may eat to the last (Burke). Thirst usually encreases with the advance of the disease, in keeping with the free urinary secretion. Rumbling and gurgling of the bowels are common and even tympany at times (Lingard).

In the advanced stages the picture is one of great anæmia, marasmus and general debility. When moved the animal will stumble over the slightest obstacle, even the litter, recovering himself with effort and difficulty. If he should fall he is liable to remain down indefinitely, the side next the ground becoming drenched with sweat though there is no general perspiration. The hair becomes encreasingly dry, withered and erect, the skin dry, powdery, rigid and more and more firmly adherent to the bones and muscles, losing all its natural pliancy and mellowness, and becoming like that of a dead animal. It is bloodless, and

sloughs readily over the prominent bones, where compressed or bruised in lying, owing to the lack of nutritive and reparatory action. The visible mucosæ are absolutely bloodless. The muscles as a whole are wasted to an extreme degree, but this atrophy is most marked in the back and loins, along the longissimus dorsi and in the quarter in the gluteal muscles. The patient may remain recumbent, from sheer weakness, for a length of time at the last, or he may get up after a long recumbency and stand to the end.

Death may occur early with general anasarca and extreme hyperthermia (110° F.). In the great majority it appears to result largely from perforation of the stomach, clots in the heart, or general debility and heart failure.

Diagnosis. With symptoms such as are above described the discovery of the trypanosoma in the blood completes the diagnosis. Trypanosoma is found in dourine, nagana and other affections so that the discovery of it alone would not be conclusive as to the existence of surra. Nor can the discovery of the parasite always be made at the first or second attempt. The swarm of mature trypanosomata is found with the advent of a paroxysm, and as the veterinarian is often called during the decline of the attack the parasites have already retired and elude his investigation. It becomes needful to take the temperature and examine the blood daily sometimes for eight or ten days, and when with a sudden rise of temperature he finds also a swarming of the mature trypanosomata, the diagnosis is perfect. A drop of blood placed on a cover glass, pressed down upon the slide, and placed under the microscope, will show the parasite with eel-like movements among the blood globules. There may be very few during the first or second paroxysm, but they become numerous and very obvious as the disease gains its height. Evans recommends to use defibrinated blood.

They may be dried rapidly on a cover glass, fixed in absolute alcohol one or two minutes, then stained ten minutes in a mixture of the two following liquids united just before using.

A.—ONE PART OF THE MIXTURE.

Methylene blue	1 Grams.
Borax	25 ,,
Distilled water	100 ,,

B.—FOUR PARTS OF THE MIXTURE.

Eosin	.1 Grams
Water	100 ,,

The plasma of the trypanosoma appears blue, the flagellum red and the chromatin of a different shade of red. It may be permanently mounted in balsam.

Mortality. The disease is constantly fatal in horses, though the animal may survive for months.

SURRA IN CAMELS.

Haggar describes this as having remissions or intermissions as in the horse, the trypanosoma disappearing during the intervals to appear with the hyperthermia. The temperature may rise to 106° F. and the animal wastes away to a veritable skeleton. A remarkable feature of the disease is the formation of immense abscesses containing a thick, cream-colored pus on the sides of the chest in the vicinity of the pad, and in the sheath and scrotum or udder near to the stifle pad. The mortality is nearly as great as in the horse or mule, yet the camel drivers say that a small percentage recover.

SURRA IN BOVINES.

The domestic ox, the sacred cow of India (Lingard) and the water buffalo (caribao) of the Philippines contract Surra. It is interesting to notice that in these animals the disease is relatively mild and recoveries are frequent.

In the Indian buffalo it causes dulness ; advancing emaciation ; slight temperature variations ; muco-purulent inflammations of the conjunctiva, cornea, and pituita ; and occasionally eruptions under the breast or belly.

Among *lesions* were atrophy, softening of lymph glands, enlarged liver and spleen (slight), petechiæ and blood extravasations on the pericardium, epicardium and other serosæ, and on the intestinal mucosa which sheds its epithelium in patches.

Stall enzoötics in cattle do not seem to affect horses casually. The disease is easily conveyed to rabbits, house rats and mice, dogs, cats and apes. Goats and ducks appeared to be immune (Penning).

TREATMENT OF SURRA.

In well established cases in the horse, ass or mule no treatment has succeeded in saving the patient. Almost every germicide has been called into requisition but without good effect. Among these may be named: mercuric chloride subcutem, iodine and potassium iodide intratracheally, iodoform subcutem and intravenously, oleum terebinthinæ subcutem and intravenously, potassæ bichromas by veins and stomach, cinchona alkaloids and arsenic, phenic acid and iodine, quinine, hydrargyri biniodidum, santonin, potash. The claims of different agents, notably arsenic and phenic acid, have been supported by the manifest improvement of condition under their use and the disappearance of the trypanosoma, but both these conditions may often appear during intermissions, without medication. The usual outcome is that the animal dies and the only claim that can be made is a slight extension of life. This is favored by dry, clean, airy stables, change of water, rich grain and succulent food (oats, rice, linseed, barley, gram sorghum, bran, middlings, salt, etc.), with iron, arsenic, and other tonics, yet the best that can be said for them is that they have deferred somewhat the enevitable death.

Prevention of Surra. The first consideration is to avoid placing equines and especially horses and mules, or camels in the infecting localities in the rainy season and just after it. Oxen and buffaloes can be used in such places with greater impunity. If they must be used in such localities, place the stables or pickets in dry locations well apart from marshes, and stagnant water. keep the stables dark during the surra season, open to light on one side only and with screens in the windows. Make a deep pit for the manure, keep it well darkened and screened and turn every particle of manure into this several times a day so that the stable shall be perfectly clean. All rubbish heaps should be similarly dealt with. The flies breed in the manure and decaying organic matter.. After each sweeping of the stable sprinkle the manure in the pit with some disinfectant, phenic acid, tar water,

copperas, etc. Smear the skins of the animals with tar water, coal tar water, naphthalin, daily if necessary to prevent the attacks of the flies. Other suggestions in this line can be found under *parasites*.

An important consideration would be to seclude every animal attacked with surra. The flies can only carry and inoculate the poison when there is some source from which they can obtain it. Until we shall learn how many animals, tame and wild, casually contract the Trypanosoma Evansi we cannot speak of how effective this may be made, but it is at least a substantial advance in the line of restriction, since the infected horse or mule in the vicinity of healthy ones is a constant peril, and as given insects attack by preference, given favorite genera, the horse flies, coming from the diseased animal are much more likely than other flies to attack the sound horses. In a surra season it would be a wise economy to destroy the infected equine at once, as according to all past experience, sentence of death has already been passed upon him, and his preservation even for an hour is hopeless for him, but full of the gravest danger for others. The carcass and all pertaining to it, blood especially, should be promptly and deeply buried and the place thoroughly disinfected.

In the same way smudges made by burning green grass or other vegetation, tar, leather or other material producing empyreumatic products offensive to the fly may be employed.

Sanitary Police. The Department of Agriculture forbids the landing in the United States or its dependencies of any animal from the Phillipines. If the infection should by any accident be imported no cost should be considered too high to secure a prompt and thorough extinction of it.

NAGANA. TSETSE FLY DISEASE.

Trypanosoma Brucii: attacks horse, ass, mule, cattle, buffalo, antelope, camel, hyena, dogs, etc. Elephants and zebra, pigeon and hen immune. Tsetse fly. Inoculation only certain channel; virulence in dead body 24 hours; in vitro 3 to 4 days, or when dried or heated (122° F.) Symptoms: hyperthermia; anæmia; leucocytosis; buffy coat; œdemas; catarrhs; wasting; debility. Lesions: as in surra; trypanosoma in blood at intervals, bone marrow, lymph glands, spleen. Immunizing unsuccessful. Prevention as in surra.

This is an infective disease caused by the *Trypanosoma Brucii*, which has been supposed to be identical with *Trypanosoma Evansi* but differs in its morphology and in its infectiveness toward a greater variety of animals. It is 25 to 30μ long by 1.5 to 2.5μ broad, less pointed at its posterior (nonflagellate) end, broader undulating membrane, more deeply staining protoplasm, and more sluggish movements. It invades the blood of horses, asses, mules, cattle, buffaloes, antelopes, camels, hyenas, and dogs, and can be inoculated on cats, rats, mice, rabbits, hedgehog, boshbok, zebra hybrids, Guinea pigs, goats, sheep, weasel, and monkey (Macacus rhesus). Elephants are immune though they suffer from T. Evansi, and the zebra is immune though a soliped.

The *tsetse fly* (glossina morsitans) is credited with being the main agent in transmitting the parasite to mammals, (see *Diptera*).

Kanthack, Durham and Blandford found that Guinea pigs were more resistant than rabbits, surviving the inoculation for a longer time. Bruce also found the native South African sheep and goats more refractory than others, yet their hæmatozoön was as deadly to other animals as that of horse or dog. Pigeons and South African hens proved refractory. Manifestly in Africa the present races of some animals are the products of the survival of the fittest.

Inoculation. The disease has been produced experimentally by inoculation only. Feeding experiments on rabbits, cats and Guinea pigs, the sucking by puppies of an infected dam for 14 days, coitus, and even the careful dropping of infected blood under the eyelids failed to convey it. Lousy rats with sores on face, one cat doubtless scratched by a bone, and one rabbit supposed to be infected by sexual congress (Rouget) offer exceptions, which in the light of the general results must be looked on as probable inoculations through wounds.

Successful inoculations were made with blood, lymph gland, spleen, bone marrow, aqueous humor, serum, œdema fluid, and testicular juice. The effectiveness of inoculation did not vary materially with the different fluids used, with the amount, nor with the point selected for its insertion, subcutaneous, intravenous, intraperitoneal, or on a mere scratch.

Virulence is lost in the dead body in 24 hours or less; when the infected blood is kept aseptically *in vitro*, in 3 or 4 days at

most ; or when completely dried. Heating to 122° F. for 30 minutes sterilized, while at 114.8° F. for half an hour the trypanosoma became nonmotile, but not quite nonvirulent.

Symptoms. Hyperthermia is marked about the time of the appearance of the infusoria in the blood. (Horse 106.7° ; dog 104° ; rabbit 105.8°), and in the horse there was a paroxysm with each new swarm of the parasite. In one ass intermissions were not observed. In rabbits there was no constant ratio between the temperature and hæmatozoa, hyperthermia was constant or nearly so. The horse had marked hyperthermia up to death, dogs and bats showed a striking descent even to subnormal, before death.

Anæmia is a marked feature the red globules being greatly reduced, and they show a tendency to clump in masses instead of forming rouleaux. The serum added to healthy blood has the same effect on that. *Leucocytosis* is not constant nor excessive (15,000 to 34,000 per cmm. at the highest). In clotting the blood may form a buffy coat.

Œdema is common in horse, rabbit, cat and dog especially about the head, legs, belly and genital organs. Rabbits often show swellings, excoriations and sores of the labia, prepuce and penis favoring inoculation by this channel.

Conjunctivitis is common in cats, dogs, rats, rabbits, often in connection with facial œdema, and corneal opacities and ulcers, and turbidity of the aqueous humor may follow.

Nasal catarrh often interferes materially with breathing.

Muscular wasting and *debility* are prominent phenomena, especially in horse, dog, cat and rabbit, the loss of weight reaching 20 to 30 per cent.

Lesions. These are like those of surra. Enlargement of the lymph glands, spleen and liver, firmness, friability and dark color of the spleen ; effusions, petechiæ and even hæmorrhages of the serosæ, lungs and stomach, and great atrophy of the muscles and adipose tissue are prominent features. The liver is always fatty in rabbits. In the shafts of the long bones, the fat is replaced by red marrow. The bone marrow is sometimes red, at others pale.

The *trypanosomata* in the blood vary greatly. In infected rats and mice they appear 3 or 4 days after inoculation, are almost

constantly present thereafter, and as a rule, encrease steadily up to 2,000,000 or 3,000,000 per c.m. In dogs they appear in 4 to 6 days, in cats in 5, in Guinea pigs in 5 to 6, and in horses in 7 days. In rabbits there were found 60,000 per cmm., in dogs 100,000 to 300,000, and in Guinea pigs 200,000 to 500,000. In one Guinea pig they did not appear until 6 weeks after inoculation and then rapidly encreased to a fatal termination. More commonly a few can be found about a week after inoculation, and then they disappear for a variable period. They have been found in the bone marrow when they could not be recognized in the blood. Again, after subcutaneous flank inoculation in the rat, they were found in the corresponding inguinal glands 1 to 3 days before they could be detected in the blood. After death they are found most abundantly in the bone marrow and spleen, but they have not been found in the intestinal contents nor urine except in the case of hæmorrhages or local sores.

No soluble toxin appears to be formed, and *no immunization* is effected by the serum. Blood serum kept several days until the infusoria had died and then passed through a Berkefeld filter had no apparent effect on the animal economy even in large doses. Blood heated to 122° F. was equally harmless. The same is true of extracts of organs and of bile from infected animals.

Attempts to secure *immunity* by injecting the blood serum of affected animals, after it had been sterilized by heat, filtration, or standing one week *in vitro* proved of absolutely no effect. The blood of the fœtus almost at full term proved valueless, and the young born of infected mothers proved fully susceptible when inoculated. The serum of the Guinea-pig, which is naturally somewhat resistant, proved no protection to other animals. Bile mixed with infecting blood *in vitro*, kills the trypanosoma, but such blood has no protective effect on animals. Sewer and white rats inoculated and reinoculated with the common rat trypanasoma (T. Lewisi) until immune from that organism, show a full susceptibility to the trypanosoma of nagana.

Flesh feeding and *vegetable feeding* have made no difference in the susceptibility in the case of rats. Feeding with the hæmatozoa has produced no immunity.

It is evident that prevention must follow the same lines as in Surra, due consideration being had of the greater number of genera susceptible.

LUNG PLAGUE OF CATTLE.

Synonyms. Definition : infectious, cattle fever, with long incubation, insidious onset, excessive pulmonary exudation, infarctions and sequestra. History : ancient, modern ; England, Norway, Sweden, Denmark, United States,—its extinction in 1887 to 1892—Massachusetts, New Jersey, S Africa, Australia, Tasmania, New Zealand. Causes : contagion only. Bacteriology : Nocard's organism cultivated in collodion capsules *in vivo*. as actively motile refrangent points, morphology uncertain. Modes of transmission, exhalations from sick inhaled by susceptible, ingestion of infected food, pastures, watering troughs, ponds, commingling of herds, attendants, dealers' stables, manure, unrestricted commerce, convalescent cattle, breed, hot seasons. Lesions : profuse exudation in lymph net-works of lungs, marbling, parenchymatous cell proliferation, compression and grangrene of lobulets, thrombi, sequestra, infarction, fibroid sac, or stalactite like fibroid peribronchial formations, hepatization, pleural effusions and false membranes, pericardial, bronchial, lymph glands congested. Incubation : 6 to 30 days, protracted cases, bearing on quarantine. Symptoms : conditions affecting gravity, breed, excitement, heat, chill, susceptibility, usually insidious, infrequent cough, roused by cold water, dusty food, exertion, etc., hurried breathing when driven, slight auscultation râles, hyperthermia (103° to 108° F.), dulness, anorexia, suppression of milk, stiffness, no pandiculation, troubled breathing, auscultation, and percussion signs of extensive consolidation, tenderness of chest walls, in bad cases stands obstinately, head extended, mouth open, tongue protruded, grunts with expiration, heavy breath, nostrils dilated, nasal and buccal discharge, rapid emaciation, fœtid diarrhœa, erect hair, pale, scurffy, adherent skin, varied chest râles, abortion. Mild in winter, severe in summer. Loss of one-third or one-half weight in one week. Chronic cases, fibroid and necrotic changes, sequestra. Diagnosis : Anamnesis, inoculation : from *tuberculosis* by high fever, rapid, extensive infiltration, early succession of new cases, failure to react under tuberculin, absence of tubercles ; from *bronchitis* and *pulmonary strongylosis*, by the succession of cases in place of many at once, the extensive exudation, the evident cause (exposure or infected place), and by the lesions ; from *fibrinous pneumonia*, by absence of climatic cause, the more extensive consolidation, more troubled breathing, and coexistence of old and recent lesions : from *infectious pneumonia* by the greater area consolidated, more exalted hyperthermia, more marked dyspnœa, the absence of white points of alveolar cell proliferation, and by the old and recent lesions ; from *septicæmia* hæmorrhagica, by its occurrence at all seasons, on all soils, by the absence of sanguineous swellings at other parts, by the absence of coccobacillus from the exudate, and by its non-inoculability on sheep, horses, pigs and rodents ; from *needle in pericaridium*, by its epizootic prevalence, fever, and the absence of preliminary gastric and later cardiac, morbid phe-

nomena; from *emphysema* by the fever, absence of drum like tympany, and presence of consolidation; from *aspergillosis of the lung* by the fever, more rapid progress and extended consolidation, by the harmlessness to birds, by the absence of aspergillus from expectoration and lung; from *insolation* by the absence of the causes of heat stroke, of the implication at once of a number of exposed animals, of the excessive hyperthermia, the brain symptoms and sudden death. Treatment: a poor economy, to be punished when extinction is attempted. Prevention: importation under three months quarantine, surveillance of three months more in case of single animals; applied to lung plague countries and those adjoining; disinfect cars and boats that have been used in lung plague country; exclude hay and fodder from lung plague country; also litter, bags, head ropes, horns, hoofs, hides, hair. To extirpate: stop all ingress of possibly-infected cattle, stop movement of cattle, prohibit common or unfenced pasturage, kill without idemnity in case of violation, record all cattle, make necropsy of all dead, kill and pay for all infected herds, disinfect all infected premises and things, use horses in removing and plowing under manure. Unaided owner may: breed all his stock, in buying bull quarantine him, allow no intercourse between his herd and others, get new cattle from herds only that have had no illness nor losses for a year, and no exposure, and bring by thoroughly safe route, in disinfected cars or boats, then quarantine, under special attendants. Immunization; by a first attack, inoculation in tail, intravenous, of sterilized exudate. Conditions permitting and forbidding immunization.

Synonyms. Bovine Pleuro-Pneumonia, Contagious Pleuro-Pneumonia, Peripneumonie (Fr.), Lungenseuche Vieseuche (Ger.), Pneumosarcia, Peripneumonia Exudativa, Epizoötic Pleuro-Pneumonia, Pulmonary Murrain, Slijmlongziekte (Dutch) Pulmonea dei Bovina (Ital.), Phush phush pirdaho, Pheepree (Bengalee).

Definition. An infectious febrile disease, occurring casually in cattle only, so far as known, and characterized by a prolonged incubation (10 to 90 days, a tardy insidious onset, inflammation of the bronchia, lungs and pleuræ, a profuse exudation into the interlobular connective tissue and chest, a very extensive area of consolidation, pulmonary infarction, and sequestra.

History. It is difficult or impossible to identify this disease among the various animal plagues in ancient times. Yet when lesions are given it may be admitted in different cases. Aristotle, writing 350 years before Christ says "the cattle that live in herds are subject to a malady which causes the breathing to become hot and frequent. The ears droop and they cannot eat. They die rapidly and the lungs are found spoiled." Here the facts that

cattle alone suffered, that large herds suffered most, that the lungs were the seat of very marked lesions, and that fever and mortality were both marked, point forcibly toward lung plague. Valentini's description of a fatal lung disease in cattle also stands out prominently in the obscure records. The first full and definite report is that given by Bourgelat of its prevalence in Franche-Compté in 1769. Later the records are frequent and from all over Europe, indicating its general prevalence before as well as after Bourgelat.

Much more important are its definite extensions into new countries after a long interval of immunity as showing that with trustworthy records such invasions can always be satisfactorily accounted for by the introduction of cattle or their fresh products from a previously infected area. Into Holland it was imported from Germany in 1835 by Vanderbosch a Guelderland distiller and spread over the whole country. Attempts were made in Friesland, and at first successfully, to stamp it out by slaughter and disinfection, but the demands of the trade toward England grew so enormously that it was being constantly introduced anew and the measure was abandoned. The British Consul at the Hague, in 1839 sent some Dutch cows to a friend in County Cork, Ireland, which led to the general infection of that island and indirectly of Great Britian. In 1842, under the Free Trade Act, England became at once deluged with lung plague cattle from Holland, Belgium and France on the one side, and Ireland on the other. Soon the whole island had been infected except exclusively breeding districts (Welsh and Scottish Highlands, etc.,) into which no cattle were ever introduced from outside.

In 1847 an importation of English cattle into Sweden conveyed the disease, where it prevailed for some time and even infected Denmark through shipments of cattle, but was finally extirpated by the pole axe and disinfectants.

In 1860, Norway imported infected Ayrshire cattle for the Royal Agricultural College at Aas, but the imported and exposed stock were promptly destroyed and the previous immunity of the country has been maintained up to the present.

Denmark was infected in 1848 by importation from Sweden, and on different other occasions from Germany and England, but by prompt and rigid suppressive measures, stamped out the dis-

ease on each occasion. This was true also of Schelswig so long as it remained a part of Denmark, but the infection entered in the commisariat parks of the German army in 1864, and the principality remaining in German hands, the lung plague became permanently established.

Mecklenberg-Schwerin and Oldenburg also maintained vigorous suppressive measures and for long, kept clear of the infection.

Into Brooklyn, lung plague was introduced in 1843 through the purchase of an English ship cow by a dairyman (Peter Dunn). From this centre the infection spread until it prevailed in connecticut, New York, New Jersey, Rhode Island, Pennsylvania, Maryland, Virginia, District of Columbia, Ohio, Indiana and Illinois.

In 1879, measures were taken in New York and New Jersey to extripate the disease but failed to receive the requisite continued support and were practically abandoned. It was only in 1887 when the infection had reached Chicago, the largest cattle market in the world, that the National and State Governments were aroused to the gravity of the danger, and, the Federal Government supplying the funds and the Illinois Government the authority, the beginning of a real attempt at extinction was seriously undertaken. The author acted as chief of the national veterinary force in Illinois in 1887, and takes great pleasure in recording that at the end of three months from the date of his arrival, the last acute case of the disease had been disposed of and the frightful danger that had threatened the nation through the Chicago stock yards had been removed. The supervision was maintained for a year later, and some old sequestra were later found in the lungs of cattle slaughtered, but no acute nor dangerous cases of the disease. This was followed by vigorous suppressive work in other infected states and by September, 1892, the quarantine was raised and the nation pronounced free from lung plague. The last cases were met with in New Jersey, early in 1892. In this last anti-lung-plague crusade the National Government inspected 1,605,721 cattle in 166,951 herds, made 356,404 necropsies, purchased 21,961 head of cattle, and disinfected 4,128 premises. 7,438 cattle were found affected at the necropsy.

Another line of infection was started by 4 Dutch cows, landed at Boston in 1859, and before the end of the year the infection

had extended into 20 different townships in Mass. In 1860, a State Commission was created, with Dr. E. F. Thayer as veterinary commissioner, and in the next few years they destroyed 1,164 cattle, and stamped out the plague at a total cost of $77,511.07.

An importation of infected cattle into New Jersey was made in 1847, by Mr. Richardson, who on discovering the nature of the disease, made an end of that particular contagious centre, by slaughtering his whole herd at a cost of $10,000.

South Africa was infected in 1854 by the landing of a Dutch bull at Cape Town. As there were no railroads and all inland carriage was conducted by ox-wagons, it soon spread over the colonies and extended thence into the native states, and has continued to the present time.

Australia was infected in 1859 through an English cow landed at Melbourne. When the disease was identified, the whole herd was slaughtered and paid for, and the farm quarantined. But the quarantine was broken by a teamster turning his work-oxen into the pastures under cover of night, and the infection escaped and has prevailed over Australia to the present day.

Later the disease extended in a similar manner, to Tasmania and New Zealand.

Causes. The one essential cause of lung plague is contagion from a preëxisting case. Before the days of bacteriology, this had been demonstrated as conclusively as any truth can be. All extensions into a new country or district could be traced to direct importation from a preëxisting area of infection. Until such importation such lands have been immue from time immemorial; at once after, the infection has spread from the imported animals as a centre; if the stock is divided up and scattered, several primary centres are formed from each of which the plague makes extension. Again, isolated islands (Channel Islands) and purely breeding districts (Scottish Highlands) into the herds of which no store cattle from outside are admitted, remain immune through centuries, no matter how prevalent lung plague may be in the countries immediately around. The immemorial immunity of the Western Continent, up to the date of the arrival of the now famous Dunn cow, the continued immunity of Canada and Mexico after the infection of the Eastern United States, and the

immunity of the United States since the last infected cattle were destroyed in 1892, are equally conclusive in this respect.

Bacteriology. The history of lung plague forcibly illustrates how harmless microbes of large size, that may be easily discovered, and which, existing in the environment, readily find their way into diseased and susceptible parts (in this case into the bronchia), may be held to be the pathogenic cause. Willems and Van Kempen, in 1852, found microbes in the exudate. Lustig in 1885 found four separate microbes in the lesions, 1st, a short, thick, liquefying bacillus to which he attributed the disease; and 2d, 3d, and 4th, three forms of micrococcus. Poels and Nolen, in 1886, demonstrated bacilli of variable size (0.9μ), solitary, in pairs and chains, cultivable in different media, and inoculable by injecting such cultures into the lungs, but the resulting lesions were not marked by the full lung plague exudate. Arloing, in 1887, separated from the exudate the *bacillus liquefaciens bovis* a very short, slender bacillus, often in pairs with flagella, motile, staining easily in anilin but not in Gram's solution, quickly liquefying gelatine as a culture medium and assuming a form that might be taken for micrococci, quickly obscuring peptonized bouillon, and growing on potato. The exudate placed in a thermostat at 95° F. encreases in potency. The bouillon cultures injected under the skin or into the lung, produced characteristic lesions of lung plague.

In the light of the later experiments of Nocard and Roux in 1897-8, it would appear that Arloing's bouillon cultures were probably complex, containing not only the *bacillus liquefaciens bovis*, but also, the infinitesimal microbe which is the true cause of the lung plague. In seeding culture media with the exudate taken with all possible precaution against contamination, from the interlobular pulmonary connective tissue, they and others constantly failed to obtain results. Better success attended their efforts with Martin's culture bouillon for producing diphtheritic toxin. Five pigs' stomachs are minced, pounded to pulp, mixed as follows: stomach 200 grs., pure muriatic acid 10 grs., water 50° C., 1000 grs., left in a thermostat at 50°C. for 12 hours, (to 24), then heated to 100°, to destroy the action of the pepsin, then lowered to 80° C, alkalized, filtered from flocculi that formed, heated to 120° C., and filtered. This is then mixed with pepton-

ized meat juice prepared as follows: fresh beef or veal, minced and pulped, 500 grs., and water, 1 litre, are kept for 20 hours at 35° C., the liquid expressed, 5 grs. common salt added, mixed in equal proportions with the peptonized liquid from the pig's stomach, heated to 70° C. to coagulate albuminoids, filtered, alkalized and sterilized. To this mixture is added $\frac{1}{25}$th part of blood serum (sterile) from the rabbit or cow.

This bouillon was inoculated with the pulmonary interlobular exudate, enclosed in collodion capsules, having very thin walls, and inserted aseptically into the abdomen of the rabbit. In 15 days the rabbit was sacrificed, and the capsules enucleated from their envelopes of exudate and cells. The contents showed the slightest possible shade of opacity, but they contained neither cells nor any other definite organism. Under a magnifying power of 2000 diameters the liquid contents were found to be full of brilliantly refrangent points, actively mobile, but so minute that their form could not be made out even when staining was resorted to. The contents of these capsules when inoculated on cattle produced the unequivocal phenomena of lung plague infection in a period of from 8 to 15 days. Other collodion culture cases inoculated from this produced the same cultures in the peritoneum of the rabbit. Collodion cases charged with the uninoculated peptonized bouillon, and placed in the rabbit's peritoneum remained absolutely clear, with no refrangency nor motility under high powers and with no infectivity when inoculated. The rabbits used for the infective cultures often became emaciated to the last degree and even died, but their tissues proved in no respect infecting to culture media nor when inoculated. The attempt to cultivate the germs in collodion cases in Guinea pigs completely failed.

This ingenious form of culture devised by Metchnikoff for experiments on the more delicate organisms, has been used by Nocard to accustom the tubercle bacillus of mammals to grow in the bird and opens up great possibilities for future investigators. The collodion, being impermeable to leucocytes and bacteria, allows these to grow almost together, only on the opposite sides of the collodion wall, restrains phagocytosis, and protects the microbe against the destruction which would otherwise overtake it.

Modes of transmission. The exhalations from the sick convey the infection to susceptible healthy cattle. Yet even this was

denied by one veterinary teacher in Great Britain in the latter half of the 19th century. The transmission of the disease by immediate contagion was denied by those in authority in Great Britain up to the end of the century and this delusion contributed largely to the loss of many millions by the nation. Experiments made at Brown Institution were held to sustain this, but not an atom of evidence was furnished to show that the exposed animals were susceptible ones. Diseased lungs kept for a year at 21° F. proved infecting when inoculated (Laguerriere).

Contagion through the air in the same stable was admitted even by the English sceptics. It may be carried in this way for forty yards, or if dried on dust or light materials to a great distance.

Infection through food soiled by breath or nasal defluxion is a common cause. In our great swill stables during the prevalence of the plague, it was notorious that the disease advanced rapidly along the line of a feeding trough to the sixty or more animals using it, and that the rapidity of the advance was determined largely by the fact that the first animal was at the higher end of the trough. If at the lower end there was no upward current to carry infection to the others. *Open pastures* where the sick have fed and *watering troughs* or *ponds* are common sources of infection. The permanence of infection in and around large cities is largely due to the common pasturage by different herds in succession on the same unfenced lots waiting to be purchased for building. For this reason the plague always extended in summer when the cattle frequented these lots, and diminished in winter when strictly confined to stables and yards.

Mingling of different herds on *great unfenced areas* has been the main cause of the maintainance of the infection from time immemorial in the hills and forests of central Europe and on the boundless Steppes of Europe and Asia. This alone is chargeable with the permanence of the affection, in spite of all efforts for its extinction, in South Africa, Australia, Tasmania and New Zealand.

Contagion carried by attendants, cattle dealers and even *dogs*, is generally recognized, I have elsewhere quoted the case, in E. Lothian, Scotland, in which the son of the steward, who was cattleman on an infected farm, was the means of infecting first, his father's cow, and later the whole of the stock on the place; also

the case at Quincy, Mass., in which a farmer coming straight from the slaughter of sick cattle, infected his own herd ; also the case of Mr. Jewell, of Long Island, who carried the infection from the herds visited, to his own family cow in a thoroughly secluded stable.

Infection through infected and emptied stables, was a common experience in dealing with the lung plague in America. Cows kept alone and only introduced after the predecessor had died habitually contracted the disease, though brought, through the most carefully guarded stockyards, from healthy districts. Stables have continued infecting for a year after having been vacated, (Friedberger and Fröhner).

Infection through manure spread on ground to which susceptible cattle had access was an occasional occurrence.

An *active unrestricted commerce* is however the most prolific means of infection in cultivated countries. Slowly as the disease progresses, and long as the animal diffuses infection, it soon attacks and kills or immunizes all the susceptible animals in the single herd, and if no other susceptible animal is bought or born into the herd, the germ in due time loses its pathogenic potency and infection is at an end. In this way many centres of infection started in herds on well fenced farms have worn themselves out. But the case was far otherwise in the city dairies and swill stables. The trade demanded that the stalls emptied by death should be filled up to consume the swill, or supply the milk route and thus fresh susceptible cattle were constantly exposed in the infected stable. The dairy cows were supplied by dealers who charged exorbitant prices for them and held a chattel mortgage for the amount. Tney had come through the infected dealer's stable, and if they did not come up to the milk yield promised, they were either passed on to another dairy or returned to the dealer's stable carrying back a new load of infection. The wagons used to carry the cows through the city were constantly infected and infecting. The swill stables became the final destination of the surviving cows that had gone dry and thus infection constantly gravitated into them.

In Europe where the great cattle and meat trade with England and the constant demand for cattle to consume the marc of the beet sugar and other factories, cause a continuous draught upon

the infected districts the great western centres of enterprise and commerce have found it impossible to exterminate the plague.

In the country districts in England outbreaks were almost exclusively confined to the times when fresh cattle were purchased at one of the great fairs. Hence the late autumn, the period of laying in feeding cattle, witnessed the greatest extensions.

In Ireland, as shown by Ferguson, the whole cattle trade seemed to be arranged as if for the speedy and universal diffusion of infection. Calves, bought by jobbers from all sources, were bunched together with every opportunity for intercommunication and infection. As soon as they were a little further matured, they were assorted in lots and sent to a fair, where they were sold, and found their way to great common pastures, and this process was repeated again and again until they had reached full maturity. On the way to and from each market they were quartered over night in a public yard which had just been vacated by other animals, often diseased, traversed the same roads, and drank and fed from the same troughs which diseased ones had just used.

The *introduction of apparently recovered cattle* is generally credited with the infection of a fresh herd and Friedberger and Fröhner quote with approval Walley's assertion that a recovered animal with a sequestrum in the lung can infect a herd into which it is taken. I have failed to produce even local exudate, subcutem or in the lung, from sequestra which were much more recent, but the animals operated on were rendered immune and inspired me to carry out a system of immunization by the use of the sterilized fresh exudate. The actual date of the expiration of virulence in the necrotic lung tissue has never been demonstrated and probably varies in different cases.

The *receptivity* of different breeds has been discussed and the Holsteins and Shorthorns have been thought to show the greatest susceptibility. These are among the cattle that are most prized, bought and sold, and exposed to infection. The Shorthorn especially has a great development of the circulatory system, including the connective tissue with its great lymph sacs and channels, and the microbe we are dealing with shows a marked preference for the lymph system, in which it finds its appropriate field of development. Susceptibility is greatly encreased by a *warm*

climate or *hot season*, under which large herds may die without a single exception, the disease running a fatal course in two or three days. In cooler climates 20 per cent. will often escape at first (French Commission Experiments, 1849).

Lesions. If death has taken place early, the lesions are usually essentially pulmonary, though they may implicate the pleuræ and mediastinum. In many cases the lung alone is involved, yet even then the predilection of the microbe for the lymph network of the interlobular tissue is strongly manifested in the serous infiltration of that, rather than of the lung structure proper. This determines the much talked of marbled lung, the pulmonary lobules standing out at first as a more or less deep red brown or black, while the marbling is caused by the yellowish or grayish infiltrated tissue surrounding each pulmonary lobulette which appear set in, as in mosaic on the surface of a section. Sometimes a blood extravasation discolors the interlobular exudate as well, yet it retains its soft liquid appearance which sufficiently distinguishes it from the firmer lung tissue. These yellowish interlobular markings vary much in thickness, but in acute cases this may be up to half an inch or even more. In the lesions of longer standing the interlobular exudate has usually coagulated, or even undergone fibroid organization, so that it compresses and condenses the lobules which it surrounds.

The pulmonary lobules may show the earliest changes of the alveolar and lobular capillaries, with formation of an exudate, and an active proliferation of small round cells in and around the alveoli. Later the exudate coagulates, forming the familiar red hepatization, and this in its turn may pass into the gray, or it may liquefy and undergo absorption and resolution. Other changes are not uncommon. The excess of interlobular exudate will compress the pulmonary lobules so as to reduce their size and expel the blood from their structure, giving them a pale color, or this compression becomes still greater, completely arresting the circulation and inducing lobular gangrene. Thrombi in the afferent and efferent vessels contribute to the necrotic change, and sequestra of varying size, from that of a nut to that of an infant's head or larger, are formed. The earliest stage of this necrosis is usually infarction of the lung, a definite area of which becomes saturated with dark blood cells so that in contrast

with the rest of even the hepatized lung, it is almost as characteristic as the marbling. The farther process of this necrosis is varied. Most commonly the exudate surroundng the necrotic mass becomes organized into white fibrous tissue and forms an investing sac in the interior of which is the dead lung tissue, showing for a time distinctly, the bronchia, blood vessels, lobulettes, and interlobular tissue. This gradually becomes detached and floating in a liquid débris, slowly undergoes solution, and is absorbed, the sack meanwhile closing in on the cavity. A large sequestrum may be a year or sixteen months in undergoing complete solution. In other cases the pulmonary lobulettes undergo an individual softening while the interlobular tissue becomes organized and when cut across, the lung presents a distinct honeycombed appearance. In still other cases a considerable area of both lobulettes and interlobular tissue is necrosed and liquefied, while the exudate, around the bronchial tubes, that supplied it, becomes organized, and on the necropsy the latter are found to constitute a thick branching mass of a very characteristic appearance.

The newly affected lobulettes have a watery or gelatinoid or dropsical appearance and if freely incised give out a large amount of serum and flatten down in doing so. When hepatized the cut surface is granular, and microscopic examination shows the terminal bronchia and alveoli filled with a fibrinous exudate containing great quantities of red blood globules and leucocytes. The distension of the lung is enormous, so that when the entire organ is infiltrated it may weigh from 50 to 100 pounds.

In recent cases the lung may be extensively affected without affecting the *pleuræ*; in other cases both are early involved. In advanced cases the pleuræ are always implicated. First there is the subpleural infiltration over the affected part of the lung; later the pleural surface has reddened arborescent patches, with a slight solid exudate, and a yellowish (sometimes blood-stained) serum collects in the bottom of the cavity; later still the affected portion of the lung is covered more or less thickly with false membranes, while others cover the organ or the parietal pleura below this level, and hang in shreds or bind the lung to the ribs. In old standing and recovered cases these may be largely represented by dense, white fibrous investments covering the lung or

the rib, or establishing permanent adhesions. The amount of pleural effusion may be about two gallons in bad cases, and like the invasion is either unilateral or bilateral. The *pericardium* is usually more or less involved in pleuritic cases.

The *larger bronchia* sometimes contain false membranes.

The *bronchial*, *prepectoral* and *mediastinal lymph* glands are often enlarged, congested and infiltrated with an abundant exudate.

Similar infiltration of the lymph glands of the pharynx, mesenteries, sublumbar region and groin are described, together with hypertrophy and congestion of the intestinal follicles, and congestion of the muscles and inter-muscular tissue, of the articular and tendinous synovial sacs, and even of meninges of the brain and cord. I know of no facts to show that these conditions result from anything else than the toxins and the general constitutional disorder. The indications are that the microbes are speedily destroyed in the circulating blood, and intravenous injections and caudal inoculations alike fail to cause the characteristic lesions in the lung, their favorite point of casual election.

Incubation. This usually lasts from 6 to 30 days, being greatly abridged by hot weather and often prolonged in the cold. Delamotte claims to have seen it extended to 5 months, and in one case a calf turned out on Montauk Point, L. I., from an infected place was noticed ill on the 104th day. Australia, South Africa and Norway were each infected by cattle that had been three months out of the native infected land. I have seen cattle pass three or four months after purchase in an unhealthy condition, yet without cough or other recognized diagnostic symptom and then come down with lung plague. Such doubtless explain the alleged protracted incubations, the system has been invaded, ill health ensues, but the lungs are only slightly affected for a length of time. In other cases one animal in a herd has had a mild attack, which escaped notice, and it was only later that the disease was recognized in a second victim, infected from this first.

The bearing of this on imported animals is evident, An animal imported alone should be kept under surveillance for a month or two after quarantine, while in any considerable herd the disease would certainly manifest itself during the prescribed three months detention.

Symptoms. These vary greatly with the animal and its environment. Other things being equal it may develop more suddenly and violently in the obese Shorthorn, Angus, Hereford, Ayrshire or Dutch, and less so in the spare Brittany or Jersey. In very hot weather the attack is very sudden and severe. A chill from exposure, an attack of bronchitis or pneumonia, the excitement attendant on parturition, on traveling by rail or driving may precipitate and aggravate the seizure. Under some such conditions there may be sudden and extreme hyperthermia, rapid pulse, oppressed breathing, percussion and auscultation evidence of extensive pulmonary consolidation and death in two or three days, while the body is still plump and fat.

Individual susceptibility appears to have influence, the same stable presenting simultaneously cases of acute and fatal type and others that are slow, and insidiously progressive. In newly invaded countries and in bovine families that have not been exposed to the infection for many generations the tendency is to a higher proportion of severe and fatal cases, while in herds native to districts that have been continuously or frequently exposed, mild cases tend to predominate. The more susceptible strains of blood have been killed out, and the surviving strains show a greater power of resistance.

Apart from the predisposing environment the tendency of lung plague is to set in slowly, insidiously, and for a time almost without outward symptom. For a week, fortnight, month or more there may be a slight cough heard only at rare intervals and neither painful nor specially troublesome. Though sometimes hard, it is more commonly small, weak, short and husky, noticed only when the animal rises, drinks cold water, goes out to the cold air, or eats dusty or fibrous fodder, and is usually attended by arching of the back, extension of the head, opening of the mouth and protrusion of the tongue. For weeks there may be no indication of constitutional disorder, appetite, rumination, milking, and other functions appearing to be normal. Driving the animal may unduly accelerate the breathing, and a careful auscultation may detect an unusually loud blowing sound behind the middle of the shoulder, a mucous râle, or a wheeze. In some cases the disease never advances farther, the trouble subsides and the subject is thereafter immune. Cases of this kind occurring

as the first in a herd, explain some instances of what are claimed to be specially prolonged incubations.

In the great majority of cases further symptoms appear, hyperthermia sets in, varying in different animals from 103° to 108°F., the animal becomes dull, depressed, loses in appetite, rumination and milking, omits pandiculation on rising, shows stiffnesss of the hind limbs, sometimes knuckles forward at the fetlocks, wanders apart from the herd, is found lying apart, shows extra thirst, bloats slightly, and shows some constipation. Pulse and breathing are accelerated, auscultation signs are more marked, and on percussion, areas of flatness may indicate lobular consolidation, usually more extended than in ordinary pneumonia with the same grade of constitutional disturbance. The muzzle becomes hot and dry, the roots of the ears and horns hot, and the hair stands erect along the dorsal aspect or in patches over the body. Pinching of the dorsal spines, sternum or intercostal spaces, may cause marked wincing and a deep groan. The eye is dull, lacking in prominence and clearness, and the lids are often partially closed. At this stage improvement sometimes ensues and after inappetence and suppression of milk for one or two days, the patient may take to feeding and milking as before, and apparently recover, though with a large pulmonary sequestrum.

In the continous and violent cases all the symptoms are aggravated. Fever may run to its extreme height, there is complete anorexia and suspension of rumination, pulse and breathing are rapid, the victim no longer lies down but stands with feet apart, arms and elbows turned out, head extended nearly on a line with the neck, mouth open, tip of the tongue projecting, and each expiration accompanied by a moan, so loud that it may be heard at a distance (often 50 yards). The breath is heavy, feverish, mawkish. The flanks heave violently, the nostrils are widely dilated and discharge mucopurulent, often bloody liquid, strings of fœtid saliva drivel from the open mouth, there may be tympany or even colic, the eyes are sunken, and the conjunctiva and nasal and buccal mucosæ are of a dull brownish and yellowish red. Emaciation advances at a rapid rate, and the constipation may be superseded by a profuse, fœtid diarrhœa which wears out the animal. The skin is dry, scurfy, withered, pale, and clings firmly to the bones, and the interior of the vulva may show the pallor of

anæmia. The spine, sternum and intercostals are more than ever tender, and pressure on the tender areas may detect a lack of movement of the lung which is felt on the healthy parts.

On percussion very extensive areas of consolidation are revealed by the dulness and flatness. If pleuritic effusion exists the resulting flatness is extreme below and up to a given horizontal level, the line of which may, however, be elevated at points by consolidation of the lung at such parts. Auscultation may reveal almost any of the abnormal sounds of pneumonia or pleurisy. Absence of respiratary murmur over large areas, with blowing, heart or abdominal sounds in unwonted situations (where the murmur is absent), and abnormally loud murmur where the lungs are still pervious; crepitation around the margins of the consolidated portions; the creaking sound of stretched false membranes, scarcely distinguishable from crepitation; mucous râles; wheezing sounds of various pitches; exceptionally splashing sounds, and if the animal has just risen, the metallic tinkling sound. In other cases the pleural friction sound is prominent.

Abortions are common in pregnant animals.

Course. This varies greatly. In our northern states in winter, many would seem to recover after a few days illness; in summer, many died in a few days with excessive exudate, dyspnœa, and prostration. Others died early from tympany of the rumen. In others still, profuse, fœtid, colliquative scouring led to an early death. In extreme cases there would be a loss of one-third or one-half the weight in a single week. In the less rapidly fatal cases all the symptoms became aggravated, the emaciation progressed, and a liquid pultaceous condition of the bowels continued for two, three, four, or even six weeks, the animal finally dying in a state of marasmus. In such cases the shrunken, pallid skin and mucosæ bespoke an extreme degree of anæmia.

Recoveries may take place from comparatively advanced stages, but they are liable to be slow and imperfect, the animal remaining unthrifty for a length of time. In some of the more favorable cases, when the recovery is less interrupted or retarded, fattening may take place rapidly, so that it would appear as if the loss of a portion of lung, and the lessened consumption of hydrocarbonaceous matter contributed to the deposition of fat.

In some cases the active morbid processes subside, but are not quite arrested and the malady assumes a chronic form, the exudates become organized, causing a sclerotic or morbid condition of the lung and especially of the interlobular tissue, which compresses and carnifies the lung tissue, or leads to necrotic changes and the formation of sequestra, or again to liquefaction and the formation of vomicæ. Here the proliferation of the germ may go on, though little new tissue is invaded, and the animal may remain infecting for a long period. The patient remains thin, and weak, is easily blown under exercise, has a small, accelerated pulse, little power of digestion or assimilation, shows frequent tympanies, and may have profuse diuresis or diarrhœa. The cough is frequent, often paroxysmal, easily roused, loose, mucous, painful and may be attended by muco-purulent discharge. Such animals are liable to die in the end in marasmus.

In some cases the malady is complicated by other infections, leading to the formation of abscesses in the lungs, pleuræ, chest walls, bronchial glands, joints or elsewhere, or tuberculous deposits may complicate the lung plague, and there may be an early death or a long drawn out decline.

Diagnosis. So many different affections resemble lung plague closely during life, that a certain means of diagnosis is a great desideratum. Yet many points help one to reach a reasonably certain diagnosis:

1. Does lung plague exist in the country? If not, the presumption is very strongly against it.

2. Have the suspected animals been recently imported from a lung plague infected country? or have they been in contact with animals recently imported from such a country?

3. Have they been fed on provender imported from such a country?

4. Have they been attended by a man recently from such a country and whose clothes have not been sterilized?

5. Have they been carried on any imperfectly disinfected car or ship which has carried the cattle of such country?

6. Has the affection attacked a considerable proportion of the herd at the same time? A rare thing with lung plague.

7. Has the attack come on slowly and insidiously? Very characteristic of lung plague.

8. Has hyperthermia been marked? Indicative of lung plague.

9. Have a succession of cases occurred at intervals of several days or weeks, irrespective of weather? Suggestive of lung plague.

10. Have the indications of consolidated lung been early and over extensive undivided areas? Like lung plague.

11. Have cases been milder in cold dry weather and more violent in hot and moist? Such is lung plague.

12. Does the same subject present lung lesions of very different ages;—infiltration with tough, elastic lung; red hepatization; gray hepatization; black infarction; sequestra, etc.? Bespeaks lung plague.

13. Do the infiltration and red hepatization show a marbling with very extensive filling and turgidity of the interlobular and subpleural connective tissue, and abundant effusion into the chest? This is the nature of lung plague.

14. Does the lung exudate when inoculated in the tail of a susceptible bovine animal (one that has never had the disease), produce local inflammation and exudation and procure immunity? This characterizes lung plague. Does it fail to cause inoculation-swelling, in an animal that had lung plague. Lung plague it still more indicated.

15. If the disease had lasted long enough in a place to determine, has it affected any other than bovine animals, and does inoculation of the lung exudate into any other genus of animal cause the disease? Other genera are immune from lung plague.

Tuberculosis is distinguished by the habitual absence of the high temperature, the numerous circumscribed areas of flatness with wheezing or other abnormal lung sounds, in the midst of a general field giving the normal respiratory murmur, by the enlargement or induration of superficial lymph glands, by the response to the tuberculin test, and by the lung tubercles and diseased lymph glands,—congested, indurated, caseated, calcified—found at the necropsy. Inoculation of other genera causes tuberculosis.

Simple bronchitis is usualy connected with climatic change or exposure, tends to affect a number at once, gives the blowing and mucous râles, without indications of extended lung consolidation or pleural effusion, and after death does not furnish lesions of all ages, recent and remote.

Verminous bronchitis, attacks the young only or mainly, and never seriously injures the mature, involves all or nearly all the young in the herd, shows less hyperthermia, and less extensive consolidation, more wheezing in the lungs, and a free expectoration in which the worms (embryos or adults) may be found by careful search.

Simple fibrinous pneumonia is likely to arise from climatic conditions or from exposure, attacks several at once and none later, has less abrupt rise of temperature, less tenderness of the chest walls, less dyspnœa, less obstinate standing, and after death, less interlobular exudate and marbling, and no presentation of old and recent lesions on the same lung.

In the *infectious pneumonia* occasionally seen in western cattle, the distinction must be made mainly by the less elevated temperature, less dyspnœa, the lack of the early signs of extensive consolidation of the affected lung, and after death, absence of the extensive interlobular infiltration, and in the predominance of the whitish points or mottling indicating the broncho-pneumonia and the cell proliferation in the terminal bronchia and alveoli.

The pulmonary form of *hæmorrhagic septicæmia* (Wildeseuche) is distinguished by its enzoötic character, in connection with wet lands, its occurrence in the summer season, its association with other forms of localization, in the abdomen, muscular system, etc., its dependence on a cocco-bacillus, easily found in the exudate, and its inoculability on the smaller ruminants, horses, pigs and rodents.

The *perforation of lungs and heart by a sharp pointed foreign body* from the reticulum, occurs sporadically in one animal which has usually been kept indoors ; it shows as a rule less fever, may be preceded by tympany or other gastric trouble, shows a line of consolidation from the reticulum forward, is earlier or later complicated by morbid heart-sounds or rhythm, and commonly shows a pitting swelling beneath and beside the sternum.

Pulmonary emphysema is sporadic only, usually devoid of sudden hyperthermia, or constitutional disorder, has a dry paroxysmal cough, wheezing sounds on auscultation, and a drumlike sound on percussion. If associated with bronchitis or pneumonia and attendant fever, the case is more equivocal.

Aspergillosis of the Lung usually advances more slowly, in the comparative absence of sudden hyperthermia, shows more limited

areas of consolidation, less dyspnœa, and usually affects smaller animals (birds) as well. At the necropsy the presence of aspergillus in bronchia and lung tissue is conclusive.

The *acute pulmonary congestion* of heat stroke is sufficiently identified by the conditions under which it occurs, its sudden and rapid progress, the implication of the brain, and its occurrence in other animals similarly exposed.

Such conditions as *atelectasis*, *pulmonary actinomycosis*, *distomatosis*, or *echinococcus*, the *congestion of mercurialism*, etc., should be readily recognized by the attendant conditions, analysis or necropsy.

Attempts have been made to diagnose lung plague by securing a reaction, local or general, as the result of injection of the sterilized lung exudate or cultures, but they have proved eminently unreliable. The susceptibility of the serum to change under manipulation, the existence of hyperthermia before the injection, and the disposition to local infiltration under the lung plague poison are apparently insuperable obstacles.

Treatment. No treatment has been devised that would warrant the preservation of the infected animals when the alternative of prevention is available. The question is an economic one, and with a disease that is so insiduous, with such a long incubation, with so many occult and chronic cases liable to escape observation and recognition, and with such a constant and prolonged exhalation from the lungs of the virulent material and the certainty almost of the diffusion and preservation of the latter, no more suicidal policy could be adopted than the preservation and treatment of the sick.

If allowed at all, treatment should be conducted in an isolated locality, well secluded from visitors and wandering animals including birds, and under the most intelligent antisepsis. It should proceed on general principles according to the indvidual manifestations of the disease, and might include serum therapy from immunized animals.

Prevention. For a country like the United States, now happily free from the lung plague infection, the important object is its permanent exclusion. For this the federal quarantine for three months now in force ought to be effective. The only question would be in the case of small importations of one or two animals,

which might introduce infection under an apparently prolonged incubation, or with a chronic type of the disease. Small importations of one or two, might be dismissed at the end of three months quarantine under a *ticket of leave* system, under which they and the herd into which they are taken can be kept track of for three months more. In the larger imported herds the possibility of the escape of infection under a three months quarantine is so infinitesimal that it may be practically ignored.

Similar precautions must be taken against the importation of cattle from a country reputed to be free from the lung plague, but which imports cattle from an infected country without imposing the three months' quarantine.

The use, for transportation of cattle from one part of an immune country into another, of cars or boats which have been used for transporting cattle in an infected country can only be permitted after the vehicles in question have been thoroughly cleansed and disinfected and attested so by the official experts.

The landing in an uninfected country of hay, fodder, or cattle-food of any kind, or of litter, grain bags, head ropes, manure, or other article coming from an infected country must be prohibited until such article shall have been thoroughly disinfected.

Horns, hoofs, hides, hair and other products introduced into the country must be disinfected. The treatment adopted to exclude anthrax would be amply sufficient for lung plague.

Measures to Extirpate the Infection. Stop all accessions of possibly infected cattle from outside. Proclaim the infected area, prohibiting all entrance and egress of cattle, and all movement of cattle from herd to herd within the area, except under special license, based on the soundness of each herd for six months antecedent. Prohibit absolutely the pasturage of cattle on public highways and unfenced or insufficiently fenced places. Send to instant slaughter cattle found in such places in violation of this rule. Make an accurate census, with individual description of each bovine animal in the infected district, and make a necropsy of each such animal dying at the hands of the butcher or casually. Hold the owner guilty of a misdemeanor with heavy penalty, for every bovine animal that goes amissing in the infected district without official post mortem examination. When an infected animal is found in a herd have the whole herd, marked, appraised

and slaughtered under official supervision, with necropsy of each animal. If any herd has been losing animals, or had sick animals within a year, buy and kill the whole herd. Indemnify the owners to the amount of at least two-thirds or three-fourths of the sound market value for all except the advanced and acute cases of the disease, and such animals as have been moved into the State less than three months before. These latter may be sacrificed without indenmity. Disinfect thoroughly all infected premises and things at public expense. Close the fields against all outside cattle for three months. Burn all hay, straw, litter, and grain in the infected buildings or see that they be fed to horses, sheep or pigs apart from where cattle are kept. Burn or disinfect all manure or have it drawn out and plowed under by horses, and the wagons and implements used in doing so thoroughly disinfected. Allow restocking of the disinfected premises from sound districts only, and keep up the strictest supervision and control of the herds for from three to six months.

For private Control in the absence of Government Action. The stock-owner in a secluded locality, not bordering on a highway or railway can as a rule secure the immunity of his herd by such measures as the following. Breed all the stock on the farm. If a change of blood is required buy the bull young and keep him strictly by himself for at least four months, allowing him to mingle with other cattle only after he has been thoroughly attested. Allow no animal to go outside for service or any other purpose and afterward return to the herd. Allow none to enter from without for any such purpose. If from any cause cattle must be bought, secure them from healthy herds and transport them in thoroughly disinfected cars, boats and by healthy roads, and never through an infected district. Place them in special premises at least one hundred paces from all other cattle, and under special attendants for three months.

Immunizing through a First Attack. When lung plague was at its worst in Great Britain, Mr. Harvey a dairyman on a large scale in Glasgow practiced the method of buying heifer calves and exposing them in his infected stables until they contracted the disease. He had a loss of 20 per cent. and the surviving 80 per cent. were then turned out on his farm and raised and when they came in milk, were sent into Glasgow as new milch

cows immunized from the plague. He thus reduced his losses to the minimum of one fifth of the inexpensive calves, and warded off the heavy losses previously sustained in the valuable milch cows and preserved the still more valuable trade in the milk of healthy animals. The method was a mere temporary makeshift, depending for its success on the permanent maintenance of the lung plague, but so long as there were no well considered government measures for its extinction its permanence was assured in any case, and Mr. Harvey was working no injury to any one, while he was substituting a profitable occupation for a losing one, and supplying his customers with milk from sound cows in place of those that were continually coming down with the plague. Under official measures for the plague-extinction his attempt would have been most reprehensible, but in the absence of such measures it was highly meritorious.

Inoculation in the Tip of the Tail. This is an advance and in some respects an improvement on the Harvey system, as the infection and lesions are localized in the tail, and the mortality is reduced to 2 per cent. In practice a recently attacked animal is selected, and a portion of the lung which is strongly infiltrated but not yet hepatized. This is laid upon a clean scalded plate and incised with a clean scalded knife when an abundance of a clear yellow serum drains out. This is drawn up into a sterilized hypodermic syringe, and the tails having been washed and sterilized, the nozzle is inserted under the skin of each in succession and a drop or two of the liquid discharged subcutem. If despatch is important the washing may be dispensed with and the nozzle wiped and dipped in strong carbolic acid between each two successive insertions.

Various modifications of this procedure have been made. To avoid the inevitable entrance of ærial germs, flamed pipettes have been inserted into a puncture into the turgid lung, made with a flamed knife, the point is then broken and the serum is drawn up to fill the vacuum caused by the heat employed in sterilization. The point is then sealed by melting the glass in the frame of an alcohol lamp, or by melted wax.

Nocard washed the surface of the infiltrated or hepatized lung with boiled water, then with a sterilized knife cut out a deep segment so as to leave a conical cavity and covered this with an

awning to keep off dust. The space soon fills with the draining exudate in a very pure condition.

Pasteur inoculated a calf in the dewlap or behind the elbow, where an enormous engorgement forms, involving the whole ventral aspect, and supplying an almost unlimited amount of serum, which may be collected with such precautions against contamination as are indicated above. The product is thus secured at a comparatively cheap rate, and the risk of its diffusion on the air in breathing is lessened enormously.

Another method is to employ the serum from the swellings in the inoculated tails and carry it on indefinitely from tail to tail.

Arloing in his turn employed the cultures in vitro of his pneumo-bacillus, but with the modern evidence that this is not the infective germ, such cultures can not always be implicitly relied on.

In place of the hypodermic syringe, Australians have used a simple thread soaked in the exudate and drawn through beneath the skin. Others have used a small lancet with a groove hollowed out in the middle of the flat surface of the blade on one side, and which carries in the required drop of the serum.

To preserve the exudate for some time against decomposition, it has been kept on ice, or mixed with chloral hydrate or phenic acid (½ its volume), and glycerine (½ its volume) for two or three months.

Injection into the veins practiced by Burdon-Sanderson has the advantage of producing no local lesion whatever and yet securing a fair measure of immunity. It is, however, a much more delicate operation as it entails a possible though remote possibility of producing capillary thrombi, and some danger of infection of the wound in the vein. To avoid this latter, the jugular vein is raised as for phlebotomy, and a short needle is passed into it. A longer and more delicate needle is now passed through this and the injection of a few drops of the exudate is made through the latter. The small needle is then carefully withdrawn to be followed a few seconds later by the large.

Injection of the Sterilized Exudate Subcutem. In 1881 having found that liquids obtained from old sequestra, produced no local lesions when inoculated subcutem, but secured immunity for the subjects, I inoculated ten susceptible cattle with the fresh pul-

monary exudate which had just been heated for 30 minutes to 180° F. No local inflammation nor exudate occurred in any of them, but subsequent inoculations with fresh unsterilized lung plague exudate were resisted in the same way. Six of these immunized cattle were subsequently placed in two infected stables and herds, (Mr. Butts, Brooklyn, N. Y., and Mr. Christopher Slade, White Hall, Baltimore Co., Md.) and retained there for three months without showing the slightest indication of disease. Later I applied the measure on a number of herds with throughly satisfactory results. About ten years later a similar resort was had by Arloing and Rossignol with corresponding success.

Two advantages come from this method : 1st, there is no local infection and no marked swelling so that the injection can be made on the side of the neck where the skin is thin, and clean, and the connective tissue abundant, and where there is less risk of extraneous infection than in the too often dirty or filth soaked tail ; 2nd, as no living germ is introduced there is no possibility of propagating the disease to other neighboring susceptible animals. Two per cent. of loss, of animals inoculated by the Willem's method is counted on, but with the sterilized virus there is not even a remote probability of loss. Infection cannot occur from the animal injected with the sterilized virus, so that it can be safely applied among cattle that have not been exposed to infection, but which are likely to be in the future, and these injected cattle can be left to mingle with others that have not been injected without a shadow of danger to either.

Conditions permitting and forbidding Immunization. Immunizaton is permissible or commendable in all cases in which lung plague is already widely spread in a land destitute of fences and in which cattle roam at large, and herd mingles freely with herd. Here the extension of the disease is inevitable and continuous and effective measures for extinction are impossible. It is permissible where the plague is widely spread among cultivated and fenced farms, but where no authoritative measures are in force for its extinction. In such case, with the Willem's system the inoculated herd should be kept thoroughly secluded in premises or well fenced pastures, apart from any highway, and not adjoining any other cattle pasture.

When on the other hand official measures are in force for the extinction of lung plague, every form of immunization based on

the production of the pathogenic germ in the living body of the bovine, or even in vitro, is to be unqualifiedly condemned. The risk of the escape of the infection through subtle, unsuspected channels is too great to allow of its reproduction in any form. By restriction of cattle movement, slaughter and disinfection, extinction of lung plague is easy and certain, but, whatever may be true of an individual herd, no country has ever permanently extinguished lung plague infection, when the virus was systematically multiplied for uses in inoculation.

CATTLE PLAGUE.

Synonyms. Definition : infectious fever of polygastrics, with sudden onset, violent progress, high mortality, congestions of mucosæ, petechiæ, concretions on buccal vulvar mucosæ and on skin, erosions of gastro-intestinal mucosæ, and pulmonary interlobular emphysema. Historic notes : ancient—China, Hindoostan, Steppes ; in middle ages—Europe, Britain ; recent—Europe, England (1714 and 1740), Scotland (1770), Central Europe (1796–1816), Southern Europe (1827), Germany (1830–1), Egypt (1841), Britain (1865), France (1870–1), S. Africa (1881), Abyssinia (1890), Japan (1892), The Philippines (1898–9). Animals susceptible : ruminants, peccary, (swine), Bacteriology ; minute corpuscles in cell nucleus (Semmer, etc.), which are held back by Berkefeld and Chamberland filter. Accessory causes : such as favor preservation, multiplication and diffusion of germ. Susceptibility varies with previous exposure of the race. Immunity after one attack All liquids and secretions of the sick are virulent ; also manure, hay, straw, dust, stables, troughs, cars, boats, loading banks, yards, milk, flesh, fat, sausage cases, hairs, horn, hoofs, wool, bristles, hides, bones, halters, harness, shafts, poles, goads, boots, clothes, feet (animals), wheels, runners, vermin, wild animals. Virulence lost in drying. Manure preserves for weeks. Lost at zero, and at 131° F. Lesions : congestion, petechiæ, hæmorrhages and erosions on fourth stomach, small intestine, rectum, vagina and mouth, emaciation, sunken eyes, diarrhœa, wart-like epidermic elevations, concretions on mouth ; conical papillæ dark, like port wine ; petechiæ and extravasations in subderma and submucosa ; swollen intestinal glands ; spleen normal : liver, pale, soft ; kidneys swollen, congested, petechiated, softened ; lungs with spots of congestion and extravasation and emphysema : petechiæ on heart and pericardium : blood has excess of fibrine and leucocytes, black. Incubation 2 to 9 days. Symptoms ; hyperthermia (104° to 108° F.), white epithelial concretions on gums, followed by abrasions, congestion of visible mucosæ, weariness, debility, thirst, constipation followed by diarrhœa, tender loins, drooping head and ears, weeping eyes,

grinding teeth, rapid pulse, expiration with arrest and click, suppression of milk, relaxed sphincters. May become aggressive or soporific. Diagnosis: by rapid and deadly progress, and manifest infection; from *malignant catarrh* by the active spread, numbers attacked, concretions on mouth, and known exposure; from *thrush* by the high fever, contagion to old as well as young, and severe abdominal symptoms; from *aphthous fever* by the high temperature, the absence of distinct vesicles on mouth, teats and feet, by the comparative immunity of swine, and by its high mortality; from *dysentery*, by the early hyperthermia, the concretions in the mouth, by rapid general extension irrespective of filth and crowding, and by the implication of stomachs and small intestines, rather than the large; from *gastro-enteritis*, due to chemical irritants, by the lack of such manifest cause, and its rapid progress from herd to herd; from *anthrax*, by its rapid spread beyond an anthrax locality, the buccal and skin concretions and desquamations, by the insuceptibility of horse, dog, and rodent, by the absence of splenic enlargement or incoagulable blood. In sheep: mortality in Steppes, 30 to 50 per cent.; in new countries 90 to 95 per cent. Treatment: to be condemned where its permanence is not accepted Serum-therapy: blood serum of immunized animal subcutem. Prevention by immunization: mixture of virus and bile; inject with highly immunized and defibrinated blood, and expose to the sick, only admissible where extinction is despaired of. Exclusion: exclude all ruminants and their products which come from suspected lands, or admit on certificate and quarantine, or for slaughter only. Extinctions: Trace and kill all ruminants that come in proximity to every infected animal, or to any place or thing where it has been, disinfect throughly the carcasses, products, places and things, register all ruminants around a wide area of possible infection, make necropsy in every case of death, appraise and sacrifice any herd showing the infection. Each seaboard state should provide for instant action by the Federal Government. Question of extinction in the Philippines.

Synonyms. Pestis Bovina, Rinderpest, Magenseuche, Viehpest, Viehseuche, Pockenseuche (German). Pest Bovine, Typhus Contagieuse, Typhus du gros Betail (French). Tifo Bovino (Italian). Dzuma (Polish). Tchouma Reina (Russian). Low peng (belly sickness, China). Pushima (Hind., Burm.).

Definition. A contagious fever of polygastric mammals (bovine, ovine, caprine, cervine, exceptionally porcine), characterized by sudden invasion, rapid advance, hyperthermia, great constitutional disorder, congestion and blood extravasations of the mucosæ generally, but especially of the gastric and intestinal, epithelial and epidermic hypertrophy in the form of white concretions or warty-like masses on the mouth, (vulva), and skin, followed by erosions, by pulmonary interlobular emphysema, by a catching,

arrested inspiration, followed by an expiratory moan, and by an early and very high mortality.

Historic Notes. As the most rapidly developing and deadly of the cattle plagues, this attracted the greatest attention of people in earlier times, and thus its invasions and ravages can be more satisfactorily identified, than those of the tardier and somewhat less deadly lung plague which usually followed in its wake.

Sanctus Severus and Vegetius Renatus indicate its advent into Western Asia on the borders of the Caspian and Black Seas, coincident with the irruption of the Mongols in the first Christian century. It still prevails in China and adjoining countries, including Hindoostan, and since that date the Steppes near the Black and Caspian Seas have been looked on as the perennial home of the plague. Before 376, A. D., the chronicles of epizoötics in Europe suggest anthrax affections which prevailed widely in man and beast, and since that time the special plagues of cattle come into prominence. At this date the Huns began a great onward movement from the region of the Caspian and Black seas into Dacia (Hungary), Northern Italy, Germany and Gaul, and this was the occasion for a general diffusion of Rinderpest over these countries.

After this date cattle plague spread widely on the occasion of any great European war in which the eastern nations were involved or which was so general or continued as to draw upon Eastern Europe for the supply of the commisariat parks. One great epizoötic culminated in 810 after the wars of Charlamagne; one occurred in 820 in connection with invasion of Hungary by the Franco-German army; one in 1223 to 1225 laid waste Central Europe and is said to have reached Great Britain; in 1232–4 it again gained a wide extension following the invasion by hosts of Mongols from Siberia; great extensions are recorded in Italy in 1616 and 1625 during the 30 years' war; in 1709 Charles XII wintered with his army in the Ukraine and his return was followed by the most disastrous mortality ever seen in Europe and which lasted from 1710 to 1717. This reached England in 1714 and was there stamped out by killing and burning the sick, disinfecting the buildings and closing up the infected pastures. Paulet claims that Europe lost 1,500,000 head of cattle in the first three years of this invasion. It continued more or less prevalent in the eastern coun-

tries of Europe, and, following the course of war, entered Italy in 1735, and extended westward. Again in 1740 in connection with the war of the Austrian Succession it extended westward invading the Western Countries from France to Denmark inclusive, and once more extended into England, where it prevailed until 1756 and caused an unprecedented destruction. It was finally stamped out as on the previous invasion. During this invasion Europe lost 3,000,000 head of cattle (Delafond). In 1770 another invasion of Great Britain occurred through the landing of infected Dutch hay at Portsoy, N. B., but this was quickly suppressed by the destruction of every bovine animal in the infected herds, and the thorough disinfection of premises, supplemented by a daily scrutiny of all cattle within a radius of 18 miles. In the second half of the 18th century cattle plague prevailed more or less generally in all Continental Europe, except Norway, Sweden and the Spanish peninsula, into which no cattle were imported, and carried off 200,000,000 head of cattle (Freidberger and Fröhner). In 1796 to 1816 the cattle plague followed the marches and countermarches of the various armies in connection with the French revolution and the Napoleonic wars, causing unheard of losses through Europe. In 1827 it spread widely in connection with the war of independence in Greece, and again in 1830–1 a wide extension occurred in connection with the Polish revolution. In 1844 Russia lost 1,000,000 head of cattle. In 1841 a shipment of Roumanian and Anatolian cattle to Alexandria, Egypt, carried the plague and in two years upwards of 350,000 head of Egyptian cattle perished, only a few being left. From this date the great development of manufactures in the Western European nations, and especially in Great Britain, the consequent increasing demand for beef, and the inauguration of rapid transit from Eastern Europe by steamer and rail, introduced an era of the extension of the cattle plague by commerce rather than war, and Röll gives the losses in Austria alone in 1847 to 1864 at 500,000 head. In 1865 a cargo of cattle from Revel on the Baltic, landed the infection at Hull, whence it speedily extended over the entire country, and prevailed for 18 months, but was stamped out by vigorous measures of destruction and disinfection. In all 279,023 head were reported attacked of which 233,629 died or were killed and 40,165 recovered. In 1865 the plague was once more

imported into Egypt, this time from the Danubian Principalities. A wide extension took place in the parks of the French and German armies in the war of 1870–1, as many as 43 departments in France having suffered. In 1872 it was imported from Russia into Great Britain but was speedily extirpated, and again in 1877 from Germany when it spread somewhat more widely but was easily suppressed. In 1881 it was introduced into S. Africa in Asiatic cattle during the war in the Transvaal and coming after the long continued prevalence of lung plague it threatened the cattle interests with ruin. In 1890 it reached Abyssinia by cattle sent for the supply of the Italian Army and almost exterminated the large buffalo herds in E. Africa, (Schillings). In 1892 Japan suffered through importation from the main land. The latest extension of cattle plague was in 1898–9 into the Philippines in the shipments of Asiatic cattle sent for the supply of the American army, and there as elsewhere in unfenced countries it is proving the cause of disastrous losses.

Animals Susceptible. In spite of its name—cattle-plague, Rinderpest—this affection is not like lung plague peculiar to bovine animals. Yet bovine animals are by far the most susceptible, by them it is mainly propagated, and upon them comes the greatest mortality. Infection, however, extends to all other ruminants,—sheep, goats, deer, elk, antelopes, gazelles, aurochs, yaks, camels, dromedaries, buffaloes, etc. Swine have the stomach partially divided and show a certain susceptibility, it killed the peccaries in the Jardin des Plantes, Paris, and Viseur in France and Pluning in Sumatra, claim to have seen cases in the domestic pig. The horse, dog, rabbit, bird, and man are immune.

Bacteriology. In a disease with such destructive changes in blood and tissues, bacteria are found, almost of necessity, in the seats of the lesions and even in the blood. No constant microörganism has, however, been isolated, cultivated in artificial cultures, and successfully inoculated on other and susceptible animals. Saweljeff isolated sporulating motile bacilli which break up into micrococci, and streptococci, with the culture of these on agar he produced what he believed to be cattle plague. Metchnikoff found a short bacillus with rounded ends, forming cocci and leptothrix-like threads, nonliquefying, and producing cattle plague in calves. Sacharow found a bacillus 0.25μ to 1.5μ long and Tokishige a very small short bacillus the cultures of which

produced rinderpest in cattle. It would seem as if here as in the case of lung plague, the experimenters had retained the real but invisible ultramicroscopic, pathogenic agent in what they took for pure cultures. Semmer attributes the disease to fine corpuscles which have so far eluded current methods of staining and cultivation, and that they exist in the number of from one to six in the enlarged cell nucleus. Nicolle and Adel Bey sustain this position, having found that the unseen virulent germ passed through the more open and thinner Berkefeld filter, but failed to traverse the denser Berkefeld and Chamberland porcelain filters even when favored by a somewhat higher temperature. As this filtration usually removes the germ and renders the liquid noninfecting they hold that the real germ is almost certainly intraleucocytic. When in exceptional cases a few pass through the filter it is held to be only such as were free in the liquid, and these are usually so small in number, that inoculation with the filtrate does not kill, nor always produce appreciable symptoms, but only immunity.

Accessory Causes. The essential cause being the germ, accessory causes are of necessity such as contribute to the preservation of that microbe and its introduction into the systems of susceptible animals.

Susceptibility has a powerful influence even in races habitually subject to rinderpest. The highest susceptibility inheres in cattle, and yet the surviving cattle of the Steppe race, which has been exposed to the infection for centuries, mostly recover from the plague, while fresh cattle imported into the Steppes perish almost without exception. Sheep and goats contract the disease but it is more severe and deadly in the latter than in the former animal. Both, however, can carry the infection back to the bovine animal, as can also the whole group of ruminants. The Guinea pig contracts the affection by inoculation and may thus become an indirect means of conveying infection from ox to ox.

Immunity follows a first attack. Calves born of cows that passed through cattle plague during the last months of gestation are usually immune.

Exposure to infection arises in various ways. All of the secretions of the diseased animal are apparently infecting, and the virus posesses great vitality, so that the channels of infection are almost endless. It is carried in the manure, washed on in streams,

and drains, dried up on hay, straw, feathers, and other light objects, or in dust, and blown about by the winds, left in stables, in feeding and watering troughs, in railroad cars, steamboats, ferry boats, loading banks and yards, it is carried in the fresh milk, flesh, fat, sausage cases, hairs, hoofs, horns, wool and bristles, in hides and bones, in halters and harness, on wagon shafts and poles, on goads, on boots and clothes of men, and the feet of dogs, birds and other animals, on the wheels of vehicles, the runners of sleighs, and by vermin and wild animals. The various infected products, however, soon lose their virulence after drying. Galtier assures us on the basis of the experiments of a Russian Commission, and the experience of France, Belgium, England and other countries that dried or salted hides can be introduced with perfect safety, and that rendered suet, and dried skins, horns, bones and hairs are equally harmless. On stalls, mangers and racks on the other hand, in an obscure and still atmosphere, virulence may be preserved for three months (Müller, Dieckerhoff). Again in litter and manure in the open air, and even in yards and pastures it may retain its vitality for weeks (Chauveau). The infection is destroyed by a temperature of zero, or 131° F. (Semmer).

Whatever determines a movement of animals from an infected locality, determines the extension of the plague, hence war, and commerce, the food demands of a large and encreasing manufacturing population, the inauguration of new routes of rapid transit by steam over land or sea all contribute in their various ways to the extension of rinderpest.

Lesions. The most significant feature of the morbid lesions is their concentration on the fourth stomach, small intestine, rectum, oral cavity and vagina. The respiratory apparatus, eyes, skin, muscles, and nervous system suffer to a lesser extent. If the case has gone on to a fatal result there is usuaily marked emaciation, the natural openings (mouth, nose, eyes, anus) are soiled with morbid discharges (muco-purulent, feculent) the thighs smeared with offensive liquid fæces, and the skin may be yellowish red, or dark, with a general scurfy condition and distinct eruptions, especially of rounded wart-like epidermic concretions on teats and udder. The eyes are deeply sunken, the conjunctiva of a yellowish red, and the lips and muzzle dry, swollen and it may be eroded.

The buccal mucosa is swollen, fœtid, with marked epithelial desquamation and more or less deep and extended erosions on the upper and lower lips, gums, dental pad of the upper jaw, cheeks, hard palate, and root of the tongue. There may still be some of the characteristic, white, epithelial concretions, or the epithelium may hang in loose semi-detached shreds, or there may be extensive areas of abrasion, in transverse cracks or broad patches, and finally extensive petechiæ. The conical papillæ on the cheeks and dorsum of the tongue are especially liable to dark red petechial discolorations.

The subderma and submucosa are also suffused with these congestions and petechiæ, and like the epidermic layer show a marked increase of all elements and of the cell nuclei.

The congestions, petechiæ. desquamations, erosions, are found on the fauces, pharynx, gullet, nasal mucosa, trachea, and bronchia, to a greater or less extent in different cases. There may be even limited areas of superficial necrosis and even the formation of false membranes. In the diseased epidermis and epithelium, in the necrotic plaques and false membranes there are spores and mycelia of fungi and bacterial growths.

In the rumen, reticulum and manifolds, the mucosa and submucosa usually show limited areas of thickening and punctiform or arborescent congestion with softening and even detachment of the epithelium.

The abomasum is profoundly involved, especially in the vicinity of the pylorus. The folds are of a deep blood red or purple, or blackish, port wine hue, or they may be in part brownish red, or when necrotic, slate colored or mottled. The peptic glands are swollen, elevated and dilated, and patches and spots of softened and loosened epithelium are easily detached leaving a deep red surface with, it may be redder oozing points. Ulcerous sores may show on the summits of the folds. The contents are a viscous, fœtid grayish yellow, or reddish liquid, without solid ingesta.

The small intestines show similar lesions, deep, dark red congestions, most intense on the summits of the folds, softened, loosened, ragged patches of epithelium, erosions, ulcers, circumscribed sloughs, casts of the gut formed by desquamating epithelium, and congested, enlarged, and prominent agminated and solitary glands are more or less in evidence. The contents are

liquid and yellowish, grayish or reddish and fœtid. Microscopically the distension of the glands, (of duodenum, Lieberkuhn and Peyer), the proliferation and softening of the epithelium, the enlargement of the nuclei of these and of the cells of the submucosa, and the profusion of microörganisms are marked features.

In the cæcum and colon the lesions are usually less prominent, though swelling, softening and desquamation of the epithelium often exists, and points and patches of congestion, ecchymosis, and necrosis are not uncommon, particularly in the cæcum, and may extend to the muscular layer.

The terminal portion of the rectum is especially liable to marked congestion and blood extravasation, with more or less desquamation and erosion. The summits of the mucous folds are often of a dark red (port wine) hue, and as this is everted and exposed in defecation or later by the relaxation of the sphincter it becomes a marked lesion even in life.

The spleen is normal in strong contrast with anthrax in which similar gross lesions are often found on the mucosæ and especially the rectum.

The liver is usually rather pale and soft, as in other cases of high fever, and centres of necrosis may be present. The gall bladder contains a variable amount of thin bile. The pancreas is nearly or quite normal. The lymph glands generally, and especially those of the mesentery and abomasum are congested, enlarged and softened.

The kidneys are congested, often petechiated, swollen and softened, with centres of necrosis. The bladder, vagina and uterus show mucous congestion, and thickening uniformly or in spots with muco-purulent secretion.

The lungs show spots of hyperæmia, extravasation and at times hepatization, but a very characteristic lesion is the interlobular emphysema already referred to as connected with the abdominal pain and the sudden arrest of inspiration. This gives a gross appearance of marbling as in lung plague, only in this case the inflated interlobular tissue is dark colored instead of white as in lung plague, and it collapses at once when incised.

The encephalon, medulla and nerves present more or less hyperæmia and even exudation.

The heart is usually pale except in the spots infiltrated by blood, but it may be mottled with petechiæ and particularly on the endocardium. The pericardial fluid is often red. The blood is at first little altered, but later undergoes marked changes, notably an increase of fibrine (50 per cent.) and a decrease of water (as in Asiatic cholera). There is marked leucocytosis, and distortion of the red globules. Before death it becomes black and incoagulable.

The whole capillary system tends to be relaxed and over-distended (congested).

Incubation. As seen in England the incubation was from four to five days. Four to nine days are claimed by Galtier as the extremes, yet he quotes incubations apparently as short as 12, 24, 36 and 48 hours respectively. A source of fallacy rests in the prevalence of cattle plague in the district and the possibility of infection through unsuspected channels, before the recognized exposure. Something must also be allowed for the greater susceptibility and the larger dose of the poison which would tend to shorten the incubation. Thus the western improved breeds, which suffer a mortality of 80 to 95 per cent. (Refik Bey), and the winter season, when the virus is concentrated in small ill ventilated houses, both tend to shorten the incubation. At the Albert Veterinary College in 1865, Gerlach went direct from the infected stable to the sound one, and in 48 hours several of the cattle in the latter had a manifest rise of temperature. Roloff observed the elevation of temperature in 36 hours after exposure, and Raupach and Ravitsch as early as 11 hours after the inoculation.

Symptoms. The earliest symptom is abrupt hyperthermia, (104° to 108° F.). This is virtually pathognomonic in a herd or locality in which the disease exists, or in suspected animals presumably coming from an infected region. The temperature usually rises to its height on the third or fourth day, and falls materially when other symptoms are developed. In some instances death supervenes during the high temperature, but in the majority the temperature goes below the normal before death.

Often on the second day, or the third, white epithelial eruptions or concretions appear on the inner sides of the lips, closely resembling the eruption of muguet (thrush) and often showing also on the dental pad of the upper jaw and along the gums of the lower.

In connection with the high temperature, which is absent in *thrush*, this symptom is virtually pathognomic. These concretions are more or less abundant in different cases, sometimes so slight as to be easily overlooked, and in other cases encreasing up to the sixth day, and covering not only the lips, dental pad and gums, but extending back over the hard palate, pharynx and gullet. The concretion is as soft as cream cheese. easily detached and leaves a bright red abrasion (not ulcerated) when removed.

By the fourth day there are dry or staring coat, sometimes rigors, sometimes small, accelerated pulse (60 to 120 per minute), some loss of appetite and impairment of rumination, dry muzzle, a general flush of the mucosæ (mouth, vagina, rectum), and deep blood red discoloration of many of the buccal papillæ. There is weariness or debility, the animal remaining down much of the time, also marked thirst, constipation, the fæces covered with mucus or blood, rapidly advancing emaciation, and marked tenderness of the skin, especially of the loins. The head and ears are drooped, saliva drivels, the mouth has a fœtid odor, the eyes weep, and there may be grinding of the teeth. The skin may be hot and the white and delicate parts (udder, teats) suffused by a deep blush. The milk is decreased at first and finally completely dried up.

As the malady advances the belly becomes tender, the constipation is succeeded by diarrhœa with much attendant rumbling, the fæces are at first watery, greenish and acid, and later highly offensive, yellowish brown or gray, and alkaline. These become encreasingly profuse, fœtid and liquid, assuming perhaps a pea soup or rice water consistency before death. The sphincter, at first firm and quickly responsive, finally undergoes permanent relaxation, with constant exposure of the dark red mucosa.

When the disease is fully established the respirations often become highly characteristic. There is not the sudden catching and shortening of the inspiration as in pleurisy, but a sudden closure of the glottis with an audible clicking sound in the course of the expiration, and, after a perceptible interval of holding of the breath, the expiration is resumed with or without an accompanying moan. This phenomenon is so characteristic as to be largely diagnostic. Whether it is due to abdominal pain roused by the sudden forward movement of the diaghragm, or to

nervous disorder alone, it is a valuable symptom always to be looked for. It further explains the interlobular emphysema usually met with in the lungs in this disease.

In the more violent cases death supervenes from the sixth to the ninth day, but in the indigenous breeds of Eastern Europe and Asia, which represent a *survival of the fittest*, the great majority suffer mild attacks and recover, and even in newly invaded countries, when the invasion has spent itself and the less susceptible are largely attacked, a fair proportion survive for a longer period and even recover.

In these milder, protracted, or surviving cases the skin symptoms are likely to come out prominently. Chief among these and very constant at the height of the disease is an abundant unctuous exudation which dries on, forming a crust, comparable to what is seen on the skin in the fevers of swine. At different points, notably on the teats, udder, inner sides of the thighs, and arms, and on the neck, lips and face generally, are epidermic concretions having a warty-like appearance, and respectively seated on a very slightly swollen and congested point of the dermis. The deeper layers of these epidermic concretions often soften, so that they have been mistaken for vesicles and pustules, and hence the error by which cattle plague was held to be but a malignant form of cowpox. But these concretions are histologically distinct from vesicles; there is no liquid exudate on the papillary layer raising the epidermis in the form of a little sac, but merely an excessive production of the cuticular cells with abnormally large nuclei, as happens generally to cells in active proliferation, and an admixture with these of spores and mycelium of fungi which have no special significance.

Papules, vesicles and pustules may form on the diseased skin and are described by different authors, but they are not characteristic of the disease as are the epidemic proliferations.

In some rare cases nervous symptoms appear, the animal moves unsteadily without proper sense of balance, it may toss its head and horns as if attacking an enemy or it may sink into a somnolent or comatose condition. Wasting advances rapidly, abortion occurs in pregnant females, the weakness becomes extreme, the animal remains constantly down, unable to rise, rests his head on the ground, breathes heavily and stertorously and perishes with or without convulsions.

Diagnosis in Cattle. Individual symptoms of cattle plague may be found in other diseases, but the aggregate symptoms, in a rapidly spreading and fatal affection, and connected with a previous prevalence of the disease in the country or district, or distinct evidence of the introduction of infection should in every case obviate error. The chief diagnostic symptoms are: a sudden, very marked rise of temperature ; a congestion or blush of the visible mucosæ generally (mouth, nose, vulva, eyes); the formation of white curd-like ephithelial concretions on the inside of the lips, dental pad, or gums of the incisors ; the formation of red spots or petechiæ on the mucosæ ; later the desquamation of the softened epithelium with the formation of erosions or even ulcers ; the wart-like rounded epidermic eruptions on the skin ; the great abdominal tenderness with tucking up of the abdomen, and sudden arrest of the expiration with a sharp clicking sound and moan ; the fœtid, watery diarrhœa and exposure of the dark red, congested and hæmorrhagic rectal mucosa ; the discharge from nose, mouth and eyes ; the characteristic fever odor ; and the rapid progress of the disease from animal to animal, and to a fatal issue. The rapidly advancing contagion and its fatality in cattle, sheep and goats, together with the immunity of horses, dogs, birds and human beings are important elements in diagnosis.

Malignant Catarrh of Cattle is distinguished by the absence of active contagion, one or two only in a herd being attacked ; by the absence of the curd-like concretions on lips, dental pad, and gums ; by the involving of the matrix of the frontal horns ; by the great congestion, swelling and discharge from the eyes ; and by the impossibility or improbability of the cattle plague contagion in the particular locality. In localities where cattle plague actually prevails, it may sometimes be impossible to distinguish at first, and then every precaution should be taken to prevent diffusion of infection in case it should be cattle plague.

Thrush of the mouth, though causing an eruption of the same kind, occurs in sucklings only, is unattended by fever, marked congestion, or severe abdominal symptoms.

Foot and Mouth Disease, though equally contagious, and spreading with the same rapidity, is easily distinguished by the very slight hyperthermia, the vesicular character of the mouth and mammary eruption (the cuticle being raised by an abundant clear

straw-colored exudate); by the absence, in nearly all cases, of severe abdominal disorder; by the all but constant vesiculation of the interdigital space, and by its mild and non-fatal issue. Swine contract it as readily as cattle and sheep, and horses, dogs, birds and men by inoculation.

Dysentery is marked by the absence of the early, abrupt hyperthermia, though the temperature may rise gradually to a high point; also of the curd-like concretions on the buccal mucosa; by the earlier onset of the fœtid diarrhœa, and by the indisposition to contagious diffusion apart from the confined, foul, crowded buildings. The lesions are mainly on the large intestines while in cattle plague they are on the small intestines, fourth stomach, mouth, throat and skin.

Gastro-enteritis and Stomatitis from corrosive agents and irritant bacteria, can be traced to a definite local cause, do not extend beyond the herd or animal poisoned, and usually occur where the possibility of cattle plague can be excluded. There is usually an entire absence of the white epithelial concretions, of the blush of the mucosæ generally, and of the wart-like epidermic proliferation.

Anthrax of the alimentary tract and rectum is distinguished by the fact that it is largely an enzootic disease, not spreading widely by simple contact; that it is easily transmisible to horse, dog and man; that it lacks the buccal epithelial concretions and characteristic desquamations and the warty-like skin products; that it shows marked enlargement and engorgement of the spleen; and that the blood and local lesions contain the large sized, square ended anthrax bacillus.

Diagnosis in Sheep and Goats. This is based on the same phenomena as in cattle; the sudden and exalted hyperthermia, blush or petechiæ of the visible mucosæ, concretions on the lips, gums and skin, epiphora, salivation, prostration, emaciation and diarrhœa. These last symptoms are, however, less marked than in cattle and the mortality and infectiousness are materially reduced. Pneumonic complications are much more common in sheep.

Mortality. Among native cattle in the steppes the mortality is 30 to 50 per cent. whereas elsewhere it is 90 to 95 per cent. Among sheep in Austria it reached 60 to 66 per cent. Among camels in Asia and Africa the fatality proved as high as among

cattle. The Italian buffalo usually recovered after seven days illness.

Treatment. The therapeutic treatment of cattle affected with cattle plague has been eminently unsatisfactory and is so certain to become a means of extension of the disease that it is legally prohibited in all countries, in which the plague has not been allowed to become generally diffused. Where it has become general in an unfenced country in which accordingly its permanence is virtually ensured, it may be employed.

Serumtherapy is advocated by Refix Bey. An animal is hyperimmunized by repeated inoculations. His blood is then withdrawn and the serum obtained from it is injected in a dose of 25 cc., subcutem, at a temperature of 104° F. The temperature is taken from the 3d to the 5th day and if it does not at this time rise above normal the treatment is ended. If still above normal a second dose of 25 cc. is given. It is claimed that the serum is harmless and may be safely given in a single dose of 50 cc. in case it is impossible to keep watch of the animal for five days.

Prevention by Immunization. Semmer attempted this by inoculating cattle with virus which had been weakened by heat or by passing it through the body of a Guinea pig. The results were, however, far from perfect and even in Russia the method failed of any wide acceptance. Koch and Edington in South Africa practiced extensive inoculations with a mixture of the virus in bile. Still better results are claimed by Danyoz, Bordet and Theiler in the Transvaal. These treated the animals by injecting 25 cc. to 50 cc. of highly immunized blood, defibrinated, and while the subjects were thus rendered temporarily insusceptible, they were exposed to infection by contact with diseased animals. In the Philippines a minimum virulent dose is given with the immunized blood.

As with serumtherapy, measures of this kind are only permissible in a country in which cattle plague is already generally diffused, and where there are no fences to limit the continued diffusion of the infection. The preservation of the cattle artificially infected until highly immunized, and again of the sick cattle requisite to give the disease to the cattle operated on, and finally of these last through the mild attacks that are to render them immune, affords an endless number of loopholes for the escape of contagion which would forbid the adoption of the

method whenever the extinction of the disease is possible. When on the other hand a disease is already spread universally in a country destitute of fenced enclosures, in which herd mingles with herd in the most perfect freedom, and where accordingly extinction is impossible, the method is admissible and even commendable as a means of reducing the otherwise ruinous mortality.

Exclusion and Extinction of Cattle Plague. For countries adjoining lands infected with cattle plague such general measures as the following are imperative : Prohibition of all imports of cattle, sheep and other ruminants, camels and swine from such infected countries, also of the fresh hides and other products of such animals, and of litter, fodder and other things that may have been stored in the buildings with infected cattle or otherwise soiled by them. Prohibition of imports of all cattle or other ruminants from adjacent countries (which may be plague-free), but through which animals have been carried in undisinfected cars or boats that had been in previous use for such species of animals drawn from infected countries, or that had been passed through infected countries, yards, buildings, loading banks, chutes, piers, gangways, or other places, or furnished with fodder, halters or appliances from such infected localities. In Eastern Europe the practice is to patrol the frontiers day and night to prevent the smuggling of cattle through from the infected country. Infected animals or herds, that it is sought to pass, are turned back or slaughtered. Sheep from countries that had been previously infected are often admitted on affidavit of the official veterinarian in the country from which they come, that during the three months before they left, there had been no contagious disease of cattle nor sheep in the locality, and on the further condition that they shall be slaughtered at the point of entry, or, if brought by rail, at the nearest slaughter house approached by such railroad after entry. In France, sheep, sent from Russia by sea, in French bottoms, certified as above by the Russian authorities, and accompanied on the voyage by a French veterinarian and certified sound by him, are allowed to circulate freely after three days detention at the point of arrival without evidence of disease.

In the United States the 90 days quarantine of cattle (dated from the time of shipping at the foreign port), and the 15 days

for sheep and other ruminants under strict veterinary supervision is safe as regards the importation of cattle plague in live animals. The greatest danger will doubtless come from intercourse with the Philippines, which were infected with the cattle plague during the recent war. The greatest possible precautions as regards the carriage of cattle on transports or other ships, will be necessary. Not only should no Philippine cattle be imported, but no vessel, that has carried Philippine cattle or sheep, should be allowed to take on board home cattle nor other ruminants until it has been thoroughly disinfected.

Cattle or sheep should be rejected when imported on ships which, on the same voyage or a recent one, carried fresh hides or other fresh products of animals, derived from a country in which rinderpest exists.

Hides that are thoroughly dried and salted, those that have been freely exposed for one week to the sun and air, and such as have been treated by active antiseptics, (caustic quicklime, mercuric chloride, lime chloride, formalin, phenic acid, etc.), need be held under no such restriction. The same applies to thoroughly dried, sunned and aired hair, wool, hoofs, horns, bones and sinews. Rendered tallow is equally safe.

Extinction of Cattle Plague in a Country. This should never be called for on the American Continent. The introduction of such a deadly disease, with such a short period of incubation, and such severe symptoms and rapid course, would argue a most reprehensible carelessness, which it is to be hoped will never be shown by the Federal Bureau of Animal Industry. Yet under the stress of a great European war this plague invariably overleaps the barriers successfully maintained in time of peace, and the same has happened to the two great English-speaking powers in connection with the wars in South Africa and the Philippines respectively and therefore it cannot be said that importation is impossible under any possible circumstances.

In case of a recent importation the infection would be easily controlled. Trace at once to its destination every bovine animal and other ruminant that arrived on the infected vessel, and all that came in contact with them since, or with the gangways, wharfs, streets, highways, yards, houses, fields, cars, loading banks and other places and things that might have been contami-

nated; stop all cattle and sheep traffic or movement for a large area around each centre of possible infection caused by such cattle; remorselessly kill every head of such animals carried by such vessel, or that came in direct contact with them; bury, burn, boil or dissolve in mineral acids, every animal thus exposed; thoroughly disinfect the importing ship, and every house, place or thing the imported stock came in contact with, together with all the dejections and debris, and even the surface of the graves; make a census of all cattle within the different quarantined areas, and hold the owners or custodians responsible, under a heavy penalty, to report every death and every case of illness; whenever the cattle plague is found in a place dispose of the entire herd as has already been done with the infected imported stock, and in a very few weeks the plague can be completely extirpated. The violence of the individual attack, and the very short period of latency, makes the work incomparably easier than the extinction of lung plague. There is never a long period of uncertainty (incubation), there is virtually never a slight or occult case of the disease, there is no equivocal chronic form of the affection. The attack is made boldly and above board, and can be met successfully if met promptly and energetically. The danger in such cases lies, less in the nature of the disease, than in the army of foolish, even if well meaning, meddlers, who denounce the temporary interference of trade, the payment of indemnities to the cattle owners, the interference with private property, the destruction of valuable thoroughbred herds, the interruption of the dairyman's business, the cost of disinfection, and a thousand other things, and who too often succeed in hampering and delaying action, until the infection has reached and spread over great unfenced territories thereby getting beyond control, or, short of this, has so established itself as to necessitate the outlay of a hundred thousand for every hundred that would have been demanded at first, and a long continued restriction of trade in place of the very transient interruption required by early, sharp, decisive action.

One of the most important prerequisites is that every state, but especially those on the seaboard and with ports of entry, should enact such laws as would make it possible for the Executive to act at a moment's notice and to call in the help of the Federal Government to make an early and effective application of the

only successful remedy. Most things can wait for the call, assembling and action of a legislature; the infection of cattle plague can not. Such laws are not superfluous. If never called for they still show a wise provision against a terrible, though remote, possibility; if really called for and they are not found ready, the great cattle industry and even the agriculture of the continent may be largely sacrificed by the neglect.

As guardian of the interests of The Philippines the United States is to-day called upon to consider the question of exterminating the disease which our interference brought upon the islands. On the unfenced lands of these islands we have to face on a smaller scale the problem of stamping out the plague which has baffled the wisdom of Europe and Asia. The individual islands may perhaps be taken independently, the cattle collected in small herds under fence, and by the sacrifice of a few the remainder of any herd that shows infection may be immunized, and the premises where they are confined disinfected until finally no more cases occur. But whatever method is adopted the seclusion of all within well fenced areas is the most important consideration. No nation has ever succeeded in extirpating this nor any other important infection in animals when they are allowed to run at large and mingle freely, herd with herd, on unfenced land.

SPIRILLOSIS IN GEESE.

Sakharoff in 1891 reported an epizootic of geese caused by a spiral anærobic microörganism (*spirochæte anserina*). Later, in 1899, Cantacuzene reported a similar affection and found that the leucocytes of the spleen were active in destroying the spirochæte. The symptoms of the affection were those of general dulness and prostration resulting in an early death.

The main lesions were found in the spleen and liver, the former greatly enlarged and engorged with blood, and the latter swollen, with fatty and even necrotic degeneration of the parenchyma. Similar lesions were found in the cardiac muscle and other solid organs. The spirillum was found in the blood as well as in the substance of the affected tissues. The spirochæte anserina is carried from goose to goose by an argas (Marchoux and Salimbene), that of relapsing fever of man by the bed-bug.

Immunization has been secured by injections of the blood of the diseased goose, after it has been heated to 55° C. for five minutes, but if heated for ten minutes the immunizing power was destroyed. The blood became nonvirulent forty eight hours after it had been removed from the live goose, but its injection into healthy geese produced immunity.

The channel for infection of the goose has not been proven, but prevention should be sought by the general methods, exclusion and quarantine of strange geese, the confinement of the healthy on uncontaminated ground, the supply of water from flowing fountains or close, deep, cemented wells or after boiling, the furnishing of green food from uninfected sources or after cooking, and finally the raising of new stock from the eggs washed in boiled water, hatched in incubators, and raised on cement, stone or wooden floors, and on sterilized food.

SPIRILLOSIS IN CATTLE IN EAST AFRICA.

Synonyms. Red-water, Diarrhœa, Fever.

Theiler describes a spirillosis of cattle prevailing in the Transvaal and E. Africa, and which has been hitherto confounded with

piroplasmosis (Texas fever). The piroplasma however is absent from the blood, while spirilla are present.

The *symptoms* resemble those of Texas fever: temperature 99° to 106 F., anorexia, weakness, debility, jaundice, petechiæ on the visible mucosæ, oppressed breathing under exertion, dulness on percussion of groin, pulmonary areas and the dependent parts of the chest.

Among the lesions are a deep icteric hue of the fat and white tissues generally; a flabby gelatinoid appearance of the adipose tissue due to a liquid exudate replacing the fat; effusion straw-colored or bloody into the pleural, pericardial and peritoneal cavities; dropsical swellings of the head and dewlap; petechial spots and more extensive hæmorrhagic patches on the serosæ especially; a firm brown condition of the liver; bile brownish and viscid; lymph glands enlarged, softened; blood contains basophile granules, and distorted, broken down globules (poikilocytosis).

Bacteriology. Spirillum Theileri. The microörganism is twisted in the form of a corkscrew, or the two ends may meet forming a ring, or again they may cross each other once forming a loop, or a second time forming the letter 8. It is usually attached to a blood corpuscle, white or red, or rolled round it. When free it moves with an undulating motion till it reaches another globule. The largest of the spirilla are 20μ to 30μ by $\frac{1}{4}$ to $\frac{1}{3}\mu$. There are very few observed in some cases, and in others as many as six may be detected in a single microscopic field. At intervals they completely disappear to be absent for perhaps ten days and then come in a fresh swarm, in the manner of the spirilla of relapsing fever of man. They can be stained by the common aniline dyes, viz. methylene-blue, fuchsin.

INTERNAL INVASIONS BY BACILLUS NECROPHORUS.

The *B. Necrophorus* has been successfully inoculated on, or found in pathological conditions in deer, reindeer, horses, asses, hogs, kangaroos, dogs, chickens, pigeons, rabbits, Guineapigs and mice.

It has been found in nodular masses of necrotic degeneration in the *livers* of deer, cattle (Mohler, McFadyean) and hogs

(Mettam, Edelmann); in the *lungs* of horses (McFadyean, Nielsen, Caudwell); in the *heart* (Bang, Mohler); in the *spleen* (Jensen, Georgewitsch); and in the voluntary muscles (Mohler, Caudwell). In a number of cases these internal colonies were referred to their primary locations or entrance channels in the feet, *umbilicus*, *skin* or other superficial part.

The microbe has been found in *necrotic stomatitis* in calves, and pigs (Mohler), and in lambs (Damman, Loffler, Diem). Also in lesions of *diphtheria of birds* (Ritter, Jensen, Leth).

The *ulcerative ano-vulvitis*, and *necrotic vaginitis* and *metritis* appear to have furnished the microörganism (Mohler, Ellinger). It has been found in *necrotic omphalo-phlebitis* in the young as well as in its sequelæ, *hepatic* and *pulmonary nodules*, and arthritis (McFadyean, Jensen, Olt, Mettam, etc.). It is sometimes present in the deeper layers of the *necrotic ulcers of hog cholera* (Schutz, Bang, Jensen, Th. Smith, Mohler). Among superficial necrotic lesions in the horse the microbe has been found in *quittor* (Bang, Gutenacker), in *necrotic dermatitis* (Fröhner, Davis) and *necrotic scratches* (Bang, Hell), and in *necrotic turbinated bones* (Cadiot).

It is impossible, however, to accept all of these as the result of the same unmodified organism. *Necrotic stomatitis* has followed *foot-rot* in the same place but far more commonly foot-rot in sheep will prevail without entailing any of these other forms, and the same is true of omphalitis; ano-vulvitis, and enzootic quittor. Under such circumstances each form spreads in the genus affected and largely or exclusively confines itself to the organ or organs which give the disease its name, without showing the disposition to attack other genera and organs. The different forms of the germ may own a common origin, as do the different types of bacillus tuberculosis, or the different Pasteurellas, but it is evident that in such a case they acquire new habits in other environments, and that, in most outbreaks, the main danger is along the narrow lines in which the microbe is manifesting its virulence at that time and in that place. Ano-vulvitis does not seriously threaten an outbreak of foot-rot, nor does omphalitis determine fowl-diphtheria. For clinical uses we can consider each different outbreak affecting but one genus and one set of organs, as a special morbid entity, to be dealt with according to its own special habits. See Contagious Foot Rot, p. 101.

ULCERATIVE ANO-VULVITIS.

This affection was recorded as prevailing in Iowa, Missouri, Kansas and Nebraska in 1897–8, and again in Iowa in 1900–1.

Causes. From the lesions Mohler has isolated a long rod shaped microbe which he identified with bacillus necrophorus of foot-rot. He has even taken the microbe from hog cholera ulcers and successfully inoculated it in the vulva of a healthy heifer. There remains, however, the difficulty that this disease prevails in one genus only, attacking but one system of organs, proving infectious along this narrow line, and respecting pigs, sheep and other animals living in the same yard or field. If it is the same microbe which operates specifically in omphalitis, foot-rot and hog cholera it has apparently taken on new pathogenic potencies and parted with many that it shows prominently under other circumstances.

The appearance of the disease in isolated herds which have had no known communication with other herds, and even in the young nonbreeding cattle to the exclusion of older ones, suggests an enzootic origin perhaps connected with microbian growths outside the animal body and the introduction of the microbes or toxins, in food, water or otherwise. Mediate contact through a bull serving in an affected and sound herd in succession has failed to convey the disease.

On the Rodkey farm at Blue Rapids, Marshall county, Kan., eight heifers from ten to fourteen months old, suffered, while the seventeen steers of the same age and the milch cows escaped. (Steddom). Near Shelby, Ia., a bull, from a healthy herd, broke into an affected herd and served cows there, and was afterward returned to his own herd and served cows there, but did not communicate the disease. (S. T. Miller) No case is recorded to show that any bull serving affected cows or heifers contracted ulcers or other diseases of sheath or penis.

In one herd near Shelby, Ia., nineteen head of cows and heifers suffered, while the four steers in the herd escaped. In another herd of twenty-six head, in the same district, the four cows and eight of the twenty-two steers suffered. It is not therefore, confined to the females. (S. T. Miller).

In all cases the disease appeared in the cooler months, from October to April inclusive, and while the cattle were secluded in muddy yards.

In different cases they occupied the yards in common with swine, which were charged with wounding the vulva, until the general character of the outbreak forbade this conclusion. At Blue Rapids it was sixty-two days after they had been yarded with the hogs before the disease was observed.

In this case the lot was small, poorly drained and very muddy most of the time. Calves and hogs drank from the same troughs until the disease appeared. The water supplied to the calves and hogs was from a well sixteen feet deep. The cows, which escaped, were supplied with water from the Blue River.

Cows, heifers and steers were fed on a ration of shelled corn 6 parts, rye 1 part, oats 1 part, and had an abundance of fodder, consisting of prairie hay and millet in equal parts. They were in good condition, some of them fat, and nearly all dehorned. A second herd had shelled corn, kafir corn and cane, with water from a shallow well, and all (cows and heifers) suffered.

As showing the localized nature of the cause, C. Muller adduces the case near Ottumwa, Ia., in which a herd of 30 calves were attacked, and sold out, the owner filling the same yard a few days later with 30 more bought in the surrounding country, and which he put on the same rations. In about 10 days the disease appeared in the second lot.

On the Rice farm, Blue Rapids, were 60 yearling heifers, bought in Kansas City, and two home cows. In the first week after arrival 5 heifers suffered, in the second week 20, in the third week 40, and in the fourth week all the 60. The two cows mingling with them were only slightly affected.

In the Rodkey farm, Blue Rapids, cases, all of the young cattle (which alone suffered) had been raised on the farm and had not been exposed to outside cattle.

Symptoms. "The ulcer, in almost every case, started as a mere abrasion, the size of a pin-head, usually on the inner surface of the labia, near the border of the inferior commissure, gradually eating its way through until it appeared as a much larger denuded surface on the outside." (C. Miller). S. T. Miller says: "The first noticeable symptom was serous exudate, rapidly forming into

a brown scab, under which was very fœtid pus, with extensive inflammation. The affection usually occurred on the lower portion of the lips of the vulva, in heifers and cows, and in steers around the anus or root of the tail. The scabs which formed seemed to spread very rapidly, destroying more and more of the underlying tissue and forming a thicker and thicker scab. The scab, if pulled off, would expose a raw surface which would bleed very readily. In a short time a new scab would be formed."

Steddom says: "The vulvar lips thickened and continued to discharge for four or five days. In the meantime certain pustules appeared, 0.1 to 2.5 centimeters in diameter. About the fifth day these ruptured and discharged yellowish pus." The mucous membranes of the vulva and vagina were dry and slightly congested. The ulcers were covered with brown, leathery scabs, which adhered tenaciously and when detached left an angry, red, purulent, granular, elevated and pitted surface. In some of the more severe cases, one or both lips of the vulva had sloughed off, and the sore had extended 10 to 15 centimeters on the skin of the escutcheon. The pus from this sore did not corrode the adjacent skin.

The more severe cases showed loss of appetite, constipation, hurried breathing, tucking up of the abdomen, with general dullness and dejection, and great tenderness of the affected skin with stiff, straddling gait.

Pathology. This is very obscure. The primary cause of the sores is not evident, though their occurrence on the anus and vulva only, and especially on the latter would suggest an elective affinity of the poison (microbian or chemical) for these structures and their products. If we assume a pathogenic microbe (Bacillus necrophorus) in the fæces, the question arises as to the cause of the habitual immunity of the steer, and of the margin of the anus in the majority of the affected heifers. The susceptibility of heifers rather than cows may imply a previous exposure and acquired immunity on the part of the mature animal.

Again if we suspect the existence of a necrobiotic agent of organic origin (like ergotin, secalin, sphacelin) we must assume a superadded microbian infection, implanted in the primary sore and rapidly extending it. The prompt recovery under antiseptic

treatment shows that no mere chemical poison maintains the destructive process, for it is manifestly the microbicide which puts a prompt limit to the disease, and under such treatment no sphacelating agent in the blood or tissues keeps up the advance of the ulceration or prevents healing.

Treatment. The ulcers healed rapidly under cleanliness and antiseptic applications. The tail, anus, and vulva were washed with tepid water, and then dressed with a solution of creolin (5:100), or carbolic acid (3:100), or mercuric chloride (1:500 or 1000). The ulcers were touched with a pencil of silver nitrate. S. T. Miller followed the sublimate lotion by the subjoined ointment: iodoform 20 grains, eucalyptol 40 minims, phenic acid 20 minims, petrolatum enough to make 2 oz. C. Miller in addition to the carbolic acid lotion applied the common white lotion (zinc sulphate 1 oz., lead acetate 1 oz., water 1 qt.) and used silver nitrate on the ulcers. Four dressings on four successive days were given and in the milder cases healing was completed in 10 or 12 days.

ACTINOBACILLOSIS.

Lignieres and Spitz describe a disease of cattle in Argentina, which resembles *actinomycosis*, in the organs invaded, the steady progress by the invasion of different tissues indiscriminately, the hardness of the advancing growth, the presence of minute granules or tufts of clubbed cells radiating from a centre, and the absence of any general poisoning by specific toxins. It differs in being due to a bacterium (bacillus) in place of actinomyces, in invading the adjacent lymph glands, and in being more easily inoculated.

It is reported by the discoverers, Lignieres and Spitz, as epizoötic in Argentina, while Nocard recognized it in France and Higgins in Canada.

Bacteriology. The microbe is rod shaped, 1 to 2μ by 0.4μ, ærobic, facultative anærobic, non-motile, non-sporulating, stains easily in phenic fuchsin, picro carmine, and acid violet, bleaches in Gram's, grows at 37° C. in peptonized bouillon without producing acid or odor, acidifies lactose and glucose, and dies in 10 minutes at 143.6° F.

Animals susceptible. Besides cattle, dogs, Guinea-pigs and rabbits are easily inoculated. In horses an abscess forms.

Symptoms resemble those of actinomycosis, but it attacks especially the skin and subcutaneous connective tissue, the liver, spleen and omentum, and rarely the tongue, heart, lungs, pharynx, lymph glands, mammæ, or facial bones. At first the size of a walnut, hard and insensible, they grow as large as the closed fist, soften and fluctuate. This softening tends to distinguish it from actinomycosis. The contents do not show filamentous forms. The serum of the victims agglutinates the actinobacillus cultures.

Actinobacillosis is more readily transmitted by contagion than actinomycosis.

Treatment by iodide of potassium is equally effective, (Nocard).

NOTE ON GOOSE SEPTICÆMIA.

The following is drawn from Cooper Curtice's bulletin on *Goose Septicæmia.*

This affection in 1900 caused a loss of 3,200 geese in July and August to Mr. Cornell, a Rhode Island owner. Mr Snell lost 500.

Bacteriology. The blood and tissues swarmed with a minute bacillus having the general morphological staining and biological characters of that of chicken cholera and rabbit septicæmia. It differs from these in the failure to infect chickens, whether inoculated or fed to them. It proved deadly to geese, ducks, pigeons, rabbits, mice, and more slowly to Guinea pigs. Geese were infected by inoculation or feeding of the germs, ducks from inoculation only.

Symptoms. The geese were often found dead, and even in those noticed ill, death supervened so early that no very diagnostic symptoms were made out. The affected geese moved tardily and unsteadily, and failed to keep with the remainder of the flock. Some burrowed the head in the dirt and twisted it around, indicating, it was supposed, spasms of the throat. Some were seized with the death agony in a few minutes; in others the illness

lasted for hours, and from experimental cases it was concluded that the period from infection to death, in the majority of cases, did not exceed thirty-six hours. Some were believed to merge into a chronic condition, but the owner thought that none recovered. No diarrhœa is noted.

Lesions. The head was the seat of marked venous and capillary congestion, suggesting asphyxia. The bill and throat contained a large quantity of tenacious mucus, which was especially viscid in the nose. Extravasated blood in abundance was present in the gastro-intestinal mucosa and contents, much of it more or less digested. At some points there was abundant mucous exudate; at others the folds were only marked by punctiform petechiæ, or by bloody patches formed by their coalescence. The cæca were usually normal.

The liver showed numerous punctiform extravasations and yellow patches of necrosis extending more or less deeply into the hepatic tissue. Other congestions were seen in individual cases, implicating, in one instance, the heart and pericardium, and in another the lungs. Petechiæ were frequent on the pericardium and other mucosæ. The blood was usually black, tarry, and with little disposition to brighten on exposure to the air.

Diagnosis. It is recognized as an infection of domestic water fowl by its attacking the larger proportion of that class of animals exposed to it. It is supposed that those which escape do so because of immunity due to a previous attack, or by reason of the absence of any wound of the mouth, throat or stomach by which the germ might enter. It is distinguished from *fowl (chicken) cholera* by the immunity of the chicken in this case. It is differentiated from Klein's *diarrhœal enteritis* of fowls, by the fact that neither pigeon nor rabbit is immune. From the *duck cholera* of Cornil and Loupet, it is diagnosed by the immunity of the chicken only, while the rodents and pigeon suffer.

The germ is manifestly one of the family of bacilli of the colon group, found in the different septicæmias, but sufficiently distinctive from these other forms, in its pathogenesis, to demand a separate place in connection with sanitary work.

Treatment is hopeless from our present point of view.

Prevention is the rational resort. In the case of those raising geese from the egg, it is imperative to abandon, for the season at

least, any pastures that may have become contaminated. It would be better still to subject such pastures to cultivated crops for one or two years. The pens should be thoroughly disinfected or abandoned and burned. Mr. Cornell used his infected pens for ducks without evil result. The drainage from infected pastures or pens must be guarded against, no geese nor ducks being allowed on land through which, or on which it passes, and no water receiving such drainage being employed for geese. In the case of feeders or handlers of geese who buy the birds in large numbers from many sources, a subsidiary quarantine should be constantly maintained, by enclosing the birds in as small groups as possible in separate pens, so that infection in one pen will not endanger the whole flock. When infection is shown in a pen, the diseased birds should be at once destroyed and burned, the pen thoroughly disinfected, and the other birds returned, or better, divided up into still smaller lots, so that infection showing in one of these will not endanger the great number taken from the original infected pen. The utmost care should be taken to maintain the most perfect cleanliness in the pens of exposed and suspected geese, and to sprinkle the floors and manure liberally with an antiseptic, such as a solution of sulphuric acid in water (2:100), or of phenic acid (3:100), or of a combination of the two. This will do much to prevent the hatching of flies which act as infection-bearers, and if these can be further excluded by screens the condition will be still more satisfactory. Vermin of all kinds should be excluded and whenever possible, separate feeders and attendants should be furnished for the suspected geese, and those that have not been exposed.

NOTE ON HÆMORRHAGIC SEPTICÆMIA IN CHICKENS AND TURKEYS.

Lucet describes a septicæmia of chickens and turkeys accompanied by dysenteric discharges. The microbe resembles the bacillus gallinarum of Klein (see Vol. II, p. 254), even in the immunity of pigeons and rabits when injected subcutem. Rabbits, however, suffer when injected intravenously. The probability is that this bacillus is identical with that of Klein. Yet in this whole class of microbes of the colon group, variations, apparently superinduced by environment, appear to result at times in

a deadly pathogenesis for different genera, and epizoötics differing from each other.

NOTE ON INFECTIOUS ANÆMIA OF THE HORSE.

Carré and Vallé report an infectious anæmia of the horse, as prevalent and almost constantly fatal in different parts of France and Alsace-Loraine. It is easily communicated by inoculating the blood or urine of the sick horse, the disease developing after an incubation of 25 days. The convalescent animal transmits the disease for months after apparent recovery, and his urine is still rich in albumen. The microbe survives freezing, but is killed by 212° F. It is ultramicroscopic, passing through a Pasteur filter. This germ leaves the infected system in the urine and probably in the manure and other secretions. Sanitary considerations would demand the scheduling and isolation of infected localities, forbidding the ingress or egress of solipeds; the disinfection of all stable dung and urine produced in such localities; its removal and plowing under by oxen; the destruction of the sick with suitable indemnity to the owners; and the thorough disinfection of all infected premises and objects. The greatest care should be taken to prevent diffusion of the infection through soiled fodder, litter, harness, drainage, etc., and water should be given boiled. Purchasers of solipeds should avoid not only the areas known to be infected but also the lands which receive their drainage. For treatment see Vol. I, Pernicious Anæmia, p. 528.

DISEASE, INFECTIVE OR TOXIC, TREATED IN EARLIER VOLUMES.

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