The four bovine scourges : pleuro-pneumonia, foot-and-mouth disease, cattle plague, tubercle (scrofula) : with an appendix on the inspection of live animals and meat / by Thomas Walley.

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THE FOUR BOVINE SCOURGES.

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FOUR BOVINE SCOURGES:

PLEURO-PNEUMONIA,
FOOT-AND-MOUTH DISEASE,
CATTLE PLAGUE,
TUBERCLE (SCROFULA).

With an Appendix

ON THE

INSPECTION OF LIVE ANIMALS AND MEAT.

BY

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TO

BAMPFIELD KETTLE, Esq., F.R.C.V.S.,

MARKET-DRAYTON;

AND TO THE MEMORY OF THE LATE

EDWARD STANLEY, Esq., M.R.C.V.S.,

BIRMINGHAM,

THIS WORK IS

Respectfully Dedicated,

IN TOKEN OF MANY KINDNESSES RECEIVED FROM THEM BY

THE AUTHOR,

AND OF MUCH USEFUL INFORMATION IMPARTED TO HIM

DURING HIS PUPILAGE.

PREFACE.

THE Author has on various occasions, and by numerous friends, been asked to publish a work on Cattle Pathology. From the many claims on his time and energies, he has been quite unable to comply with their request. In the publication of the present work, he hopes to partially meet their wishes.

He has chosen the four diseases—Pleuro-Pneumonia, Eczema Epizootica, Cattle Plague, and Tubercle, for his subjects; knowing, as he does, that they are of more importance to the stock-owner than all the other ills to which bovine flesh is heir.

The three first-mentioned diseases are only too well known, the last is less perfectly so, even, it may be said, to Veterinary Surgeons.

The matter contained in this work is, on the whole, a reproduction of the subject-matter of the lectures delivered by the Author on these diseases for the last six years in the Edinburgh Veterinary College.

Those who know nothing of Tubercle may question its claim to a place amongst what may be called *the* bovine scourges, but, as will be seen on studying it, it is a more insidious (and equally deadly) foe to the stock-owner than either of the other three diseases.

Bearing in mind that this work only claims to be regarded as a reproduction of lectures delivered, the Author has not wasted his time or attempted to exhaust the patience of his readers in discussing moot points; but has simply given a plain sketch of his own opinions and ideas, which have been founded upon a long and extended experience of the diseases in question. Into the nature of one of the maladies treated upon—Cattle Plague—the Author has not had the same opportunity of inquiring as has been afforded him with regard to the other diseases; consequently, he has not

been able to make such extended remarks on it as on them. One great end he has had in view has been to so arrange his matter as to enable every Veterinary Surgeon or Student to distinguish the character of the diseases which he describes; and for this purpose he has introduced a number of drawings—coloured and uncoloured—all of which (except those on Cattle Plague,* which are copied from Dr. Smart's Report, and from the Third Report of the Cattle Plague Commissioners, 1866) have been executed—from specimens prepared by himself—under his personal supervision. For their assistance in this part of the work, the Author has to record his thanks to Professor VAUGHAN for the drawings which he executed when a student at the College; to GEORGE AIKMAN; and to GEORGE CRAIG. He has also to express his indebtedness to Professor M'FADYEAN, Mr. WM. REEKIE, M.R.C.V.S., and Mr. FRANK F. MAVOR, for much valuable assistance in the general preparation of the work.

* Specimens which were obtained by the Author for the purpose of sketches were inadvertently destroyed.

ZYMOTIC PLEURO-PNEUMONIA.

ABBREVIATIONS—viz., Zy. p. p., indicating Zymotic Pleuro-Pneumonia; P. p., Sporadic Pleuro-Pneumonia; and P. h., Purpura Hæmorrhagica—will be used for the sake of brevity.

SYNONYMS.—Technically, we have Pleuro-Pneumonia Epizootica; P. p. Contagiosa; Exudative p. p.

Provincially: Lung Disease, or Lung Ill; Distemper; Pleura; Jig; Tick; Goat; Gasper.

Of the technical synonyms of this disease, I prefer that of P. p. epizootica; or, as I shall hereafter speak of it, P. p. zymotica.

Of the provincial synonyms, lung disease is most frequently used, and it certainly is the most expressive. Distemper is applied also to the disease in many parts of the Midland Counties. The other terms are simply of a slang nature, and are used by common butchers. Goat is nearly always employed to designate the disease in the neighbourhood of Edinburgh; and those who are engaged in the traffic of the diseased flesh are known as "goat hunters," or "blockade runners"—the latter term signifying that they have to run the gauntlet of inspection; "goat" is further distinguished as wet and dry—the former implying that there is a large amount of effusion into the chest and the tissues; the latter, that there is an absence of effusion. "Gasper" is applied to those cases in which gasping for breath is a prominent symptom.

DEFINITION.—It is an insidious, exudative zymotic disease, due to a specific poison or ferment, peculiar to the ox, and having its local manifestations concentrated in the lungs and pleura.

CHARACTERS.—P. p. zy. is indigenous in the ox alone; so far as I am aware, it is never developed in any other animal; neither, in my experience, is it transmissible to any other animal. Some authorities have asserted that they have witnessed it in the sheep and pig, and, in one or two instances, in the horse. I may be pardoned if I express the opinion that the cases referred to had no direct relation to Zy. p. p., that they were, in fact, cases of catarrhal inflammation of the lungs in the third stage; or

cases of blood poisoning accompanied by asthenic exudations into the structure of the lungs.

In the *Veterinary Journal* for October, 1878, the Editor draws attention (p. 239) to a recent report of the German Sanitary Board, in which it is stated that Zy. p. p. broke out in the Brussels Zoological Gardens in 1877, and attacked two yaks, three bisons, and one buffalo,—the course of the disease in the yaks and bisons being rapid; and the pathological anatomy, in all, very distinctive.

No age, breed, or class, neither sex of the bovine species, enjoys immunity from the effects of the virus of this disease; and although, as will be hereafter shown, many animals escape the infection, we cannot assign any satisfactory local cause, or combination of causes, for this immunity; it is in accordance with the well-known law, that a certain (variable) percentage of animals prove refractory to every virus.

It is one of the most insidious diseases with which we are acquainted, and I do not hesitate to assert that it has been the cause of greater losses to British stock-owners and dairymen than any other single disease to which animals are subject. No estimate—not even an approximative one—could possibly be made of the losses sustained by it, for the simple reason, that thousands of cases never come under the cognisance of the authorities.

In its general pathological characters it differs very materially from Sporadic or Sthenic Pleuro-Pneumonia, and in nothing so much as in the fact, that its initial lesions are of an effusive, while those of P. p. are of a congestive nature.

Elsewhere, I have endeavoured to show that any serious derangement in the balance of the sanguiferous constituents inevitably leads to one or more of three conditions, viz., *effusion of serum, exudation of lymph,* or *extravasation of blood;* and that similar results are induced by depravity of the nutrient fluid—*i.e.,* by the introduction into it of organic or septic germs. Extravasation of blood is more likely to result from this cause, owing to nutritive interference with, and structural alteration of, the capillary walls, which are thereby rendered incapable of withstanding the pressure of the blood in their interior, give way, and allow of the passage of their contents into the surrounding tissues, the vitality of which is frequently, and, more often than otherwise, permanently destroyed.

In the disease under consideration, effusion, exudation, and extravasation are all present, and result from the zymotic "action," in the blood, of a specific virus or germ, of the actual nature of which we know little.

That it is a true zymotic disease, and distinct from P. p., I think I shall have no difficulty in proving; and further, that with the exception of the latter being devoid (as a rule) of infectious or contagious attributes, it is (at least in its results) intimately allied to Purpura Hæmorrhagica of the horse.

As to the character or residence of the propagating germ or virus of Zy. p. p., I have not yet sufficient evidence to enable me to speak with absolute certainty; but I have been enabled to detect spherical refractive micrococci, which in a day or two

actively oscillate, in the secondary pleural exudations, the parenchyma of the lungs, and in the local effusions and exudations produced by inoculation with the lymph of Zy. p. p. In the systemic muscles I have also observed, microscopically, similar bodies. To the latter fact, however, I do not attach much importance, as in this situation micrococci are seen in many zymotic affections.

As to the differential characteristics of P. p. and Zy. p. p., I shall in this place call attention, principally, to those which are observable during the life of the animal. P. p. is of a sthenic, Zy. p. p. of an asthenic character; P. p. occurs only as a sporadic, Zy. p. p. as an epizootic affection. In P. p. abortion seldom takes place; in Zy. p. p. it is frequent; in P. p. the lung (or its central portions) is attacked *en masse*, and shades off at the peripheries; in Zy. p. p. it is diseased in patches (always defined and more or less circumscribed), frequently located in the borders, and indicates different periods of existence. I have seen as many as seven distinct centres, varying in size from a walnut to a child's head, in the lungs of one animal. P. p. is not accompanied by albuminous or serous effusions into the muscular tissue of the body, neither are the lymphatic glands of the thorax so much diseased; while the inflammatory products are not so acrid in their character as in Zy. p. p.

In P. p. there is seldom any want of correlation between the pulse, respiration, and temperature; in Zy p. p. the pulse may register 60, and the respiration 12 per minute; while the temperature may run up to 105° or 106°. In P. p. the cough is comparatively strong and bold; in Zy. p. p. short, sharp, and laryngeal, and the grunt of the latter is always absent in the former disease. If there is expectoration, the expectorate is rust-coloured, or largely mixed with blood in P. p.; while in Zy. p. p. it consists of fibrin casts often streaked with blood, or mixed with small coagula. In P. p. the symptoms are sthenic, the attack sudden, and preceded by congestion; the injured lung tends to *organative* changes, and may be functionally restored, and the disease exhibits a certain amount of amenability to the action of medicinal agents. In Zy. p. p. the symptoms are little marked; the attack is insidious, and not preceded by congestion; the injured lung tends to *degenerative* changes, and is never functionally restored; while the progress of the disease is very slightly controlled by the action of medicines. P. p. has no incubative stage, and is neither infectious nor contagious; Zy. p. p. has a well-defined, though extremely uncertain, incubatory stage, and is alike contagious (by actual cohabitation) and infectious. In P. p. the primary effusion is of a plastic, in Zy. p. p. of an aplastic character.

The *post-mortem* differences will be considered hereafter.

In the analysis of the relative characters of Zy. p. p. and P. h., we observe that they are both the result of a morbid or poisoned condition of the blood, either from atmospheric germs, self-propagation of deleterious matter within the blood, or a loss of equilibrium in its constituents; thereby rendering it incapable of preserving its own integrity or that of the tissues it supplies. Both require much the same course of

treatment; both are most virulent after debilitating diseases; both are most prevalent in unhealthy or impure states of the atmosphere; each is marked by local or systemic dropsies, exudations, or extravasations; both are notable for the absence, primarily, of inflammatory symptoms; both show a tendency to terminate in vicarious Diarrhœa; in each the victim dies either from Asphyxia, as the result of serous effusion into the bronchia —which effusions become converted into foam by the passing to and fro of the air in respiration—or from non-oxidation of the blood owing to the amount of lung structure destroyed. In both (*i.e.*, when the lungs are affected in P. h.) the earlier *post-mortem* symptoms are analogous; any slight difference which may exist being accounted for by the variation in the structure of the lungs in the two animals. In each, minute stellate-shaped hyperæmic spots exist in the lungs, due to congestion within, and ultimately rupture of coats of the plexuses of vessels around the air-cells, with their converging and diverging capillaries. In both the bronchial mucous membrane will be found studded with scarlet spots (*ecchymoses*); and frequently mucus, mixed with blood, is lodged in the minute bronchia: this condition more particularly existing in the sound—or comparatively sound—portions of lung. In Zy. p. p. the larger ecchymoses in the tracheal and bronchial mucous membrane undergo an exactly similar change (viz., degeneration and ulceration) as do those in the Schneiderian and bronchial membranes in P. h. In P. h. the lungs present as distinctly a "marbled" character—allowing for the difference in structure—as they do in Zy. p. p., and are nearly as heavy and as full of serum. The disintegrated condition of the muscular fasciculi, where ecchymosis exists in P. h., has its analogue in the condition of the parenchyma of the lung in Zy. p. p. In each the systemic muscles are altered in character,—*i.e.*, are carbonaceous or dropsical; and the lungs, unless they are only slightly affected, are never restored to their original condition. In both the pulmonary lobules are destroyed, not only by the pressure of the interlobular effusion, and subsequent exudation and hyperplasy; but by intralobular effusion and extravasation.

We do not see local external sloughs in Zy. p. p., as in P. h., simply because muscular extravasation seldom takes place in the superficies of the body in the former. The marked difference in the two affections lies in the fact that P. h. primarily tends to local and external manifestations of its existence, terminating in Septicæmic Pneumonia or vicarious enteric disease; the reverse being the case in Zy. p. p., except in some instances where death follows as the result of Diarrhœa, brought on by the same vicarious enteric glandular affection as in P. h.

As I shall hereafter show, the disease is highly infectious—not contagious in the ordinary acceptation of the term—and is propagated in a great variety of ways; and although its usual tendency is of an epizootic, it sometimes assumes an enzootic character.

It is not hereditary in any sense of the term, *and it is questionable if it is congenital.* While not pretending to assert that Zy. p. p. is never developed congenitally

in the offspring of diseased animals, I must candidly confess that no instance of the kind has ever come under my own observation. There are, however—if my memory serves me right—one or two instances of supposed congenital transmission of the disease on record.

It has been often asserted that in Zy. p. p. one lung is more frequently attacked than the other, the preference being given sometimes to the right, at others to the left.

In my experience one lung has been as often affected as the other, and if a record of a given number of cases were kept, it would in the end be found that there would be no preponderance in favour of one over the other ; in not a few cases we find both lungs either primarily or secondarily affected in a greater or less degree.

In this, as in all other zymotic diseases in which there is a tendency to effusions, previous disease will determine the localisation of the lesions.

One attack *does not* give immunity from another.

HISTORY.—The history of this disease teaches us without doubt that it was originally of foreign origin, and it has been ascertained that, in all great and widely spreading epizootics, the virus has been introduced by foreign animals ; and while it has been known for very many years in Great Britain, long periods have occurred in which it would seem to have been entirely absent. One such notable period dates from 1757 to 1842 ; but since the latter year it has not been eradicated from our island.

According to Fleming, the disease was not definitely known until it was described by Bourgelat, a French veterinary surgeon, in 1769 ; though, according to other authorities, as above mentioned, it had a much earlier existence.

GEOGRAPHICAL DISTRIBUTION.—Pleuro-Pneumonia Zymotica either does exist, or has existed, at different periods, in most European countries, and in some of the colonies.

VITALITY OF THE VIRUS.—Is very great ; under favourable circumstances it may preserve its virulence for twelve months, or even more. This is proved by the fact that healthy animals brought into infected byres, after they have lain empty for the period mentioned, frequently contract the disease.

PERIOD OF INCUBATION.—Is more indefinite than in any other zymotic disease, with the exception of Glanders and Rabies. While the average period may be stated at from three weeks to three months, it very frequently happens that it is prolonged to four or six months, and in some instances more.

INVASION.—Is very insidious. Few external symptoms indicative of serious constitutional disturbance may be presented, and yet the disease may be well advanced. It is this insidious nature of its invasion which sometimes enables it to obtain such firm hold in cattle before any serious mischief is apprehended.

DURATION — *i. e.*, the period which elapses between the development of the symptoms and death, or the subsidence of the primary destructive processes, is from

ten days to twenty-one. The period during which an animal may suffer from the effects of the disease is extremely indefinite.

FATALITY.—Is very great, though this depends, to some extent, upon a variety of circumstances; thus, some outbreaks are marked by a greater degree of virulence than others, and this is specially the case on the first introduction of the disease into a country or district, or amongst herds which have been subjected to the lowering effects of prolonged travelling, and also amongst those whose hygienic surroundings are eminently unfavourable to the preservation of health. One remarkable feature—noticed also in Epizootic Eczema—this disease possesses, viz., that it shows a great tendency to *periodical declinations and exacerbations in its virulence and spread.* In the most malignant outbreaks, and those in which the disease is allowed to run riot, the fatality reaches 80 or 90 per cent. In the milder outbreaks it is reduced to 30 or 40 per cent. Careful attention and judicious medication undoubtedly control the fatality of the disease to an appreciable extent.

ORIGIN OF PLEURO-PNEUMONIA ZYMOTICA.

The differences in opinion which are entertained as to the origin of Pleuro-Pneumonia Zymotica are as numerous and diverse as are those which exist with reference to the development of zymotic diseases in general; and many and insurmountable difficulties stand in the way and prevent our arriving at any satisfactory conclusion. One great barrier which has stood in the way of solving this difficulty has been the propagation of the theory, that all zymotic diseases are due to the action of *recognisable living organisms,* whether these organisms be vegetable or animal, spore or bacterium; but I imagine that this difficulty is now being gradually dissipated by the discoveries which have been made, that it is not, in every instance, those fluids in which determinate forms can be detected by the aid of the microscope which possess the most virulent properties; but that, in fact, the reverse is often the case. Thus, amongst the most recent inquirers into this subject, we have Dr. HILLER, of Berlin, who has found, as the result of a large number of vaccinations (740) in recruits, that "there is no relation between the microscopical characters of the lymph and the appearances recorded on inspection."

Dr. TYNDALL has recently arrived at the conclusion, that it is not when bacteria are fully developed in putrefactive fluids that the great difficulty in sterilisation is encountered; but rather, that the greatest resistance to the action of destructive agents is observed before bacteria are developed, or at the time when "the *germ* is in its resistant stage;" and that it is most easily destroyed when it has passed into "its plastic and sensitive stage,"—or, in other words, into bacteria.

TOUSSAINT, Professor of Physiology at the Toulouse Veterinary School—in a note on Anthrax in the Dog, presented to the Paris Academie des Sciences, translated by

"R. S. L.," veterinary student, Toulouse, and published in the *Veterinary Journal*, August, 1878—expresses himself much to the same effect. Speaking of the phlogogenous powers of *bacteridiæ* or their products, he says: "The local effects due to these organisms appear to me to result from the presence of a soluble matter (*diastase?*) secreted or excreted by the parasites, and which enjoys to a high degree—though this varies according to the species (of animal, T. W.) which nourish the bacteridiæ—phlogogenous properties."

Dr. WILLIAM STRANGE, in an article on Puerperal Septicæmia (*Lancet*, 27th July, 1878), says: "Now, I take it as proved that it is the growth of bacteria in these fluids which makes them so deadly. And not alone the *growth* of bacteria, for they by themselves are harmless enough, since they enter the blood continually in thousands without producing any ill effects; but the bacteria, when planted in a suitable medium, such as decomposed blood or the products of inflammation, produce the septic poison, it may be by fermentation, it may be by secretion; and thus, although bacteria are of themselves innocent, there can be no septicæmia where they are not or have not lately been present."

Dr. BURDEN SANDERSON says,* "that the ground which the orthodox biologist holds now, as against the heterodox (*i.e.*, those who believe in the heterogenous development of disease germs), is not, *that every bacterium must have been born of another bacterium*, but that *every bacterium must have been born of something which emanated from another bacterium;* that something not being assumed to be endowed with structure in the *morphological or anatomical sense*, but only in the *molecular or chemical sense.*"

He further says, that "germs have given place to things which are ultra-microscopical; to molecular aggregates, of which all that we can say is what we have already said about ferments—*that they occupy the border-land between the living and non-living things;* and that the organic forms which occur in contagious liquids can only be looked upon as characteristic concomitants of the specific process with which they are associated." He would not, however, have us regard them as unimportant; for he says: "*Just as the crystalline structure of bodies is now known to have relation with their molecular constitution* (of which the chemists of old knew nothing), so it may turn out at no very distant date, that *relations of a most important and interesting kind may be discovered between the specific contagia and their morphological characteristics.*"

Again, in alluding to the nature of ferments generally, Dr. SANDERSON says: "It is characteristic of all ferments that they induce changes in other bodies without being themselves acted upon,—a kind of action which nothing that is known of the action of molecules serves to explain. In defining the nature of ferment action, we are therefore in a dilemma, out of which there is no escape except by compromise. A ferment is not an organism, because it has no structure. It is not a chemical body,

* "*Veterinarian*," August, 1877.

because, when it acts on other bodies, it maintains its own molecular integrity. On the whole, it resembles a chemical body, for its characteristic behaviour is such as, if it had a structure, would prove it to be living. What is more characteristic of living protoplasm than that, while maintaining its own integrity, it alters the surrounding medium."

Some such views as these were expressed by Dr. M'DOUGALL, in an exposition delivered some time ago at a meeting of the Pathological Society of London, in advocating the chemico-physical theory of the origin of the specific diseases. The great difference, however, between him and his opponents lies in the fact, that he will not entertain the germ theory at all, but says, "that contagious units are fragments of dead organic matter, whose elementary particles are in some occult state of chemical union, and capable of imparting their condition to other bodies susceptible of the change."

Thus I again assert that one great barrier in arriving at a satisfactory conclusion in connection with the nature of disease-producing materials, has been done away with, and that in all probability it will be found, as I have long felt, and as has been so well described by Sanderson, that disease-producing germs cannot be considered in the same light as organisms possessing a recognisable anatomical or morphological constitution. I have sometimes thought that the term *active principle* would be far more appropriate than that of germ.

Do Pleuro-Pneumonia and its congeners ever arise spontaneously (autogenetically)?

This question, in the present state of our knowledge on the subject of spontaneous generation, cannot be answered authoritatively.

Whatever may be the result of the experimental controversy on the subject presently raging, there will still have to be met clinical facts which, pointing to the spontaneous development of zymotic diseases, cannot be ignored. In the history of Pleuro-Pneumonia I have shown that it is believed, primarily, to have had a foreign origin. Granted that such is the case (we know little of the history of special diseases in ancient times), is that any argument against the possibility of its being generated in our own country? I see no good grounds for entertaining such an opinion; given the concatenation of circumstances necessary for its production, it may as well originate in Great Britain as in any other country.

Time was when neither Glanders or Rabies were believed to have, under any circumstances, a spontaneous origin. Clinical facts coming under the observation of careful and experienced veterinary surgeons have proved that both these diseases may, and do, originate spontaneously; and in the development of Splenic Fever we have equally as strong and convincing a proof of the autogenous development of zymotic disease.

Only recently has it been acknowledged that Splenic Fever is infectious and contagious; numerous now are the proofs that such is the case,—one very notable instance of the kind, the notes of which I have in my possession, was brought under my notice by the late Mr. DEWAR, of Midmar; and that Splenic Fever frequently originates anew there can be no doubt, for of late it has made its appearance in districts far remote from any previous outbreak, and in *home-bred stocks.* I am quite aware that it may be urged that the vitality of germ-life is so wonderful that these new outbreaks may be traced to germs that have lain dormant and undisturbed for an indefinite number of years; but this is, indeed, stretching a point, especially when we consider that such a disease is unknown to the memory of the oldest inhabitants of some of the districts in which it now makes its appearance.

Speaking only very recently (at the Sanitary Congress of Leamington), Dr. RICHARDSON said "that the germs of zymotic disease are developed within the glandular system of animals." Thus, he asserts, "under certain influences affecting glandular action, the poisons may be made to originate *directly,* through nervous impression, *without the intervention of an infecting particle,*—extreme nervous depression acting on the glandular nervous supply, paralysing the glandular function, and altering the characters of glandular secretions."

What may be the precise concatenation of circumstances in existence now to produce certain zymotic diseases it is perhaps difficult to say with any approach to accuracy, but I think we may look for an explanation, to a great extent at any rate, to the difference in the general management of stock at the present day as compared with that of the past,—artificial feeding, artificial agriculture, artificial manuring, artificial breeding, each contributes its quota towards a combination of circumstances which may, which must, be inimical to the general welfare of animals.

Reverting to Zy. p. p., while I am not prepared to bring forward any direct proof of its spontaneous generation, I may be permitted to say that circumstances came under my observation in the earlier days of my professional career which appeared to me to support the idea of spontaneous generation. Many of the veterinary surgeons in the north of Scotland hold very strong views in favour of autogenous development, and the disease certainly makes its appearance under such remarkable circumstances as to enable us to say that if it is not developed spontaneously, its germs have a wonderful tenacity of life; and, what is almost of more importance, to enable us to say that it is propagated by *mediate* as well as by *direct infection* or *contagion.*

As bearing very closely on this question, I think I may advantageously direct attention to the fact, that the medical profession now recognises the spontaneous origin —from exposure to certain effluvia—of a form of Pneumonia in the human subject which is propagated by contagion, and whose lesions are totally distinct from those of Sporadic Pneumonia.

Is Pleuro-Pneumonia an Infectious or a Contagious Disease, or both?

Before attempting to answer this question we must inquire briefly into the application of the words infectious and contagious, though I may remark in passing that the two terms are now often used indifferently.

The great majority of experts define an infectious disease to be one which is propagated by the pulmonary and cutaneous *exhalation*, and the pulmonary and cutaneous *inhalation* of the germs of the particular disease; and a contagious disease, one in which there must be actual contact of fluid or solid matter, containing the elements necessary for the propagation of a disease, with a living body; and further, one in which the virulent principle can be located in a particular solid or fluid of the body, and which will with certainty be reproduced in the system of a healthy animal when it gains access thereto, and in its reproduction will give rise to the development of local manifestations exactly identical (only differing in degree) with those which marked its existence in the system of the original host.

With which of these definitions does the nature of Zy. p. p. agree? I answer, unhesitatingly and unqualifiedly, with the former. If we take the fluid from a small-pox or vaccine vesicle and introduce it into the system of a healthy animal, we produce modified—but distinctive—Small-pox and Vaccina. Can we make the same assertion with regard to Zy. p. p.? No; for up to the present time (so far as I am aware) no person has succeeded in producing a *true* form of the disease by the artificial introduction into the system of a healthy animal of any product or products derived from the body of an infected beast.

The opinion has been promulgated, dogmatically, by some veterinary surgeons, that Zy. p. p. is *only* propagated by "the actual cohabitation of healthy with diseased animals." Is this in accordance with clinical experience? Certainly not. It may be in accordance with experimental experience; but, unfortunately, experiments are too often fallacious in their teachings,* and although they are in many ways extremely valuable, their results cannot be placed in opposition to clinical facts—one natural positive proof is worth a myriad of artificial negative ones.

In a very large experience, dating from my earliest connection with the profession, and gained in extensive stock-raising and dairying districts, I have seen outbreaks of the disease occurring on farms which were perfectly isolated, and in which the whole of the stock was home-bred. I have seen dairy after dairy decimated of its denizens, and, after every precaution in the way of disinfection had been taken, and the byres left empty for periods varying from three to twelve months, the disease again break

* The truth of this assertion has never been more forcibly proved than in the result of the experiments lately carried on at the Brown Institute in connection with the nature of Foot-and-Mouth Disease.

out on the re-introduction of fresh stock, even though the animals had been brought from districts in which the disease was unknown and although every possibility of their coming in direct contact with diseased animals had been carefully guarded against.

In a matter of so much importance, I have thought it advisable not to allow my opinion to rest on its own merits, but have taken the pains of obtaining the views of many veterinary surgeons, whose extensive experience of the disease entitles them to speak with some authority on the subject. These views I here introduce :—

My colleague, Mr. BAIRD, says that he has seen the disease make its appearance in remote and isolated districts in Fifeshire, where the stock had been home-bred for many years, and where the animals had never come in contact with others.

Messrs. PETER TAYLOR, Manchester; BLAKEWAY, Stourbridge; ROBERTSON, Kelso; M'GILLIVRAY, Banff; THOMSON, Aberdeen; CARLESS, Stafford; DAYUS, Dorrington; EDWARDS, St. Alban's; ALEXANDER GREY, sen., Edinburgh; WAUGH, Stirling; STORRAR, Chester; AITKEN, Dalkeith; CUNNINGHAM, Slateford; REID, Leith; RUTHERFORD, Edinburgh; GOOD, Ludlow; WELSBY, West Derby; KETTLE, Market-Drayton; Professor WILLIAMS; the late Mr. DEWAR, Midmar; DUNLOP, KIDNEY, and GIFFEN, Belfast; DORIS, Cookstown; KERR, Ballymena; all concur in the opinion that Zy. p. p. is propagated by other means than by actual cohabitation.

Mr. CARLESS says: "Zy. p. p. can be propagated by exposing healthy animals to the excrements of those which are diseased (especially in the advanced stages of the disease); also by hay, straw, &c., kept in the place with them, as easily as by direct cohabitation."

Mr. M'GILLIVRAY says: "Although I consider actual cohabitation the most common and the most easy way in which Zy. p. p. is propagated, my experience proves that it is also spread by indirect means; as by straw, hay, dung, &c., which have been in contact with diseased animals. This is indirectly proved by the fact that healthy cattle brought into sheds from which diseased ones have long been removed become the victims of the disease." He further quotes a remarkable outbreak (in support of this assertion) which came under his notice three years ago. A farmer in Mr. M'Gillivray's neighbourhood had Zy. p. p. amongst his stock, but, after losing a tolerable number, he got clear, disinfected his byres, and also plastered the walls. Twelve months after, the rats infested the whole of the farm buildings, and, along with other mischief, made extensive operations on the old walls; tunnelling holes through them in every direction, and turning out a good deal of the plaster from the old crevices. This debris fell into the forestalls out of which the new stock of cattle were feeding; the result was a fresh outbreak of Zy. p. p. Mr. M'Gillivray took great pains to ascertain the possibility, or otherwise, of this outbreak being due to any other cause than the one cited, but failed to find any.

Mr. WAUGH says: "I have seen the disease break out in a large stock, housed in

byres from which diseased cattle had been previously removed, twelve months after the new stock had been put in."

Mr. JOHN AITKEN has related to me a case in which a gentleman had two cows in his possession,—one for four, the other for three years; the former was sold fat, and a short time afterwards the latter evinced symptoms of the disease, and was slaughtered. These animals were effectually isolated by a high wall, and it was proved that no other cattle had come in contact with them.

Professor WILLIAMS says: "I have seen the disease break out in a byre which had stood empty for a period of three months; every precaution having been taken to avoid contact with other animals in the introduction of the new stock."

Mr. JAMES THOMSON says: "I am decidedly of opinion that Zy. p. p. is not propagated by any other means than by the air becoming impregnated with the breath of a diseased animal, and subsequently inhaled by a healthy one. I believe that the infecting distance is greater than is generally supposed; but it is necessarily much influenced by currents in the byres, and the strength and direction of the wind in the open fields."

Mr. STORRAR says: "I believe that Zymotic Pleuro-Pneumonia can be, and often is, propagated by other means than by direct cohabitation. In my own practice I was satisfied that a woman who attended a diseased stock was the means of communicating it to healthy animals."

The late Mr. WM. DEWAR said: "I am convinced that P. p. zy. is frequently propagated by other means than by direct cohabitation (if required, I can prove the assertion). I believe it to be propagated by the dead animal, and by excrementitious matter."

Mr. GEORGE FLEMING has recently communicated to me the history of an outbreak on the farm of an intimate friend of his in Staffordshire, in which no possible means of infection could be traced, except by the indirect means of a butcher.

Mr. H. EDWARDS says: "I had a cow in my possession for a period of several years; my brother requiring a cow, and not being able to find one to his taste, I offered him mine; he accepted her, she was removed to his house, a distance of four miles (totally isolated from all others); he had her two years in his possession. In December, 1874, she was attacked by Zy. p. p., for which, as inspector, I ordered her to be killed, and afterwards made a *post-mortem* examination. Zy. p. p. did not exist, neither had it existed for a very long time, within my radius as inspector."

In the *Veterinary Journal* for December, 1876, E. F. THAYER, V.S., Commissioner of Contagious Diseases among Cattle, Massachusetts, U.S.A., says: "P. p. zy. was first imported into Massachusetts in 1859, from Holland, and by the sale of cattle was carried to different sections of the State. The first outbreak occurred sixty miles distant from the farm where the Dutch cattle were kept, confirming the opinion of those who believed in the contagiousness of the disease." Mr. Thayer further quotes

the opinions of Delafond, of France; Hertwig, of Berlin; Verheyen, of Brussels; Ischeulins and Hermann, of Switzerland, in support of the view that the disease is spread by other means than by direct cohabitation; and he also asserts that "the experiments made in Massachusetts, by the order of the Governor and Council, were conclusive of the contagiousness of the disease; also, of the fact that an animal should be in the early stages of the disease in order to convey it to others."

The controversy between the Magistrates of Shrewsbury and their Inspector, Mr. Litt, on the one hand; and the Privy Council Authorities, on the other, is so recent as to render it unnecessary for me to allude to it any further than to say that Mr. Litt adhered to the opinion which he had given, that Zy. p. p. was propagated by other means than by direct cohabitation,—as by hay, straw, dung, &c.; and that in this opinion he was firmly supported by the Magistrates.

In the *Veterinary Journal* for August, 1876, the Editor, Mr. GEORGE FLEMING, quotes from a paper on the subject, by Mr. Lydtin, of Eppingen, Baden (a gentleman with whom I am acquainted, and whose intelligence is above the average), a remarkable instance of propagation of the disease by means of dealers carrying with them flesh from a diseased animal, and leaving it on healthy premises. Mr. FLEMING further remarks that several instances of propagation of the disease by means of flesh are recorded by foreign veterinary surgeons.

Finally, some support is given to the idea of propagation of the disease by mediate means by the fact that the "Contagious Diseases (Animals) Act, 1878" contains a provision that no dung, fodder, or litter which has been in contact with diseased animals shall be removed until it has been, as far as practicable, disinfected; though, of course, this provision may have been only inserted as a precautionary measure.

CIRCUMSTANCES FAVOURING THE PROPAGATION AND EXTENSION OF PLEURO-PNEUMONIA.

These are, on the whole, similar to those which favour the propagation and spread of all zymotic diseases.

They are, *firstly*, bad hygienic surroundings,—*i.e.*, bad ventilation; bad drainage; absence of light; accumulations of filth within or contiguous to the byres; damp; low roofs; wooden and thatched erections; lofts; accumulations of cobwebs; site of byres, —the higher the elevation the healthier they are; overcrowding.

These circumstances not only favour the spread of the disease, but they determine, to a great extent, the length of its residence on a farm or in a district. Frequently have I seen the disease linger for months in buildings such as I have described,—built in low situations and contiguous to streams,—while in neighbouring farms, built on elevations with every attention paid to the hygienic surroundings, the disease has

never made its appearance, or, if it has, has only taken a feeble hold, and been easily eradicated. Wood, hay, straw, filth, cobwebs, collections of dust, are splendid receptacles for the virus of disease; and in many of the old-fashioned buildings the ceiling is so low as scarcely to allow of a man standing in an upright position; the floor of the loft, too, is often composed of bundles of sticks (brushwood) laid across joists, or rough planking with wide inter-spaces between the planks, through which the hay and straw projects. In many instances the lower stratum of hay or straw is not disturbed for years, and is covered with large cobwebs. It can easily be understood that disease germs (the vitality of which is great) would linger in such places for an indefinite period, and that if the hay, straw, or cobwebs became disturbed, the atmosphere of the byre would become charged with the germs, which would find favourable pabula in the systems of any new inhabitants of the byre; thus the disease is propagated and nursed in a district for years.

Secondly, Indiscriminate congregating together of cattle—as in fairs, markets, auctions, agricultural shows, leys, &c., and overstocking—is a very frequent and certain means of widely disseminating the disease.

Thirdly, Travelling by rail, canal, or sea, or in cattle-floats by road; these conveyances not having been properly disinfected.

Fourthly, Lowered vitality of the circulatory and nervous systems by debility,—whether arising from previous illness; innutritive or impure food, or impure water; excessive medication, excessive bleeding, or excessive drain in milking; by exhaustion from long travelling, or from the throes of parturition, and in the case of sea-travelling, the want (in bad weather) of a sufficient supply of pure air; starvation, exposure to inclement weather, or alternations of temperature and change of climate (hence foreign animals contract zymotic diseases more readily than do home-bred animals, until they become acclimatised),—may be all included under this head.

Fifthly, The distribution broadcast amongst healthy animals of substances or beings (*fomites*)—capable of acting as carriers or bearers of disease germs—which have been in contact with diseased animals.

FOMITES may be divided into, 1st, Living, or active; 2nd, Dead, or passive.

In the first group we have man, carrying disease germs about with him in the textures of his clothes, or on his boots, or skin of his hands; and animals or birds with fur, wool, or hair coats forming also splendid carriers. The feet of animals or birds, the beaks of birds, and the probosces of flies are certain disseminators of virus.

In the second group, all substances, which from their porous structure are capable of absorbing germs, such as all kinds of wool, hair, cloth, flax, sawdust, shavings, hay, straw, chaff, meals, dung, tan, hides, skins, hoofs, offal, flesh, &c., may be included.

VAGARIES IN THE SPREAD OF P. P. ZY.

These are very numerous, so much so, in fact, as to have led many observers to assert that the disease is neither infectious nor contagious.

But these very vagaries tend rather to prove than to disprove its infectious nature, for it is a fundamental principle of the spread of zymotic diseases that vagaries in their propagation arise; and this is due to the well-known laws which govern all of them. These are, *firstly*, That the poison shall be of sufficient potency and vitality to reproduce itself when brought into contact with favourable pabula.

Secondly, That the system of an animal shall be capable of receiving it.

Thirdly, That once having received the poison, it shall be capable of providing it with a suitable pabulum and a sufficient amount of nourishment to enable it to grow and multiply.

Fourthly, That the means of conveying the poison to healthy animals shall be forthcoming.

1*st*, then. It very often happens that the poison is of such a mild character, or has been so deprived of its virulent properties—by dilution, or from the system of its host not furnishing it with suitable nourishment—as to do away with its subsequent power of reproduction; dilution takes place also in travelling, by admixture with atmospheric air. Its potency is also reduced by exposure to disinfectants, frost, and other forces inimical to its wellbeing.

2*nd*. The systems of nearly all animals are quite proof against certain poisons; this is notably the case with the disease under consideration, as it can scarcely be propagated to any other animal than the ox, no more than Variola Ovina can be produced in any other animal than the sheep.

3*rd*. Although an animal may have received a poison into its system, and though it may have found the particular pabulum in which it is usually reproduced—as the liver, lungs, intestines, &c.—circumstances may have been previously in operation which shall have led to the entire removal from the system, or at least its complete alteration, of the material upon which the particular poison usually feeds and multiplies; thus, medicines—catalytic or purgative—setons, rowels, vaccination, inoculation, or the pre-existence of another zymotic disease may have so altered the blood and the secretions of the various organs as to render them incapable of supplying food for the nourishment and development of germs.

4*th*. The means of conveying poisons are often absent, simply by virtue of the adoption of a system of perfect isolation; and although the germs may be carried by the atmosphere, the latter may be so charged with ozone, or be so attenuated (as in high elevations), as to destroy their vitality, or to fail in carrying them.

Referring more particularly to P. p. zy. itself, we find that many cattle, although

placed under circumstances never so favourable for its reception, withstand the effects of the poison altogether; others suffer but very slightly from it; while in a byre it frequently develops itself first in the cow which is farthest removed from the one which introduced the disease; not only this, cows in byres several yards distant from the one into which the disease has been introduced will frequently become the victims of the disease, and those cohabiting with the affected animal escape.

Again, a lot of cattle may be purchased, and subdivided into several smaller lots, and sent to different parts of a district or farm; in due time the disease may break out in one or more lots and the remainder never show a symptom of it.

A relation of one well-observed outbreak will sufficiently illustrate the remarkable vagaries sometimes exhibited in the spread of the disease:—

P—— W—— had six cows in his possession for a period of six months; no fresh animals were introduced during that time, nevertheless the disease made its appearance at the end of the six months. Whence came it? Necessarily, by spontaneous generation, or from one of the following sources—viz., through the medium of fomites, from retention of virus in the cow-shed, or from the retention of the virus in a latent form in the system of one of the animals.

A case of Zy. p. p. occurred in this byre on the 3rd April, 1876, about which date other premises were rented, a new shippon erected, and, immediately adjoining, a double-stalled shippon or turnip-house. The outer partition and upper part of the front wall of the latter consisted of boards taken from the old byre. Two cows (one of them twelve months in), the remnant of the old stock, were removed to the new shippon, and, on the 25th April, two others were purchased at Hawick, one of which was placed in the byre above referred to as being composed partly of boards from the old one, and on the 14th August (four months after purchase) was attacked by Zy. p. p.; the other was stalled between the old cows, and did not contract the disease. I saw one of the two old animals above referred to (a Dutch cow), eight or nine months after the date mentioned; she was perfectly healthy, and although stalled in the midst of the disease, proved refractory to the virus.

A very important question in connection with the spread of this disease is the possibility or otherwise of an animal exhaling the virus during convalescence, or for any period after apparent recovery. I am myself convinced that the disease is propagated not only during convalescence but for a tolerable length of time afterwards, especially if the diseased portion of lung communicates with a bronchial tube, and several instances have come under my observation in which animals said to have recovered from the disease were the means of introducing the infection into healthy byres. I have also known instances in which old stagers have been kept amongst stocks of cows for considerable periods, the disease all the time continuing its ravages, and after the slaughter of the affected animal or animals the malady has ceased.

In a paper which I read on the subject several years ago at one of the meetings of

the Scottish Metropolitan Veterinary Medical Association, I brought the question of propagation by convalescents prominently forward, and one of the main arguments I then used in favour of the view expressed was the fact, that in the case of several convalescents which I had had the opportunity of watching, I had found a permanent temperature of 103° F., and that these animals were very susceptible to the action of adverse influences. Since that time I have seen several instances in which animals having recovered from the disease, although never re-exposed to the contagion in any way, have developed secondary Zy. Pleuro-Pneumonia, either in the healthy lung or the healthy portion of the diseased lung, after the lapse of a considerable period from the primary attack.

I find, too, other evidence from two independent sources which strongly favours the view that the disease is propagated by convalescents.

This evidence is to be found—*a*, In an article in the "*Veterinarian*," by Professor Ferrein, of Berlin; *b*, In the Annual Report of the Veterinary Department of the Privy Council Office for 1876.

Another very important question about which there would seem to be some diversity of opinion is the possibility of this disease being coexistent in the system of an animal with another zymotic affection, such as Cattle Plague or Foot-and-Mouth Disease. In my mind there exists no dubiety on this point, as I have seen Pleuro-Pneumonia and Foot-and-Mouth Disease in one and the same animal; as a rule, however, the symptoms of one are preponderant.

SYMPTOMS AND COURSE.

The character of the symptoms and the rapidity and violence, or otherwise, of the course of Zy. p. p. depend materially upon the amount of poison received into the system, its virulence, the constitution of the patient, and the attention which is paid to the animal in a medico-hygienic sense.

For convenience of description it is preferable to divide the course of the disease into stages.

THE PREMONITORY SYMPTOMS are in every respect identical with those of other specific febrile affections, *and they are dependent upon the incubative action which is going on in the system.* They are—tendency to isolation; irregularity of appetite and probably of bowels; slight listlessness; erection of hair, especially if the animal is exposed to cold, or cold and wet; unequal external temperature; slight shivering, if carefully observed; and slight diminution in quantity of milk in milch cows.

The most suspicious and reliable symptom is increase of the internal temperature; in almost every instance it is elevated from 1° to 2° F., and if the disease

is already existent in a herd it forms the best guide for isolation and careful watching.*

First stage.—The symptoms of this stage will be violent or otherwise as invasion is rapid or insidious. In very many cases invasion is so stealthy or so little marked as to prevent the true nature of the disease being recognised until it has advanced even to the second stage. In other cases the symptoms of the first stage are well marked.

They are an aggravation of the premonitory—the erection of hair, which is often dry and hard, the decrease in quantity of milk, the irregularity of appetite, the elevation of temperature, and the tendency to isolation are more pronounced. If the animal is at grass, it will be found in the early morning (or if a cold wind prevails, or there is a cold shower) huddled up under a hedge, wall, tree, building, or stack, or in a ditch or hollow quite apart from its fellows; the back will, at the same time, be arched.

The secretion of dew on the muzzle may or may not be suppressed; frequently it is secreted—though in diminished quantity—throughout the whole course of the disease. Rumination, though usually arrested, may be carried on until the disease is well advanced; so in the secretion of milk, usually diminished in quantity, it may continue to be secreted until convalescence is accomplished.

The animal ceases to lick the skin, but even this natural action may be persisted in until the second stage. In some instances pressure along the dorsal region causes the animal to shrink; but this is not peculiar to the disease,—is, in fact, often absent, and is an accompaniment of common cold, rheumatism, pleurisy, &c.

A cough may or may not be present—as a rule it is, but is overlooked; if not heard during the examination of the patient, means should be taken to induce it by bustling the animal about for a few minutes, by compressing the larynx, or by turning it from a warm into a cold atmosphere. It is more likely to be evinced if the air is cold or foggy, and is always pronounced, frequent, and irritable if the bronchial mucous membrane (especially of the anterior lobes) is the primary seat of the morbid lesions. The cough is characteristic,—usually single, short, and sharp, with the mouth open, the tongue protruding, and movement of the body suppressed as much as possible in order to prevent the production of pain in the act.

Grunting is not so invariable an accompaniment of this stage as of the second; if it is present, it is performed synchronously with the act of expiration at irregular intervals, and is most certainly induced by giving the animal a sharp poke with the knuckles on the affected side, by the application of pressure over the intercostal spaces or by causing it to turn sharply round. Striking the ribs, also, causes the animal to incline the head, with an oscillatory movement, towards the side which is struck.

* The Veterinary Surgeon to the Privy Council (Professor Brown) recommends in this, as in all other zymotic diseases, that if suspicion is aroused, and the temperature is elevated, quarantine should be insisted on until the suspicious symptoms subside.

The grunt will be of a short painful character if the pleura is affected; oppressive if the lung-structure only is implicated.

The pulse is tolerably strong, and, on an average, varies from 60 to 75 per minute, though in some cases—especially where there is much bronchial irritation—it may rise to 90 and become very weak.

The respiration is usually somewhat hurried—more so in some cases than others—but is seldom laboured in this stage. There may be rigors.

The temperature rises to 104-6°, and in severe cases to 107°. The elevation is easily detected—though not accurately measured—in the absence of a thermometer by placing the forefinger under the tongue. The external temperature is more or less variable. The animal may or may not lie down. The bowels are usually somewhat torpid, and the secretion of urine diminished; and if the animal has been exposed there may be an increased flow of mucus from the nose and eyes. The mucous membranes are not much injected, and the eye, as a rule, is tolerably bright.

In having recourse to auscultation, for the purpose of detecting abnormal sounds in the lungs, great care must be exercised to examine every accessible portion of the chest as the disease is often at this stage very circumscribed, and a careless examiner may not detect what to others would be of great diagnostic assistance; the fact, too, that the liver impinges on the diaphragm on the right, and the rumen on the left side, and thus diminishes the posterior area of auscultation and percussion, must be taken into consideration. This area will be especially diminished if the stomachs are very full and the animal pregnant.

The abnormal signs which may be detected on auscultation are—(*a*) Sibilus (wheezing), from diminution of the calibre of the small bronchial tubes by pressure of the effused serum external to them, or from tumefaction of the membrane itself; (*b*) Crepitation, from the effusion of serum into the bronchial tubes; (*c*) Friction sounds, produced by the rubbing together of the partially dry pleuræ; and (*d*) Patches of dulness, from effusion into the lung-structure and the air-cells. Increased murmur (puerile respiration) may be detected in the sound portions of the lung.

Percussion—unless the lung is consolidated by effusion—assists us but very slightly in this stage.

Second stage.—If the fever has run high in the first stage, it will be somewhat subdued in the second, and *vice versa*. The temperature may show slight declination or may vary; the pulse much as in the first, though it may become very frequent, up to 90 or 100. The respiration may be laboured; it is always distinctly accelerated and the grunt and cough are more constantly present. The animal shows great disinclination to move, and, as a rule, when it does so, evinces stiffness, though I have seen young undomesticated animals in such a state of excitement as to cause them to rush over anything or everybody which came in their way. This is misleading to a young practitioner who has only seen the disease in quiet dairy cows. Recubation may be performed

regularly, or the animal may stand obstinately in one position with the nose protruded.

The natural functions—appetite, rumination, milking—are, except in mild cases, more or less in abeyance; the quantity of urine is diminished.

In those cases in which the bronchial membrane is most involved, tubular casts, more or less large, of lymph may be expelled by coughing and expectoration. These masses of lymph may be colourless, or of a pale-straw colour tinged with blood (from rupture of small vessels), or mixed with mucus and air-globules.

Auscultation and percussion reveal important changes in the affected lung. There may be complete consolidation of the whole or part of a lobe, with blocking up of the bronchial tubes, as indicated by the absence of auscultatory murmurs: or dulness on percussion, over a larger or smaller area. On the contrary, there may be, on auscultation, bronchial respiration and even a modification of bronchial voice (bronchophony); both being due to the larger bronchial tubes remaining patent, while the lung-structure around them is solidified. The bronchial voice only applies to the grunt or cough the sound of which is conveyed to the ear through the consolidated lung and the ribs.

If the consolidation is confined to the central part of a lobe, the posterior remaining comparatively free so as to allow the air to permeate through it, a loud (almost whistling) to-and-fro murmur is heard.

Absence of sound is due sometimes, not to pulmonary consolidation, but to the formation of pleural adhesions; while if there is effusion into the chest without consolidation or adhesion, there will be absence of murmur, and the water may be heard gurgling during the respiratory movements. The characteristic breathing of dropsy of the chest in the horse is seldom seen in the ox, and only in a modified form. All pleuritic sounds (except in fresh areas of disease) disappear when consolidation of the lung, adhesion of the pleuræ, or effusion into the chest takes place. If the left lung is consolidated, the sounds of the heart are often conveyed to the upper part of the right chest, at which point they are very audible; as also around the cardiac area.

Puerile respiration in the sound lung becomes more pronounced as the respiratory functions of the affected lung become abolished.

In the third stage, there is total suspension of all the natural functions; the pulse is frequent, weak, and indistinct; respiration, short and quick (abdominal), and may be accompanied by a flapping valvular sound in the region of the upper flank, synchronous with respiration—produced by sudden tension of the diaphragm; there may be also twitching of the muscles of the flank.

The eye is dull, but may be bright and retracted in the orbit; the extremities cold; the muzzle dry; emaciation rapid; elbows abducted; ribs on affected side bulged (*gibbous*). There may be knuckling over of the hind fetlocks, and dropsical swellings of the subcutaneous cellular tissue of the space between the jaws and of the sternum may take place—when between the jaws it is termed *wattles.* Effusion is most extensive when the

anterior lobes are affected, and the pericardium involved. Cough and grunt are increased in frequency; the former being weaker, the latter more painful; the nose is protruded to the fullest extent so as to facilitate respiration; muco-purulent discharge from the eyes and nose is in some cases present; diarrhœa (the discharges being very fœtid) sets in, as also does tympany (hoven)—the latter being most aggravated if brewers' draff or potatoes have been the staple articles of food; pain on pressure over affected side is diminished owing to the mortification of the lung, and this diminution may mislead the tyro; there is constant gnashing of teeth; the breathing on the approach of death becomes oral; there is quivering of the facial muscles, and dribbling of saliva and, if the animal is recumbent, it endeavours to preserve its equilibrium on the sternum, the head being protruded to its fullest extent, in order to facilitate respiration. The skin and hair become very dry and harsh; the former, especially in parts devoid of hair—as the vulva, udder, perineum, root of tail, ears, &c.—contracting a yellow tinge, and frequently covered with abundant branlike scales from desquamation of the epidermis.

On auscultation, no sounds except bronchial are heard in the affected lung; and on percussion, there is an entire absence of resonance. Puerile respiration in the sound lung is more marked, while there may be crackling, as the result of emphysema, in portions of the lung, with dulness at the lower part, the latter being the result of collapse from blocking up of the bronchial tubes with serum, lymph casts, or coagula of blood. Abortion in pregnant animals frequently takes place at this stage, and, on the whole, is favourable to recovery.

If the pericardium is diseased, the pulse becomes irregular and the respiration gasping, the latter is also marked when the thoracic effusion is extensive; in these cases the animal usually dies suddenly: otherwise, death is produced by imperfect oxidation of the blood (*asphyxia*), by suffocation as the result of regurgitation of serum from the tubes of the diseased into those of the healthy lung, the serum becoming whipped into foam by the passage to and fro of the air in respiration. Lymph casts may also block up the tubes of the healthy lung, and lead to the same result.

On the approach of convalescence relapse may take place from exposure or from improper feeding, and very frequently, also, impaction of the œsophagus—usually the thoracic portion. This impaction may be produced in the act of swallowing the food, or in regurgitation of the cud; in either case it is due to weakening of the œsophageal walls, to paralysis of the pneumogastric nerves, or to inability on the part of the animal to bring pressure to bear on the œsophagus, either by tension of the diaphragm or inflation of the lung.

Indigestion, as evidenced by tympany, colicy pains, and irregularity of the bowels, or enteritis, may be accompaniments of convalescence, hence the necessity of being extremely careful as to the quality and quantity of food until the digestive function is completely restored.

If an animal rallies—I will not say recovers, as the injured lung is *never, except in very slight cases,* restored to its original condition—it should be prepared for the butcher as quickly as possible, as after apparently thriving well for some time, it may die suddenly or commence to emaciate, from causes hereafter to be considered. I have known many so-called cases of recovery, in which an animal has been prepared for the butcher, and has fallen dead on the way to the slaughter-house. The poison, too, is not always entirely eliminated from the blood, and on the animal being exposed to any fresh exciting cause (as debility from over-milking or parturition; extremes of heat or cold, over-exertion or impure atmosphere), its presence is manifested by a secondary attack, either in the healthy portion of the affected lung or in the sound lobes.

The symptoms accompanying the subsequent stages, or results, of Zy. p. p., will depend upon the changes which take place in the involved lung structures, and will be incidentally alluded to when referring to these changes.

DIAGNOSIS.

The diagnosis of this disease is a most important matter, as failure to detect it early may lead to very serious consequences.

In the early stages it is often a most difficult thing to give a decided opinion, and, indeed, the practitioner should always be very guarded in expressing himself until the symptoms are well developed, more particularly if the disease is not already existent in the neighbourhood. I have already (*ante*, p. 3) contrasted the disease with Sthenic p. p. In addition to this affection it is liable to be confounded with—(1.) Ordinary cold. (2.) Pleurisy. (3.) Indigestion, with or without Congestion of the Lungs. (4.) Hoose. (5.) Tubercular Phthisis.

1. Ordinary cold is distinguished from it by the comparative mildness of the attack, by its evanescent character, by the accompanying catarrhal symptoms, by the lesser degree of interference with the normal functions, and by the temperature seldom exceeding 103°.

2. In Pleurisy the cough and grunt are more painful and shorter; the pain on pressure over the ribs is more marked; the mucous membranes intensely injected; the pulse hard, wiry, and very frequent; the respiration abdominal; and great pain is evidenced if the animal is made to turn suddenly. Temperature about 104-5°.

3. Indigestion is most likely to be confounded with Zy. p. p. when it is accompanied by pulmonary congestion, and when the third stomach is the seat of the disorder. The grunt of indigestion is more indicative of inconvenience than pain, and cough, if present, is louder and bolder.

4. Differential characters and symptoms of Hoose:—

1*st.* Hoose is essentially an infantile disease, and is seldom seen in animals over a year old.

2*nd.* It frequently attacks lambs and calves simultaneously, and spreads regularly and at certain times of the year particularly.

3*rd.* The cough is an irritable bronchitic or husky one, and is frequently accompanied by expectoration of mucus, intermixed with which there will probably be strongyles, their embryo or eggs; the latter being detected microscopically.

4*th.* There are seldom any pleuritic sounds on auscultation, and no pain on palpation.

5*th.* The grunt of Zy. p. p. is absent.

6*th.* If diarrhœa is present, the discharges are natural in colour and odour.

7*th.* Febrile symptoms are less marked.

5. In the acute form it may be confounded with Acute Pulmonary Tuberculosis; but in this disease the pulse is more frequent and irritable, and more in relation with the temperature and respiration; the diagnostic grunt of Zy p. p. is absent, and on auscultation there may be no consolidation, or, if present, it is patchy; previous history, too, is always a good guide.

During the course of the secondary changes in Zy p. p. it is easily mistaken for *Chronic Pulmonary Tuberculosis.* As a rule, the temperature is higher in the former than the latter, and on auscultation the partial character of the consolidation in Tubercle is detected. Here, also, history and collateral evidence must be made use of if possible.

PATHOLOGICAL ANATOMY.

In describing the *post-mortem* conditions of this disease, I do not intend to refer at any length to the miscroscopical appearances of the diseased structures, but to point out those characteristics which are cognisable to the naked eye. Before considering this in detail it will be as well to glance at the specific *post-mortem* differences existing between Zy. p. p. and other diseases with which it may be confounded. These diseases are—*a.* Pleuro-Pneumonia; *b.* Pulmonary consolidation or hepatisation following sub-acute inflammation (Broncho-Pneumonia), as the result of irritation produced by foreign bodies or parasites. The subsequent degenerative changes in a Zy. p. p. lung may be confounded with Tubercle and degenerated hydatid cysts, also with the degenerative changes of Broncho-Pneumonia, and fibroid alterations of interstitial inflammation (see Fig. 32, Plate VIII., and read remarks in connection with Tubercle); these I shall contrast in due course. Intercurrent disease of the lungs or other organs, either chronic or acute, may complicate P. p. zy., and render diagnosis more difficult.

A Zy. p. p. lung is distinguished from that of P. p. by greater weight (even in the early stages), and friability; by being much paler in colour externally, and variegated

—*mottled*—internally; by different progressive stages of disease going on in the same lung: thus a section may show healthy lung tissue, interlobular effusion, red, black, and grey hepatisation, and brick-like hepatisation (sphacelus); by being often diseased in patches, and the involved portions being always well defined and circumscribed; by the serum showing a greater tendency to gravitate to the lower and healthy portions of the lung, rendering them translucent, and of a buff or greyish-brown colour, and a solidified character—not unlike veal in appearance—without any distinct interlobular or sub-pleural effusion, and without absolute destruction of the parenchyma: by the extensive interlobular effusion, exudation, and hyperplasy; by the transudation of large quantities of serum when the lung is incised or submitted to pressure—this serum sometimes coagulating on exposure to the atmosphere; by the ulcerations and ecchymoses of the bronchial mucous membrane; and by the condition of the thoracic lymphatic vessels and glands.

In acute P. p. the lung, both externally and internally, as also the lining membrane of the blood-vessels and the bronchial mucous membrane, is uniformly red or black in colour, and devoid of the characteristics above mentioned; it may crepitate slightly on pressure. A mortified P. p. lung is intensely black, sometimes green, often of a fœtid odour, and the bronchial mucous membrane of a black or greenish hue; a mortified Zy. p. p. lung is of a brick-red uniform colour, and very friable; and, finally, while degenerative processes, when they commence, proceed more rapidly in P. p., organative changes are more stable.

b. Consolidated lung, resulting from Entozoic, or Broncho-Pneumonia, presents at first somewhat the appearance of fœtal lung; the pleura is not often involved; the bronchial mucous membrane is intensely red (Fig. 8A, *d. d.*, Plate III.), and is not often ulcerated; a section is parti-coloured—grey and red (Fig. 8A, Plate III.), the grey portions being friable and cheesy: the lung is not so heavy, does not exude much serum when pressed, does not show the same tendency to degeneration, and does not degenerate *en masse*, but in small circumscribed patches, leading to the formation of lobular abscesses and vomicæ. In Entozoic Pneumonia filaria are found, either fully matured in the bronchial tubes, or as embryos, with eggs (distinguished microscopically) in the consolidated lobules. If the consolidation results from the accidental introduction of minute foreign bodies into the bronchial tubes, the diseased lung-structure is firm; there is no interlobular effusion, and the secondary changes are most usually—(1.) Suppuration, leading to the formation of circumscribed abscesses, large or small, numerous or few, according to the size, character and number of the irritating particles: in some cases abscesses are replaced by encystment and liquefaction of the exudate, giving on transverse section a multilocular character; frequently, too, the foreign matter—as small seeds, portions of grain, or other vegetable product—will be easily detected in the bronchia, surrounded by an exudate, or lying in the centre of the cyst or abscess. (2.) Interstitial inflammation, with its usual results

—Hypertrophy and Cirrhosis. Consolidation from foreign bodies is usually located in the anterior lobes, or at the inferior border of the large lobes.

In order to render my remarks on the *post-mortem* appearances intelligible, it will be necessary to consider them in definite order, under several heads.

The Carcase.—I will preface the remarks I have to make under this head, by observing that when called upon to examine the carcase of an animal suspected of having been the subject of Zy. p. p., the veterinary surgeon should pay particular attention to the condition of the *pleura costalis,* with the object of discovering whether any diseased or thickened membrane has been removed by the butcher for the purpose of deception. It must not be forgotten, however, that the pleura is often removed with the simple view of getting rid of the unsightly appearance produced by the adhesion of a film of coagulated blood upon its surface, as the result of extravasation into the thorax from the knife having penetrated too deeply ("over-sticking"). In this case, all doubt is set at rest by the firm, healthy condition and colour of the systemic muscles. It is also stripped in Tuberculosis and Sthenic Pleurisy.

When stripping off the pleura has been resorted to, the smooth glistening appearance of the internal surface of the thoracic walls is replaced by a roughened, ragged condition, as the result of tearing through of the sub-pleural connective tissue and the costal periosteum. In many instances the butcher will attempt to remedy this by applying a little cosmetic in the shape of warm suet; the use of this material can always be detected by the minute portions of inorganised fat adhering to the parts—these presenting a very different appearance to the small masses of fat which are naturally deposited under the pleura, and which are smooth and firm, with the glistening membrane outside. Some butchers use a solution of gum, white of egg, or the effused albuminous serum from the diseased lungs, to varnish over the stripped portions,—the torn fibres of the pleura, although plastered down, can be distinctly seen under the layer of varnish.

Elevation of the fleshy portion of the diaphragm (which is usually left on the carcase) on the suspected side will frequently expose to view portions of diseased pleura which the butcher has not thought necessary to remove, under the impression, that, being concealed, it will escape detection.

I am informed by Mr. Rutherford, late Inspector of the Edinburgh *Abattoirs,* that the carcases of animals in an advanced stage of Zy. p. p. stiffen rapidly and very rigidly, more particularly if the animal has been much excited immediately before death; and further, that the flesh requires a long time to cook by boiling, and is hard when roasted. The latter facts I have frequently verified by experiment; and I have, in addition, noticed that a section of a roasted steak is parti-coloured, and that the fat does not inflame, or only very slightly so, when a light is applied to it, but sputters and chars.

In the early stages—*i.e.*, when the disease has existed two or three days—the muscles are not materially altered in character unless both lungs have been much involved or the whole of one; in which case, as also in nearly all cases where the disease has been extensive and at all prolonged, the exterior of the carcase, owing to the fat being of a deep-yellow (icteric) and the muscle of a dark-red colour, will possess a peculiar *mahogany tint* and the flesh will be dark in colour when cut across—sometimes even tarry; it will also present an iridescent hue (Fig. 8, Plate X.), and be devoid of the firm feel of healthy beef. If the disease has been extensive, these characters will be very marked about the fourth or fifth day; and in all cases the changes are due to imperfect oxidation of the blood, and, consequently, accumulation within it of carbonaceous compounds. On exposure to the atmosphere (oxygen) for a short period, the dark colour gives way to a brighter red. When hydrothorax has existed, the muscular portions of the diaphragm and the muscles of the ribs have a macerated or parboiled appearance; they are soft, thickened and easily lacerated, and a transverse section presents a peculiar yellowish-brown tint, which in most cases is replaced by a bright-red (*magenta*) hue and glazing, the latter being most observable after exposure for a few minutes and by comparing it with the corresponding parts on the healthy side of the chest. In many instances, this glazed appearance extends more or less to the whole carcase—the fore-quarters especially—and is due to *albuminous effusion.* This condition may, on a cursory examination, escape detection, and is always most certainly discovered by stripping off a portion of the peritoneum or pleura. The muscle thus exposed has at first a moist appearance, and an adhesive feel; but the coagulation of the albumen soon gives rise to the *varnish-like albuminous glaze.* It must not be forgotten that the muscles of the neck, in all carcases which have been suspended by the hind-legs for a few hours, owing to the gravitation exudation and coagulation of the serum of the blood, have an albuminous glaze; but in these cases the muscles of the hind-quarters are healthy, and not glazed.

In the latest stages the albuminous may be replaced by *serous* effusion (the albumen becoming absorbed and used-up in the meantime), and the tissues have a white, watery, and macerated (dropsical) character. Both the conditions above described will be materially modified by the kind of food upon which the animal has been fed, and the healthy or unhealthy state of the system at the advent of the attack.

Considerable alteration (physically) will also be found in the muscular structures when an animal has recovered from the primary symptoms, and succumbed to the *sequelæ* of the affection. Usually the condition last described is present under these circumstances.

Albuminous effusion is only a concomitant, and not a special attribute of Zy. p. p. It is seen in other zymotic diseases,—as Rinderpest and Eczema Epizootica,—and is due to the primary disturbance in the sanguiferous constituents by the action of the virus. The albuminous serum is probably reabsorbed in order to compensate for the

loss of the nitrogenised constituents of the blood, these having been expended in the support of the disease-producing germs, and the amount of this reabsorption will be regulated by the intensity of the tissue changes induced. The serous effusion results simply from lowered density of the vital fluid.

In estimating the dates at which these various changes in the muscular system, and also those in the lungs, take place, considerable difficulty is experienced, and accuracy is well-nigh impossible, owing to the fact that many animals may have been labouring under the disease several days prior to that on which its symptoms were noticed; this difficulty is further exaggerated by the lack of truthfulness on the part of some of the owners of animals, who, for purposes of their own, will more frequently than otherwise falsify the date of the attack. The appearances described will be further altered or modified by the manner of death of the animal, and by the amount of blood drained from the system through the carotid artery; but it does not matter how much blood may have been removed, the *mahogany hue* in the one case, the *magenta hue* in the other, and the *macerated appearance* in the last stages, are always more or less perfectly defined.

A very important sanitary question in connection with the use of Zy. p. p. flesh and milk as articles of human food must not be overlooked. In which of the three conditions is the flesh most likely to be deleterious? Before any marked physical change in the muscular tissue occurs, I can conceive no objection to the consumption of such flesh; but I have very grave doubts whether an inspector is justified in allowing it to be used when any one of the three conditions mentioned exist. I would hesitate to pass any flesh as food which gave evidence (by its dark hue) of the existence during life of a high degree of fever, or of imperfect decarbonisation of the blood; and I would be even more decided with flesh of a magenta hue, and in which albuminous effusion existed, as in the stage when this change takes place the blood, and consequently the tissues and all effused materials, are charged with the virus of the disease: and, although, when serous effusion takes place the germs are dead and the flesh as a consequence innocuous, it is innutritious—its nutrient value being reversed in the two latter conditions. The milk, as already indicated, is usually more or less diminished in quantity, even in the earlier stages; and in the later, the secretion is, more often than otherwise, totally suppressed. In very mild attacks the secretion of milk may continue in tolerable abundance throughout, and, so far as physical and visual examination goes, no change of a deleterious nature can be detected in its quality,—in fact, I have known calves, suckled on diseased dams, thrive and do well with it; but when disease exists to any extent, or when secondary changes are going on in the lung, the quality of the milk must be materially reduced.

The blood in Zy. p. p., either after or before death, does not possess any distinctive characteristics, except such as result generally from interference with respiration and from the action of any virus—*i.e.*, in the earliest stage of the disease it is nearly normal

in character; subsequently it becomes darker in colour and thicker in consistence, but coagulates readily.

So soon as dropsy takes place, the blood becomes paler in colour, of less density, and, although it coagulates with tolerable rapidity, the coagulum is not firm.

In order to facilitate the description of the physical *post-mortem* changes observed in the parts which are involved in Zy. p. p., I shall arrange these structures in the following order:—1. Trachea; 2. Bronchia; 3. Connective Tissue (subdivided into —*a. interlobular; b. lobular; c. sub-pleural*); 4. Parenchyma; 5. Blood-vessels; 6. Lymphatic Glands and Vessels; 7. Pleuræ. The subsequent changes including also 8. Epi- and Peri- cardium.

First stage.—The mucous membrane of the trachea and bronchia may show no signs of disease; or it may be of a yellowish-brown colour, be covered with an abnormal quantity of mucus, and present here and there small irregular hyperæmic patches: and in some instances petechiæ, or diffuse ecchymoses (Fig. 5, *a, b*, Plate II.) Occasionally, also, in very acute cases, the bronchial tubes are more or less filled with serum or coagulated lymph, the latter being semi-solid or largely infiltrated with air-globules; it is sometimes tinged with blood, as a result of rupture of small capillary vessels from the violent expiratory efforts of coughing. Sub-mucous effusion, if present at this stage, is only so to a small extent.

The connective tissue is surcharged with lymph, constituting interlobular, lobular, and sub-pleural effusion, and giving rise—(*a*) To the formation of bands of a white or pale-straw colour (Fig. 4, *a*, Plate I.), varying in thickness from $\frac{1}{16}$th to $\frac{1}{8}$th of an inch, between the lobules. The areolæ of the tissue are easily discernible; and if an incision is made through the parts, or pressure applied, the lymph, if fluid, exudes and quickly coagulates into a thin jelly-like mass; sometimes, and especially if the animal is in good condition, and has been dead sufficiently long to allow of the lungs becoming cold, the lymph will be found coagulated in the meshes of the connective tissue. (*b*) To a glistening, somewhat solidified, and moist appearance of the parenchyma, with a slight increase in the depth of colour (Fig. 4, *b*, Plate I.), and in friability. (*c*) To elevation of the *pleura pulmonalis* in circumscribed or diffused patches; the membrane itself being of a white or a pale-straw colour, according to the tint of the effused serum.

The parenchyma is always a little darker in colour, is of greater specific gravity, more friable, has a slight granular fracture, and very frequently its section reveals myriads of minute stellar-shaped vascular spots (Fig. 2, Plate I.) (due to hyperæmia of the capillary plexuses surrounding the air-cells) with, occasionally, small circumscribed irregular patches of a red (Fig. 2, Plate I.) or brownish-red colour.

The blood-vessels are usually empty; they may contain *post-mortem* coagula of blood or coagula of lymph, but there is no visible change in their coats—the *tunica*

intima being of its natural colour, and glistening. There may be, however, capillary obliteration by plugging.

The lymphatic glands and vessels present no material change, the former may be swollen and slightly increased in vascularity ; the latter usually distended with serum.

The *pleura pulmonalis* may or may not be involved at this stage ; as a rule it is not, and if it is there is simply the effusion of a limited quantity of colourless serum on its surface, which may collect in the thorax ; or there may be slight exudation of lymph, very friable, and but moderately adhesive in character. In occasional cases, the pleura, throughout the course of the disease, shows no change, except opacity, from alteration of the lung-structure beneath it, interference with its nutrition, or pressure of the enlarged lung.

The general characteristics of a Zy. p. p. lung in the first stage, are : increase in weight, bulk, and friability ; diminution in resilient power—and consequently increased resistance to inflation, and decrease in crepitation on pressure ; with deepening of colour, and the presence, occasionally, of ecchymoses or hyperæmic patches in the bronchial mucous membrane (Fig. 5 *a. b.*, Plate II.), vascular stellar spots in the parenchyma and effusion of serum into the interlobular and other connective tissue, giving the lung (especially externally) an appearance very similar to that produced by Emphysema, as seen in Fig. 3, Plate I. [The differential characteristics of Emphysema and Zy. p. p. in its early stages are so marked that I imagine none but a novice could possibly confound them—except, perhaps, on a superficial ocular examination. I have, however, thought it best to introduce a sketch of a portion of emphysematous lung in order to show the great visual similarity existing between it and the early stage of Zy. p. p. In Emphysema the lung is of less specific gravity than normal ; in acute Emphysema the lobules may be—as in the sketch—of a more florid hue, while in chronic they are always pale : when an incision is made into the interlobular tissue it collapses, and is always dry, and through the pleura (Fig. 3, Plate I.) the cellulated character of the tissue is discernible.]

There is no absolute loss of textural integrity ; and the general condition is simply one of *serous* and a moderate degree of *cellular* (*leucocytic*) *infiltration*, with the presence of a few coloured corpuscles.

The fluid contained in the lung-structure contiguous to the diseased portion differs from that in the latter in being largely intermixed with air-globules.

Second stage.—The tracheal and bronchial mucous membrane may be still darker in colour. The petechiæ and hyperæmic patches, when present, have commenced to undergo degeneration ; the portions of mucous membrane covering them may have given way, and allowed of the escape of the extravasated blood into the interior of the tubes—discolouring their contents.

The smaller bronchioles are obliterated, either by the effusion of serum or exudations of lymph into their cavities; by the pressure produced on their exterior by peri-

bronchial effusion and exudation or by sub-mucous exudation. In those tubes into which there is effusion alone, the serum becomes converted into foam by the passage to and fro of the inspired and expired air. The air-cells are obliterated by an exudate of lymph.

The serum, which was thrown into the connective tissue in the first stage, has now to a great extent become absorbed; and more particularly is this the case on the approach of the third stage: the interlobular tissue may become somewhat contracted, and, as a consequence, the lines formed by it may be slightly narrower. In some cases, however, the exudation of lymph into the connective tissue takes place to such an extent at this stage, that the bands become thicker (they are always more solid) than in the first stage.

The parenchyma undergoes a material change. It is more solid, more friable, and the granular character of the fracture is more determined; its colour is either red or black, depending entirely upon the amount of bronchial obliteration and capillary destruction. If the black portions are exposed to the atmosphere, they rapidly become of a bright-red or magenta hue; the two conditions (though differing widely from true consolidation) are known, respectively, as *red and black hepatisation.* (See Fig. 1, Plate I.)

The destruction of the parenchyma at this stage has been attributed to pressure exerted by the effused serum in the interlobular connective tissue. Such is not the case, however; it is due to *parenchymal effusion*, to *capillary engorgement*, and *actual rupture of the capillaries*, and differs in no respect from the state of the lung-structure in P. h. The red colour (red hepatisation) is due to capillary engorgement and to the arrestation of the coloured blood corpuscles (which have escaped from the capillaries) in the meshes of the living structure; the black colour (black hepatisation) is due to total blocking up of the bronchial tubes, and if a section is exposed to the atmosphere it quickly becomes red. The effused serum of the part, if microscopically examined, will be found to be largely surcharged with coloured corpuscles.

The parenchyma may be easily detached from the interlobular tissue, leaving the latter intact, and causing it to present a honeycombed appearance.

The larger blood-vessels and vessels of supply are more or less obliterated, either by coagula of lymph or blood. If the vessels of supply are not obliterated, the greater will be the probability of the subsequent changes in the lung-structure assuming an organative character. The capillaries are absolutely broken up or occluded.

The increase in the size of the lymphatic glands is very marked at this stage; in addition, they become softened, and, as the result of acute granular and fatty degeneration, their glandular characteristics are entirely lost; and they have much the appearance and consistence of Devonshire cream. The vessels are enormously distended with yellow lymph, and can be readily traced (often the size of a quill) from one group of thoracic glands to another and even to the hepatic ganglia in some cases.

The pleura, if affected, is thickened, roughened and opaque; and separated from the subjacent tissue by extensive sub-pleural effusion and exudation.

The pleural effusion and exudation, when present, is considerably increased. The effusion will vary in quantity in every individual case; there may be only a pint of serum in the chest, or there may be many gallons.

The exuded lymph is more cohesive, coagulates firmly, and acts as a connecting bond between the costal and pulmonary pleuræ, and the pleura of contiguous lobes. The flakes are, as a rule, of a yellow colour, easily broken down, and when stripped from the pleura, leave its surface glistening and smooth.

The general characteristics of the second stage are—A mottled (*marbled*) appearance on section, increase in bulk, density, specific gravity and friability; loss (to a greater or less extent) of textural integrity, breaking up of the capillary vessels, and, as a consequence, parenchymal extravasation; obliteration of large vessels and small bronchioles, with destruction of the thoracic lymphatic glands.

If compressed, the lung exudes a large quantity of serum (often bloody); if thrown into water, it rapidly sinks; if boiled, it presents the appearance of boiled liver—the interlobular spaces, however, being still very distinct; if macerated in alcohol, it has also the characteristics of liver, with diminution of the interlobular spaces—the latter being due to the great attraction for water exerted by the alcohol.

If the destructive process is so great as to tend towards mortification, a segregating lymph-band is, even now, formed, the centre of which will reveal, on section, a distinct vascular line; and the serum contained in its meshes will be charged with leucocytes. In some cases the lymph-band becomes the means, not only of preserving the contiguous lung-structure from further injury, but also of arresting the destructive process in the diseased portions.

Third stage.—The larger bronchia and the bronchioles are entirely obliterated, either from external pressure, or from sub-mucous or intratubular exudation and organisation of the sub-mucous exudate, with consolidation of the intratubular. The mucous membrane is always thicker, softer and darker in colour, and its longitudinal ridges are exaggerated; while in the ecchymosed patches it is distinctly ulcerated (Fig. 5, *c*, Plate II.), the surfaces of the ulcerations being of a light-yellow colour, from collections of degenerated epithelium and lymph, on removal of which the bright-red, hyperæmic (Fig. 5, *d*, Plate II.), sub-mucous tissue is revealed.

The tracheal exudation is increased, and shows a tendency to become organised. Occasionally, however, it is mottled from extravasation, as the result of rupture of small capillaries, and from congestion and inflammation (Fig. 10, Plate X.) The calibre of the trachea (and of the involved bronchi) is materially diminished, and its shape altered, by the sub-mucous and peri-tracheal exudations (Fig. 10, Plate X.), and thus the difficulty in respiration is increased in this stage. A transverse section shows—1st,

mucous membrane; 2nd, sub-mucous exudate; 3rd, tracheal or bronchial ring; 4th, extratracheal exudate (Fig. 10, Plate X.)

In some cases the larger and smaller tubes are completely plugged by solid lymph coagula, which form perfect casts of their interior and, when lying in contact with the ulcerated membrane, present depressions and elevations corresponding to the ulcers, giving them also an ulcerated appearance.

The connective tissue and parenchymal alterations in this stage will depend upon the amount of injury sustained by the parts; and the condition of the parenchyma will materially influence the condition of the connective tissue.

If the injury has been severe, and the arteries of supply obliterated, with destruction of the arterioles and capillaries, all semblance of lung-structure is lost; the part, to the naked eye, has a somewhat homogeneous appearance, is of a *brick-red* colour (the colour being due to staining by the dissolved hæmatin of the blood), (Fig. 1, Plate I.; Fig. 8, Plate III.), possesses a peculiar acetous odour, which, after a time changes to one of a valerianic character; is extremely friable, and, on section, the only trace of organisation perceptible is in the bronchial tubes and large blood-vessels which resist the destructive process for a considerably longer period than the other portions of the lung-structure. *The tubes are filled with exudate, and the vessels with coagula of blood or partially organised lymph.*

The mortified portion is now distinctly isolated from the surrounding structures by a segregating lymph-band, hyperplasy of the adjacent interlobular tissue is marked, and there is increased density of the surrounding parenchyma, which becomes translucent and of a cinnamon or dull-red colour.

If the injury has not been sufficient to produce absolute destruction of the parenchyma and obliteration of the vessels of supply, changes similar to those seen in ordinary inflammation go on. The red cells become liquified or disintegrated, and absorbed; coagulable lymph is thrown out and becomes organised very rapidly; the connective tissue becomes denser and firmer, and the interlobular portions thicker—small blood-vessels being easily traced in it by the naked eye, and fibrillisation by the aid of the microscope. The ultimate changes are, as a rule, identical with those of Interstitial Pneumonia. The amount of serum is diminished, both in the connective tissue and the parenchyma; consequently, a section is less granular in appearance, and does not exude serum to the same extent as in the early stages; the involved lobules are tougher, and of a paler colour: sometimes grey, sometimes yellow—constituting, respectively, *grey* and *yellow hepatisation*, but may eventually become involved in the fibroid changes.

The arteries and veins, as already indicated, become obliterated, either as the result of external pressure, coagulation of blood or lymph and organisation of the coagulum in their interior, or thickening and ulceration of their coats by inflammatory processes.

The lymphatic glands, if mortification of the lung has taken place, will be found

utterly destroyed, sometimes of a creamy colour and consistency, at others resembling an olive-green pulp. If, however, the destructive process has been arrested at the termination of the third stage, the gland may show a tendency to reorganisation, and become firmer, denser, and more vascular. The *vessels* are still distended with lymph—the distension being greatest if there is extensive venous obstruction. The coats of the lymphatics will also give evidence of inflammatory changes, by becoming opaque and thickened.

The pleural exudations are considerably altered; the serum having become absorbed, the albumen and fibrin alone remaining, they present a more consolidated appearance, and are more contracted; frequently being reticulated in structure, from the formation of numerous small bands. Granulation commences in the inflamed pleuræ; and on stripping off the superjacent layers of lymph, a large vascular granulating surface (Fig 6, Plate II.) is exposed. The granulations projecting into the layers of lymph leave, on detachment, numerous alveoli on the under surface of the latter. Coincidently with the development of the granulations, tough fibres are formed, which hang (when the layers of lymph are removed) like small threads between the granular bodies (Fig. 6, Plate II.) The whole of the lymph may ultimately become absorbed, and the granulations from either pleura coalesce and unite them firmly together, or unition may take place by the organisation of the exudate.

The characteristics of the third stage are: absolute loss of integrity (death), with segregation of the injured lung; hyperplasy of contiguous interlobular tissue; and increased density of the surrounding parenchyma. If the destructive process is arrested, the conditions are: absorption of the red cells; consolidation of the parenchyma; hyperplasy of the interlobular connective tissue; grey or yellow hepatisation; and in rare cases, subsequent breaking up and removal of the injured lobules by the action of the adjacent interlobular tissue, as seen in Fig. 6, Plate X. In either case, the changes in the bronchi and trachea are: ulceration and softening of the mucous membrane, and consolidation of the submucous and extratubular exudate.

Accessory Post-mortem Lesions of Zy. p. p.

These are seen: 1st, in the adjacent lung-structure; 2nd, in the stomachs; 3rd, in the intestines; 4th, in the cardiac pleura.

1st. *The adjacent portions of lung* are usually more solid than natural; the lobules translucent, of a darker colour, and less resistant; ecchymoses may exist in the parenchyma, and effusion or exudation in the bronchial tubes—in which, also, in some instances, a marked increase in the quantity of mucus is observable. The bronchial mucous membrane may or may not be altered in colour; if it is, it will be of a *coppery hue;* and occasionally ecchymoses may exist. The interlobular tissue is hypertrophied, and not infrequently undergoes granular degeneration—usually in circumscribed oval

patches—the degenerated parts assuming a greenish-yellow hue, and being easily detached, leaving loculi. (See Fig. 4, Plate X.)

Emphysema may be a concomitant; usually, however, it is a subsequent lesion.

The serum which is often found in the bronchial tubes is usually the result of overflow from those of the affected parts.

2nd. *The stomachs.* As a rule, the first, second, and third divisions are healthy. As in all chronic or prolonged diseases, the contents of the second are comparatively dry, and frequently rolled up into large masses; while the material in the third is dry and hard, the epithelium readily peeling off with the cakes of ingesta, and the latter showing the impress of the papillæ on their surfaces.

Occasionally large irregular ulcers of the mucous coat are found in the second, third, and fourth compartments: they are always preceded by circumscribed hyperæmic circles or extravasations; but are not peculiar to Zy. p.p., being simply concomitants of it, and of many other diseases in which an altered condition of the blood is a prominent characteristic. Moreover, similar conditions of the coats of the stomach are observable whenever prolonged contact of vegetable matter (containing, in all probability, acrid herbs) takes place.

Zy. p. p. may likewise be accompanied, or preceded, by Epizootic Eczema; in which disease gastric and intestinal lesions are frequent.

3rd. *In the intestines* hyperæmic patches (especially in the glands when they have taken on vicarious action) and extravasations, followed by ulcerations, are by no means uncommon.

4th. *Cardiac Pleuritis* is a concomitant of the disease; though, like Emphysema and Pericarditis, it is more frequently a subsequent lesion.

The secondary changes in a Pleuro-Pneumonia Zymotica lung are:—

(*a*) Organative. (*b*) Degenerative.

In the parenchyma and connective tissue, *organative changes* only go on when the destructive process is arrested in the second stage, and usually occur in those parts which have undergone yellow hepatisation, and in which the vessels of supply are partially or wholly pervious.

It must not be assumed that by the use of the term *organative* I mean to imply that the lung-structure is restored to its original condition—or anything approaching to it: a lung once injured by Zymotic Pleuro-Pneumonia effusions and exudations is never, either functionally or organically, restored.

Subsequently to the yellow hepatisation, the exudations gradually become lowly organised, contracted, tough, and indurated; constituting, in fact, *indurated* or *fibroid* lung; this indurated tissue may ultimately become cartilaginous, may soften *en masse*, or in numerous circumscribed spots, and form one large, or many minute, abscesses.

Another method by which organisation of the injured lung may go on is by the adhesion of the pulmonary pleura immediately covering the damaged structures to the contiguous costal or diaphragmatic pleura,—vessels from which shoot into the pulmonary pleura and thence into the adjacent lung-structure, thus supplying it with nourishment.

The ulcerations of the bronchial and tracheal mucous membranes heal, and form permanent cicatrices, in the contraction of which we have another factor in the obliteration of the bronchioles.

The tracheal and bronchial exudations always undergo organisation, and, although contracting in size, permanently diminish the calibre of the tubes, and in some cases absolutely obliterate them.

The changes which result from continued interstitial inflammation, and from vascular and bronchial involvement, are well shown in Fig. 9, Plate X.

The pleural exudations frequently become organised, forming permanent adhesions.

Dead (necrosed) lung may be confounded with, firstly, *degenerated tubercle;* secondly, *hydatid or other parasitic cysts.*

It is most likely to be confounded with tubercle when deposits of this substance exist in other organs or structures; but on careful examination the resemblance, anatomically, to the brick-like hepatisation—which persists in the most minute portion of the lung (though it is decolorised) even for some months—can be distinctly traced.

The necrosed lung is usually in very large masses, though these may be connected with smaller adjacent masses, or even with cavities: it never undergoes calcareous, and not often caseous, degeneration, though the neoplasms formed by the interlobular exudations may; moreover, tubercle is always surrounded by healthy connective tissue, and unless degenerated, is continuous with it, not separated from the surrounding tissue like P. p. lung; neither does the degenerative process in Zy. p. p. lung—unless it is interpenetrated by active granulations—ever commence in the centre, and (unless coincidentally) small masses of tubercle are not found in other portions of the lungs or in distant organs.

It is a matter of great moment, medico-legally, that a degenerated Zymotic Pleuro-Pneumonia lung should be carefully distinguished from degenerated tubercle; and not only this, but that the practitioner should be able to give a positive opinion—in the event of the two conditions coexisting—as to which of the two had prior existence, or to which death might reasonably be attributed.

Degenerated multilocular hydatid cysts are of a deep-yellow colour, show the superincumbent layers of the hydatid structure, usually possess a definite form, undergo calcareous degeneration, and may coexist in other organs (see Fig. 3, Plate X.)

Nearly all other parasitic cysts and exudations occur in small nodules, and more frequently than otherwise undergo calcareous degeneration.

Changes in the Dead (Necrosed) Lung.

Coincidentally with (or in some cases earlier) the actual accomplishment of the brick-like hepatisation, lymph is thrown out from the vessels of the surrounding structures, forming a white line of demarcation or segregation between the living and the dead lung (Fig. 8, Plate III.) ; this line is gradually added to by fresh exudations, which, successively, become organised : from the vessels of the internal surface of this encysting wall serum is thrown out, which gradually breaks down the bond of connection between the sac and its contents ; hence, in cutting through such a cyst at this period, a quantity of sanious fluid escapes. The separation of the sac from the dead structure is not always complete, and in such cases the *segregating chasm* (Fig. 8, Plate III.) is interrupted.

In the course of time (the length of which will depend upon the strength and general healthy condition of the animal's constitution, and the extent of the obliteration of the vessels of supply) great activity takes place in the cyst wall ; large masses of granulations are formed, which gradually extend from one side of the cyst to the other, and present much the same appearance as the moderator bands of the heart.

The encysted mass slowly disintegrates (liquefies), by the agency of the living granulations (the action going on all round the granulations where they pierce the dead matter), into a thin, ichorous pus, giving rise to great irregularity of its outer surface : portions of the mass, when it is forcibly removed, being left in the spaces between the granulations.

The coloured sketch (Fig. 8, Plate III., with references) illustrates the segregation of the dead lung ; and the woodcut (Fig. 1, Plate XI., with references) will explain the appearances presented, on section, of a cyst and its contents.

All parts of the sac are not equally active ; but the more vascular its internal surface, and the more vigorous the formation of the granulations, the more rapidly does the imprisoned lung become liquefied and absorbed. The granulations, as age advances, become paler in colour and tougher (more condensed) in texture ; and ultimately—if their growth is not checked—fill up the cavity of the cyst ; the lung, in fact, is replaced by an organised fibrous mass, which may subsequently become semi-cartilaginous, or undergo secondary degenerative processes. If the process of liquefaction goes on, without absorption, an abscess is formed—the contents of which may be odourless or very fœtid. In rare cases—*i.e.*, where the interlobular tissue has not been so much interfered with, but in which there has been destruction of the lobular—the former becomes hypertrophied, and the latter undergoes caseation, and is ultimately removed (by degrees) by absorption without the formation of an abscess—the appearance produced somewhat resembling tubercle (Fig. 6, Plate X.)

If the walls of the sac are inactive and of a pale colour, neither liquefaction nor absorption of the dead lung results ; and it ultimately resembles a piece of dried

leather, and bears the same relation to the cyst as the kernel of a nut does to the shell. Sometimes the cyst becomes cartilaginous, in which case the dead lung is permanently imprisoned and cannot possibly undergo any change, nor can it work any harm to the system. In some cases ulceration of the wall of the sac takes place and extends through the nearest bronchial tube, atmospheric air gains admittance, the contents of the cyst undergo decomposition, the breath becomes fœtid, auscultation reveals a cavernous *râle*, hectic fever sets in, portions of the dead lung, with fœtid pus, are expectorated, purulent infection (*septicæmia*) follows, and the animal rapidly sinks. If the cyst is completely evacuated, and the animal survives, its walls collapse, become adherent on their internal surface, and produce a cicatrix or puckering externally; and if the thoracic walls have been originally bulged (*gibbous*), they will now, though gradually, fall in.

In many instances small masses of degenerated lung become detached, and, from ulceration and perforation of the coats of a neighbouring vessel, sucked up into the pulmonary circulation: becoming arrested in various parts of the sound portions of lung, they lead to the formation of numerous small (*miliary*) abscesses, which may remain discrete; or coalesce and form vomicæ.

In other cases emboli are detached from the coagula in the injured vessels, carried away by the circulation, and impacted either in the vessels of the sound portions of lung or in those of other organs; producing infarcts, with their usual results; one result—*i.e.*, multiple abscesses (pyæmia)—is depicted in Fig. 11, Plate X.

The changes in the blood-vessels of the injured lung are those which result from occlusion and inflammation of the vessels in any part of the body, are often well marked, and have been before noticed.

Changes in the lymphatic glands—in those cases in which they are not destroyed by the active processes induced by the acridity of the lymph—are hypertrophy, atrophy, induration, and calcification; while the lymphatic vessels become occluded by the inflammatory changes going on in their walls and valves.

The degenerative changes in the pleural adhesions are molecular and fatty, and lead to empyema, with, in many instances, secondary suppurative pleuritis.

Sub-pleural abscesses occasionally occur, and even, in extreme cases, involve the intercostal muscles, ribs, and subcutaneous tissues.

The results of Zymotic Pleuro-Pneumonia, in other structures or organs than those originally involved, are—chronic indigestion, chronic tympany, chronic diarrhœa, tendency to choking, occasionally dysentery, emphysema, hypertrophy, collapse, or pyæmia of the sound lung, and epi- or peri- carditis. All the affections of the digestive organs above mentioned result from one of two causes—viz., blood-poisoning, leading to vicarious ulceration; or, interference with the tenth and sympathetic nerves, the latter cause giving rise to debility or partial paralysis of the coats of the involved organs, and consequently interference with their functions.

Emphysema results from several combined or independent causes; as partial immobility of the diaphragm, from the pressure of the hepatised lung upon its anterior surface or upon the phrenic nerve; paralysis or atrophy of the muscular fibres of the bronchioles, as a consequence of functional interference with the tenth nerve; violent coughing, induced by the lodgment of portions of dead lung in the bronchia; or from the sound lung being overtaxed by its having to take on compensatory action. Depending upon the cause will be the suddenness or otherwise of the attack of emphysema; if it has been sudden, the alteration in the condition of the victim will be very marked, and ultimate recovery—especially if the amount of lung-structure originally injured be large—doubtful.

Epi- and peri- carditis are serious results; the inflammation is intense, both membranes in the earlier stages becoming thickly bestudded with villous-like growths of an intensely scarlet hue, and analogous to those seen in the early stages of tubercle or pyæmia.

The inflammation is the result of conveyance (*auto-inoculation*) of the acrid irritant material from the contiguous diseased lung by the lymphatics,—this conveyance, as with the contaminating virus of tubercle, cancer, and erysipelas, going on only within a limited area.

The ultimate consequences of this inflammatory process in these membranes are—healthy organisation of the products, producing adhesions and circumscribing the action of the heart; great thickening and vascularisation of the membranes (Fig. 7, Plate II.); hydrops pericardii; and, degeneration of the exudate, giving rise to a large collection of pus in the pericardial sac.

It is seldom that the process extends to the walls of the heart, or to the endocardium; but the lesions mentioned frequently produce sudden death, although, in some cases, the animal may have appeared previously to be thriving and doing well.

Great care is often required in order to avoid confounding these pericardial changes with those produced by the penetration of foreign bodies from the stomach. In the latter case, the course of the foreign body can be traced—its track being marked by circumscribed induration and by discoloration, the latter resulting either from pigmentation or the formation of oxide or sulphide of iron.

PATHOLOGY.

I have purposely chosen to treat of the pathology of this affection after considering its pathological anatomy; and I have done so because I felt myself bound to notice an article on Zy. p. p. by Dr. Yeo, which has recently appeared in the *Veterinary Journal*, and in the Journal of the Royal Agricultural Society of England.

In the paper before referred to, on the Nature and Pathological Anatomy of this disease, which I contributed to the *Veterinary Journal* in 1876, I made the following remarks on its specific character :—

"From a consideration of the course and history of the disease, and of its morphological characteristics alone, we are justified in concluding that Zy. p. p. is a distinct and specific affection, and that, although the structures which have been injured by the localisation of its lesions, secondarily (unless dead) undergo inflammatory changes; primarily, the disease is a purely effusive one—*i.e.*, in the initial stage, effusion—simply and purely—is the characteristic lesion; in the second and third stage, passive are accompanied, and finally succeeded, by active processes; and in the subsequent changes (which I shall consider hereafter) inflammatory processes alone go on.

"In order to demonstrate the specific nature of Zy. p. p., we have other evidence than that above alluded to—viz., the presence, in the effused serum of the first and second stage, of an immense number of highly refractive and actively oscillating, though minute, micrococci (similar to those seen in the effusions produced by inoculations), which lose their activity, and disappear on the advent of sphacelus; being, in fact, in some instances, replaced by staff bacteria.

"Of the acrid character of the effused serum, we have ample proof in its effects upon the thoracic lymphatic glands.

"In no ordinary effusive disease—as, *e.g.*, acute Œdema of the limbs in horses—do we see such actively degenerative processes go on in the lymphatic glands as is seen in Zy. p. p., though the amount of work done is in each case equal. Again, if the effused serum is inoculated or injected into the subcutaneous cellular tissue of an animal or of man, inflammation, more or less active, is immediately induced; the results of which may be exudation and organisation of the exudate; suppuration, or sphacelus—the result depending upon the intensity of the inflammatory processes and the acridity of the injected serum."

Dr. Yeo's opinions notwithstanding, I do not see any reason to resile from the position I then took up; in fact, my convictions have, on the whole, been strengthened rather than shaken by the perusal of his remarks.

Before proceeding to consider his arguments in detail, I think I may justly complain of the cavalier-like manner in which Dr. Yeo treats mine.

In the first place, he has quoted just so much from my paper as seemed to suit his purpose, and no more, as, in referring to my summing up of the characteristic *post-mortem* appearances in the first and second stages, he has only quoted one paragraph in each instance, taking no notice of several other paragraphs which form natural corollaries in either case.

In the second place, he says that I have divided the *post-mortem* appearances into "three artificial and fanciful stages." Can Dr. Yeo divide the *post-mortem* conditions of any disease into anything else than artificial sections? He has not only divided

those of Zy. p. p. into artificial, but, in my opinion, into very arbitrary sections, especially when he seeks to recognise the pathological changes as marked particularly by two forms of exudation—viz., *clear exudation* and *opaque consolidation.*

As to the charge which he makes of my stages being *fanciful,* I have only to say that I adopted this arrangement, not only because, from a long and varied acquaintance with the clinical and anatomical characters of the disease, I felt justified in doing so, but because the division, to my mind, afforded me the best means of describing it according to the ideas which I entertained of its nature and progress.

Gladly as I—with all others who wish to see the nature of this dire malady unravelled—welcome Dr. Yeo into the field of comparative pathology, and acknowledging as I do the great merit of his paper, and also the value, to me, of many ideas which have been originated in my mind by its perusal, I may, nevertheless, be pardoned if I say that nothing which he has advanced has led me to materially alter my method of treating the subject, either pathologically or anatomically; consequently, I have introduced—with slight modifications, rearrangements, and additions—into this work nearly the whole of my paper from the *Veterinary Journal.*

The methods adopted by Dr. Yeo and myself of investigating the pathological characters of this disease are diametrically opposed to each other—he commences with what I look upon as sequels, and works backwards, while my starting-point is from the initial stage to what I may be permitted to call a critical point in the disease. I do not, as does Dr. Yeo, judge of its character by its terminations, but by the early pathological conditions.

Dr. Yeo acknowledges, as I have done myself, that there are many hindrances in the way of arriving at correct conclusions, one of the most important being that we cannot always obtain correct data as to the actual commencement of the illness. As one reason for this, Dr. Yeo blames the regulations at present in force for the suppression of the malady, as well as the insidious nature of its attack.

There *was* a time when no regulations were in force for the suppression of Zy. p. p., and this time gave to me opportunities of studying the clinical and necroscopical characters of this affection in every possible stage, from the shortest incubatory period to periods of several years subsequent to the attack. For upwards of twenty-two years Zy. p. p. has been an intimate acquaintance of mine, comparatively short intervals only during that period elapsing in which I have not had it to deal with; and I think I may say, without diffidence, that few practitioners have seen more cases, made more *post-mortems,* or had better opportunities of studying the disease in its numerous and protean features than myself.

The investigations of Dr. Yeo have led him to make several startling statements in connection with the starting-point of the disease and its clinical history. So far as I understand him, his contention is, that the primary action of the virus of the disease is on the bronchial mucous membrane, and that this membrane, and the surrounding

lung-structures are in a state of chronic inflammation for a long period before any clinical symptoms are evidenced of the presence of the disease. In fact, he goes so far as to say, "I am convinced that the lung disease *usually exists for months without being suspected*—[the italics are mine]—and invariably the beast is first thought to be sick only when the affection has spread to the pleura, and caused intense inflammation of that membrane, with its accompanying well-marked symptoms." In another place Dr. Yeo remarks that "no symptoms are produced until pleurisy, infarction, or some other complication, becomes developed." These are startling statements, I fancy, to all who have had the opportunity of watching the disease. If they were true, it would follow that all animals (and they are legion) which have been brought from districts where the disease was unknown, and have contracted it after varying periods of time —three weeks to two months—must have had the disease for a long time prior to their translation to other, and infected, byres. These conclusions and statements are quite opposed to all clinical experience; and I would ask if it is possible for an animal to suffer from bronchitic irritation for a period of months without showing some external manifestations of its existence, seeing that the slightest irritation of this membrane, in any animal, always gives rise to unequivocal evidence of its presence. Further, is the inflammation of the pleura always so intense as to lead to the production of prominent and unmistakable symptoms? I answer that it is not; the pleurisy as often partakes of a sub-acute character as it does of an acute, in fact, it may frequently be looked upon as of a *latent type;* and we well know that in practice we may have horses working up to a day or two before death with unsuspected pleurisy, and their chests half full of water. The same thing holds good in other domestic animals.

The statements of cow proprietors as to the length of time their animals have been ill are very frequently untrustworthy and misleading, as they often conceal the truth in order to avoid the penalties which are attached to their non-observance of the Privy Council regulations. More than this, dairymen have a habit of treating their cows for indigestion, maw-bound, &c., for some time before they call the attention of the authorities to the fact that disease exists on their premises, or before they seek the advice of a professional man.

The statements made by these men are, in many instances, utterly at variance with probabilities. One instance only of this will I quote, and which happened to me very recently. The animal in question was an old Danish cow, which had been purchased from the country about three months, and kept in a byre alone. I was asked to look at her late one evening, and was told that she had only been ailing for about a week, the son of the proprietor stating that she was suffering from constipation, and that he had given her large quantities of physic without any effect. A very short examination enabled me to decide upon the nature of the case, and when I informed him that the animal was suffering from old standing Zy. p. p., involving nearly the whole of the left lung, he immediately remarked, "Ah! I knew all the while it was it." Now, mark

what was revealed in the autopsy :—1stly, One of the worst cases of the disease which I have met with for a long time, involving, as I have said, nearly the whole of the left lung, and of considerable standing. 2ndly, Advanced tubercle in the apices of both lungs, with ulceration through the bronchial tubes, and intense inflammation of the membrane wherever the tuberculous products had come in contact with it. Instances of this kind I could relate *ad nauseam*.

It is not by making *post-mortem* examinations, and listening to second-hand evidence, that the clinical characters of the disease can be best studied, but by anticipating its attack, and carefully watching its progress through all its stages. In numerous instances I have been called in when proprietors have only noticed "*something a little wrong*," and where they have been quite innocent of the nature of the disease. I have had the animals killed on suspicion, or because they were fat, and found the disease present, with all the initial pathological conditions which I have described well marked.

Reverting to the bronchial membrane, and the order in which it is implicated, I may remark—1stly, That at one time I entertained some such idea as that held by Dr. Yeo—viz., that the starting-point of the disease was in the bronchial membrane; and in one of my earliest lectures on the subject I made the following remarks :—

"Great differences of opinion exist as to which part of the lung the effusion is first poured out by—*i.e.*, whether by the vessels of the bronchial mucous membrane, the connective tissue, or the lobules. For some time I held the opinion that the effects of the poison were first exerted on the bronchial membrane, and that the infective action spread thence to the adjacent structures. I arrived at this conclusion from observing that in many instances the bronchial membrane presented traces of diseased action before the surrounding structures were much involved. This opinion I have since seen good reason to modify, as in very many cases extensive parenchymal effusion and consolidation exist with but slight, and comparatively recent, changes in the bronchial mucous membrane; and I am now inclined to think that the various components of the lung are attacked indifferently."

That the latter is the correct view of the case I am more than ever convinced, and that while several, or the whole, of the component parts of a lung may be, and often are, simultaneously involved, the primary lesion may be located in either one of them individually. If its primary effects were exerted on the bronchial membrane, it is not likely that we should have so many *small* centres of disease in the lungs of one and the same animal.

2ndly, I think there is not much difficulty in explaining the fact that the greatest amount of secondary interstitial inflammation takes place at the root of the lung, when we consider that here the connective tissue is more abundant than elsewhere, that it is nearest to the centre of circulation, and that it is exposed to the multiplied action of the infective particles contained in the lymph, by the convergence of the lymph vessels

towards this particular part, in order to reach the pulmonary and bronchial glands. That the secondary inflammatory action is more intense here than elsewhere is, I think, plainly shown by the ulcerated condition of the proximal extremities of the large bronchi, and by the fibrinous plugs (by which they are usually occluded) giving evidence of far greater age at the proximal than at the distal extremities. It will be frequently found that the proximal portions of the plugs are not only opaque, dense, and hard, but that they show numerous depressions corresponding to the granulations on the interior of the tubes, while the peripheral portions are only semi-solid and translucent.

The next point we have to consider is the pleural complication, and in connection with this I must notice a remark of Dr. Yeo's to the effect that, "as may be seen from the brief summary of the views held on this subject, it seems generally agreed that the pleura, or the sub-pleural and interlobular tissue, is the part first affected." As I am, I presume, included in this generalisation, I must emphatically disclaim any participation in the view that the pleura, or the sub-pleural tissue, is first affected. As I have before stated, I believe that the pleura is seldom the primary seat of the disease, and I cannot help thinking that much unnecessary labour has been expended in endeavours to prove that it is. Many cases have come under my observation in which circumscribed patches of pulmonary disease existed without the slightest evidence of pleural disturbance; on the contrary, I have oftentimes seen the pleural disease out of all proportion to the pulmonary.

In reference to the vascular lesions, I have referred (briefly, it is true) in my anatomical description of the disease to every form of these lesions—to inflammation, thrombism, embolism, and infarction; and I will only here remark that I am satisfied that the usual form of black hepatisation is largely due to the causes to which I have attributed it—viz., to vascular engorgement, interference with the nutrition of the vessels, extravasation and pressure; with blocking up of the bronchioles, thus preventing the access of air to the imprisoned blood corpuscles. The black hepatisation is not always in the form of a cone; usually, and within even circumscribed areas, we find several lobules showing black, and several red, hepatisation side by side—or indiscriminately mixed. That the black hepatisation is often due to infarction is patent, but in my experience true infarction more frequently takes place in the comparatively healthy adjacent lung-structure than in the parts originally affected, and produces totally different physical conditions. In the case of the lung from which the section shown in Fig. 9, Plate X. was made, true infarction of a portion of one of the small lobes supplied by the plugged vessel, shown at *e e* in this figure, existed; but there was not the slightest evidence of the part having been primarily engaged in the disease. In infarction, the lobular tissue (unless degeneration has commenced) is more elastic, the interlobular tissue is stained of a darker colour, and shows less hyperplasy than is the case in the areas of ordinary black hepatisation.

As to the disease of the vessels themselves, we not unfrequently find, by careful examination, that ecchymoses exist within their walls (as shown in Fig. 9, Plate X.), and that these form the starting-points of the subsequent changes. On the contrary, we sometimes see the intima showing disease with but very slight interference with the outer coats or the vascular sheath; and it appears to me that the probability of the vascular disease originating (as I am satisfied it often does) *as the result* of thrombism or embolism is too much overlooked.

We should be careful to separate, as far as possible, primary from secondary or even tertiary lesions if we wish to arrive at definite conclusions as to the nature of this most incomprehensible and puzzling malady; and we must not forget—as I think is too often the case with investigators—that there are such things as relapses and second attacks, which, I need scarcely remark, materially alter the usual anatomical conditions of all diseased actions.

Finally, in endeavouring to arrive at some satisfactory conclusion as to the nature and pathology of this disease, we are bound to confess that we know but little on this point. Its manner of comporting itself (if I may use the expression) is totally different to that of any other zymotic affection.

It is like, yet unlike, some blood affections, and between it and other zymotic affections there are important differences. That it is due to a specific virus I have, as before remarked, very little doubt, but the exact nature and mode of action of this virus is at present a mystery: that the virus is endowed with great vitality, that it is disseminated by intermediary bearers, that, having once gained access to the system, it reproduces itself, and, like other ferments, produces well-marked local effects upon important organs, and that it produces a zymotic action in the blood—are facts proved by its clinical characters and its general behaviour: but why it should not exert any effects on the lungs when introduced into the system by inoculation, though giving rise to pathological changes in the tissues of the inoculated part exactly identical with those seen in the lungs (the same peculiar microscopical bodies which I have before described being even found in the fluids of the lungs and the inoculated tissues alike); why, after inoculation, such a comparatively long period should elapse before any effects are visible; why its period of incubation is in so many instances of great length; what becomes of the virus after it is inhaled—*i.e.*, whether it lies dormant in the bronchioles and air-cells, or becomes absorbed into the blood, and circulates and retains its vitality there, waiting until the condition of the system is favourable for its further reproduction—or, why its effects should be expended on the lungs alone, are questions which we cannot at present solve any more than we can assign a reason for the remarkable behaviour of the poison of rabies, and the peculiar elective affinities of other virus, or even of toxological agents.

That the fluids of a Zy. p. p. lung produce an infective form of inflammation is certain—the fact being amply demonstrated by its effect on the lung-tissue, and on the

tissues of animals (not only cattle but dogs) and man when introduced therein. One or two peculiarities I may note in reference to this virus, viz., that it does not appear to exert any irritative effects on mucous membranes when applied to them, as I have on several occasions had small quantities of the fluids, while making autopsies, projected into my eyes without experiencing any ill effects; neither do the diseased lungs when ingested appear to produce any other effects upon animals than, in some instances, a moderate attack of diarrhœa; and the lymph does not produce any effect on the bronchial membrane when injected directly into the windpipe, or when it finds its way from the overflowing tubes of the diseased into those of the healthy lobes; lastly, the effects of lymph on the uninvolved portions of lung, when it gravitates thereto from superjacent diseased parts are very different from what is seen in the parts primarily attacked.

It is not, I think, a remote probability that the labours of the microscopist may yet be rewarded by the discovery of some definite form of microzyme or its products either in the minute capillaries of the lung, the coats of the vessels, or in the blood itself. The subject is both a wide and tempting one to those who have the time and means of prosecuting its study at their disposal.

TREATMENT.

If determined upon, must be energetic. Nature—probably more in this disease than in any other—indicates to us by the crisis she brings about the line of treatment we should adopt.

In all cases careful attention must be paid to the hygienic surroundings of the patients; a plentiful supply of pure fresh air allowed, but without producing draughts; the skin should be protected by a light rug, but it must be sufficiently thin to allow of cutaneous transpiration; feeding need not be thought of in the early stages, and afterwards all solid matter should be withheld, and nourishing semi-fluid matter allowed, or, if the animal refuses to take it, drenching must be had recourse to. Milk, glycerine, and raw eggs are valuable in the acute stages; cod-liver oil in the convalescent; molasses, in tolerable quantity, is beneficial in any stage. Brushing the skin should be vigorously carried out through the whole course of the disease.

Medicinally, laxatives—hydragogue salines in preference—should be always administered in the outset. The abstraction of a moderate quantity of blood in the first stages does no harm if the animal's condition is good, and the pulse tolerably strong; but venesection is inadmissible in the later stages—it is absolutely injurious, even if the operation can be performed.

Skin and diuretic medicines—as cream of tartar, acetate of ammonia, camphor and nitric æther, with carbonate of ammonia—should be freely administered in the

early stages; while stimulants, with terebene, turpentine, creasote, camphor, and oil of juniper, may be substituted in the later. In the convalescent stages, iron compounds, arsenic, and hypophosphite of soda are indicated, with nux vomica or strychnine combined or alternated with sulphuric acid, as stimulants to the digestive organs. Throughout the course of the disease anti-zymotics, as the salicylates or sulphites, may be judiciously employed. An alternative course of treatment—the sedative, *e.g.*, the administration of aconite, tartar emetic, and nitrate of potash—may be adopted; but I have here indicated the line of treatment I have found most successful.

Externally.—Counter-irritation should be had recourse to. Various methods are adopted for carrying it out—as setons, rowels, hellebore plugs, and blisters—each method having its advocates; but whatever is done in this direction should be done effectually and in the early stages. Setons may be inserted in the dewlap or behind the shoulders, the best material for the purpose being rope about the thickness of clothes line, made of equal parts of hemp and horse-hair, saturated, after insertion, with an irritant, and freely moved and dressed with antiseptic applications twice a-day for several days. Rowels and hellebore plugs do not differ in their action from setons.

As a blister, mustard may be applied, and repeated if it is thought necessary, but by far the most effectual and most active counter-irritant, and the one most easily applied, is a liniment composed of—

Ol. Terebinth.
Liq. Ammon Fort. Aa. ℥ij.
Ol. Rapii ℥vj.
Ol. Crotonii ʒ.

One application of this, if applied with friction, and afterwards covered with a warm rug, is sufficient, except in very thick-skinned animals—in young and thin-skinned cattle half the quantity of croton oil will be sufficient.

PREVENTION.

The consideration of the measures to be employed in the prevention of this disease opens up a wide field for discussion, and it is a point upon which everybody connected with cattle considers himself qualified to give an opinion.

The character and extent of preventative measures recommended depend largely upon the individual views entertained of the nature of the disease—*i.e.*, whether it is infectious *and* contagious, only infectious, *or* only contagious.

At the outset, I may remark that the measures which have been hitherto adopted in Great Britain have proved totally inadequate for the suppression or prevention of

the disease, simply because they have not struck at the root of the evil—have, in fact, only skimmed the surface.

One great bugbear to the adoption of adequate legislative measures has been the cry of *what will the trade, what will the public say?* and further, the fear of interfering with the rights of the public has been urged against the adoption of proper and stringent suppressive measures. In this matter, I maintain that *the trade* requires to be saved from, and in spite of, itself. The weal of the many should not be sacrificed to the gain of the few; and although the trade would be temporarily embarrassed by the adoption of stringent repressive measures, it would in the end, by the greater freedom which could be given to the movement of animals, and by the removal of a tangible cause of dread from the minds of purchasers, be the gainer.

I have said that "the trade" requires to be saved from itself; I say this advisedly, and its justification is found in what is seen to go on every day in infected districts amongst dairymen, farmers, and dealers. If a stockowner detects such a disease as Zy. p. p., in his herd, he will, in many cases, keep the matter quiet until he has succeeded in disposing of all animals fit for the butcher or even for store purposes; after doing this, he will report; or, in some cases he will kill the affected animal, and either secretly dispose of it about his own premises, or smuggle it away, to be dressed in some quiet out-of-the-way corner, and finally conveyed into the municipal boundaries by some "blockade runner"—*i.e.*, low butcher—to be disposed of as inferior meat in the low shops, or to be converted into sausage, collops, and preserved meat or other suchlike delicacies. The methods adopted by the blockade runner to convey, undetected, his contraband carcases into a city or town are numerous, varied, and ingenious: a load of straw or hay, a few sacks of potatoes or chaff, a little household furniture, and a variety of other commodities, cleverly stowed over the proscribed flesh, serve to deceive the, often overworked, inspector. But while the original owner of the diseased animal is thus conniving at an illicit traffic for the sake of a temporary gain, he is at the same time preparing a rod for his own back the stripes of which he will inevitably feel in due course. Such men forget that events march in circles, and that the disease which they are anxious to dispose of to their neighbours may (in a roundabout way) return to them again, or the very animals they have sold may be mixed with others from which they may themselves, in all probability, subsequently purchase.

In discussing the methods to be adopted in the prevention of Zy. p. p., we require to consider them as applicable to home as well as to foreign stock, and to ask the question *whether repressive measures can be of any service which do not apply equally to every part of our own kingdom and to foreign countries?* The answer is simple, it is a negative one—manifestly, it were absurd to cleanse our own houses if we forthwith allow our neighbours to step in and soil them; equally absurd is the reverse.

It would appear on first sight that in the past our neighbours have had the

advantage, because they have only had to run the risks of a short sea-voyage and a few hours' detention at the ports in order to place them on an equal footing with ourselves, and enable them to dispose of their contaminated cattle to the best advantage; but is this so? undoubtedly it is, providing that the disease is present in a mild form, or lies dormant sufficiently long to escape detection at the port of debarkation; but if the reverse, every animal is slaughtered, and, especially if a large proportion happen to be store stock, the loss is great; while if an animal is observed in our own fairs and markets to be suffering from an infectious or contagious disease it is immediately impounded, but its companions are allowed to go free: thus our neighbours have had, and will now have, to submit to the slaughter of all animals *which have been in contact* with diseased ones, whereas we are only called upon to slaughter (from markets or fairs) *the actually affected.*

The prohibition of importation of store stock should be rigidly enforced in the case of scheduled countries, all fat animals from which should be slaughtered under careful and strict supervision at the ports of debarkation, and the hides or skins and offal *thoroughly* disinfected.

To prohibit the importation of store, and especially dairy, stock from countries which are well known to be free from contagious and infectious disorders, is simply suicidal, as we want all the dairy animals it is possible for us to obtain, particularly while our own dairies are year by year decimated by Zy. p. p., and while so few animals are raised.

Turning from the foreign aspect of the question, we have not a very pleasing picture to contemplate in our method of dealing in the past with home stock to which, and to Irish cattle, every fresh outbreak of Zy. p. p. which has come under my notice for a considerable period could be traced.

The questions to be thought of in connection with home regulations are—

Firstly, Were the laws which regulated the importation of Irish cattle sufficiently protective? To this I answer No; as it is well known that they have frequently introduced disease where it was previously unknown, although, as with foreign cattle, the blame is thrown upon them when they are not always deserving of it; in many instances, in fact, from the lowering effects of sea and railway travelling, they readily contract diseases from the first affected animals with which they come in contact, and in their subsequent journeyings spread it rapidly and widely.

A very striking instance of the way in which foreign cattle are often credited unjustly with the introduction of disease has recently come under my notice. A Danish cow was purchased by a dairyman at a public auction on the 11th September; my attention was directed to her on the following evening, when I found her suffering from Zy. p. p., in a very advanced stage. This animal was sold with a lot of Danish cows which were landed at Leith on the 9th, and inspected by me on that and the following day, and it was assumed at the auction by the purchaser that she had formed

one of the lot so landed, but strict inquiry soon brought to light the fact that the cow had been sold at the same auction some weeks previously, and in the interim had been depastured with affected animals.

2ndly, The thirty days' prohibition of the past has been absolutely useless—it was utterly inadequate, as no fact is better substantiated than that the period of incubation or the vitality of the virus of Zy. p. p. is on the average from four to six weeks, in very many cases three months, and in not a few double or treble that time. It is an encouraging circumstance that in the new Act a move has been made in the right direction by extending the period of quarantine or restriction to fifty-six days.

3rdly, Has compensation for slaughtered animals been of any real service? Still the question must be answered in the negative. In many cases—*i.e.*, where compensation has been liberal—it has simply been holding out a premium for the nursing and perpetuation of the disease, for it is a well-known fact that certain classes of animals are purchased immediately before calving at a low figure on the calculation that about six weeks will elapse before they contract Zy. p. p., and that by this time the calf, and the yield of milk will, with the compensation, leave a tolerable margin for profit. In other cases compensation has been too small to encourage owners of diseased cattle to report, thus leading them to keep its existence a secret in order to enable them to dispose of their animals to the best advantage. Compensation should undoubtedly be given, and I think the present arrangements are far more satisfactory than those which have been in force.

The grave defects in the regulations which, up to the present, were applied to Zy. p. p. were—1stly, That they were optional, and consequently were not uniformly carried out, some local authorities observing them strictly, others systematically ignoring them, or only observing them in such a slip-shod manner as to render them practically useless; hence if the disease was stamped out in one, it was immediately reintroduced from another district.

2ndly, While provisions were made which prevented the removal of animals from infected premises for a period of thirty days, no regulations whatever were brought to bear on the introduction of healthy animals into infected byres; in this way fresh fuel was constantly being supplied for the too willing fire, and the disease was eternally perpetuated. Under the new regulations this anomaly has been done away with, as "no cattle can be moved into an infected *place* until all the cattle in the infected place have died or been slaughtered, or until the cow-sheds or other places where the diseased cattle were kept have been, as far as practicable, cleansed and disinfected."

Has the system of inspection which has been hitherto adopted been of any practical value? I answer, that it has not, simply because local authorities have been allowed to appoint qualified inspectors or not, as they thought best; consequently, while one local authority appointed properly qualified inspectors, and carried out the provisions of the Act, so far as was practicable, in its entirety, a neighbouring authority satisfied

itself with appointing police constables (or other persons even less qualified) as inspectors; the result being that, in very many instances—indeed, in the majority—they not only did not take the trouble of rigidly endeavouring to find out the existence of disease, but when they had discovered its existence, they had to trust to the honesty and skill of the owners as to the actual nature of the disease from which their beasts were suffering.

On numerous occasions I have, in common with other veterinary surgeons, pointed out these anomalies, and the utter inadequacy of the law, as it stood, to control, in any way, the spread of the disease.

I, as did others, also urged the necessity of branding and registering all animals in infected districts, with a view to controlling their movements and placing them under proper supervision.

It is gratifying to find that the new Act takes cognisance of and remedies these anomalies; and I am satisfied that if the provisions of the Act, in this respect, are faithfully carried out—and I see no reason why they should not be—that not only Zy. p. p., but other zymotic diseases, will in time be expunged from the list of British maladies.

Has compulsory slaughter been of any use? Undoubtedly so where it has been effectually carried out, but, as with inspection, while one local authority has done its duty in this respect, numerous others have grossly neglected to fulfil it, and have allowed things to go on pretty much as they found them.

Has the system of disinfection hitherto adopted assisted in arresting the spread of the affection? Still a similar answer must be given.

In carrying out disinfection, two obstacles have had to be met. 1stly, The supineness of proprietors, and their great dislike to the carrying out of compulsory measures, with, in some cases, a disbelief in their efficacy. 2ndly, The badly constructed, ill-drained, and ill-ventilated byres have frequently offered insurmountable obstructions to the proper performance of disinfecting operations, or of any other, and very necessary hygienic measures.

All habitations for animals should be constructed as much as possible—above, below, and around—of non-retentive (*i.e.* non-porous) material, and material which offers little surface for the lodgment of disease germs, and which can be readily cleansed.

In disinfection as a prophylactic measure against Zy. p. p., the great vitality of the germs seems to have been overlooked, and it also seems to have been forgotten that a coat of lime-wash—unless hot—is an excellent preservative.

As in the case of Glanders and Farcy so in Zy. p. p. I have, where opportunity has been afforded, had all wood-work removed and burnt, or, failing this, had it thoroughly painted over with a coat of gas tar (the best disinfecting sheathing, in my opinion, which we possess), as also the iron and brick-work; previously scraping therefrom all paint, old lime-wash, plaster, and dirt.

As to the floors of the stalls, unless they are made of asphalte, concrete, cement, flags, or non-porous bricks cemented together, it is the wisest policy to tear them up and relay with some of the substances above-mentioned, or with selenitic composition. The thorough fumigation of byres with sulphurous acid, chlorine, or tar is of the greatest importance. I have no faith in carbolic acid except it is applied as a very strong solution. The admission of a plentiful supply of fresh air and a perfect system of drainage—with absolute cleanliness and the prevention of over-crowding—are also great helps in the prevention of disease.

Isolation.—Not less important than the other measures of prevention discussed is isolation, but to be of any real use it must be carried out systematically—*i.e.*, every possibility of intercommunication between healthy, suspected, and diseased animals must be strictly prohibited, and all the healthy animals subdivided into small lots, and kept as far apart as circumstances will allow.

Quarantine.—Where practicable,—but, unfortunately, in large towns, where space is limited and ground valuable, it is, as a rule, impracticable—quarantine should always be adopted for a period of from three weeks to two or three months, the longer, of course, the better. Even though it may not (owing to the oftentimes protracted incubatory period) always be effectual, I know, from experience, that it materially diminishes the risks attendant upon the purchase of fresh stock.

Suppression of fairs, markets, auctions, shows, and the congregation of animals for any purpose in infected districts, is a *sine quâ non* in the prevention of this disease; but, unfortunately, the suppression of fairs, markets, and auctions, even temporarily, seems to be so much dreaded by the trade, and, consequently, so much opposed, as to render it an almost impracticable measure, except in particular districts, in which all the stockowners unanimously unite in their efforts for the eradication of disease.

If healthy animals alone found their way into markets in infected districts, the matter would not be of so much consequence; but how often do diseased animals (either accidentally or intentionally, so far as their owners are concerned) gain entrance there? and I cannot help expressing the opinion that the greatest error to be found in the new Bill lies in the fact that healthy animals are allowed to be moved out of markets in which diseased cattle have been detected, exactly as though they had never been in contact with, or within a long distance of, such diseased cattle. One auction yard or market is often sufficient, under the circumstances mentioned, to infect very large districts, and cause incalculable loss, great inconvenience and alarm.

Veterinary Medical Prophylactics.—The measures for the prevention of Zy. p. p. which come under the above denomination are numerous and varied.

All kinds of medicines—disinfectants, alteratives, purgatives, tonics, &c.—have from time to time, and in different parts of the country, been had recourse to as preventatives of Zy. p. p. Since the introduction of salicylic acid into the pharmacy it has been—particularly in some parts of the Continent—administered with some

persistence, with the object of preventing not only this, but several other zymotic diseases. So have carbolic acid, hyposulphite of soda, and the iron compounds. With the exception of salicylic acid, which is, by some, looked upon as a reliable prophylactic, I have tried them all, as also purgatives and such alteratives as potassa nitratis and sulphur, but without any very material or appreciably beneficial result.

The agent, *par excellence*, which I have found of the greatest service in this respect is arsenic; and where properly and judiciously used, I have had every reason to look upon it as a most effectual preventative.

In addition to Veterinary Medical, we have what may be more properly looked upon as *Surgical Prophylactics*—viz., setons, rowels, plugs, bleeding, and inoculation.

Setons, rowels, and plugs are usually inserted in the dewlap, the two former being saturated with a strong blistering agent, such as croton liniment; while hellebore root is in greatest requisition as a plug. Of the efficacy of these measures differences of opinion exist. I had, some years ago, reason to think that setoning, when effectually done, was of much use. For a long period I have not had an opportunity of giving it a trial, but, in conjunction with arsenic, I should, had I cattle of my own, be inclined to give it a very extended one. We know its efficacy in preventing certain forms of blood disease, and it is possible that it may so alter the condition of the system of an animal—we will not stop here to consider how—as to render it to some extent, and for a certain period, refractory to the action of the virus of Zy. p. p.

Bleeding is so much contra-indicated, and I have had so many proofs of its utter inutility as a preventative of any form of zymotic, or even of blood disease, as to render it unnecessary to allude to it any further.

Inoculation, amongst the surgical methods of prevention, is the one which claims our greatest attention. It is by no means a new method. FLEMING, in his "Veterinary Sanitary Science and Police," vol. i. p. 431, directs attention to the fact of its having been proposed and performed by Willems, of Hasselt (Belgium), in 1852; and that at that time the operation was much discredited (probably from a want of sufficient observation) by a large majority of veterinarians.

Fleming also calls attention to the favourable results of inoculation in various countries—as Belgium, Holland, Northern Italy, the Southern Tyrol, North Germany, France, South Africa, and the United States—and mentions such authorities as Reynal, Haubner, Ulrich, and other eminent veterinary surgeons, as entertaining a high opinion of its utility.

In an editorial article in the *Veterinary Journal* for April, 1877, Mr. Fleming says: "The evidence in favour of protective inoculation for this disease is now, it may be said, overwhelming. In every part of the world where it has been tried, its value appears to have been amply demonstrated, often even when the operation has been rudely performed by amateurs. The scientific experimental evidence is also most decisive and interesting, and we purpose referring to it at an early opportunity. We

have now only to note that, by a decree of February 10, the Dutch Government has authorised the burgomasters to order the compulsory inoculation of all cattle which have become suspected through cohabitation with diseased ones; the operation to be performed by a qualified veterinary surgeon, and the State to defray the cost—fifty centimes for each animal. If the supply of virus fails, the burgomaster may adjourn the inoculations on the advice of the district veterinary surgeon until the latter can procure more, which he must do as speedily as possible. If the owner or guardian of the cattle opposes the inoculation, legal measures are to be taken against him. Suspected cattle which, on the information of the veterinary surgeon, cannot be sufficiently isolated at pasture, shall, with all the precautions indicated by the latter, be moved to stables, and there isolated for the period prescribed by the royal decree of October 30, 1872."

In a communication in the *Veterinary Journal* for December, 1875, by Mr. H. A. BRADSHAW, M.R.C.V.S., Portrush, strong evidence is given in favour of the protective influence of inoculation.

In Australia the matter has been extensively and patiently investigated for some years; one of the most enthusiastic of these investigators being Mr. GRAHAM MITCHELL, F.R.C.V.S., of Melbourne. Mr. Mitchell's experience has led him to entertain an opinion highly favourable to inoculation.

I have myself received independent evidence from several sources of its great success in Australia. Mr. A. GREY, jun., a student of my own, tells me that during his stay in New South Wales the protective influence of inoculation was so generally acknowledged as to lead the Executive to prohibit importation of cattle unless good evidence of their having been successfully inoculated could be produced.

It must not, however, be assumed from these remarks that inoculation has no opponents in our Colonies; contrariwise, it has many, and, judging from the communications which have from time to time appeared in colonial newspapers, bitter ones too.

Having mainly referred to the matter from a foreign point of view, we will see what is known of it at home.

About the year 1859, when I was a neophyte in veterinary practice, the subject of inoculation received a large share of public attention; and in some parts of the Midland Counties the operation was had recourse to very extensively, and was performed in a variety of ways. One enthusiast, a cow-doctor—who had by some process of reasoning arrived at the conclusion that the liver was the seat of the poison—inoculating with small portions of the livers of affected animals. From some cause the operation at that time fell into disrepute, and was abandoned.

When I commenced my collegiate course, scarcely an animal came into the city of London that did not carry palpable evidence of having been subjected to inoculation; most of them, indeed, claiming close relationship with the Manx cats. About this time, too, or somewhat earlier, the subject received much attention in Scotland, and some of the most eminent veterinarians practised inoculation extensively. From causes which

I cannot explain, but certainly not always from ignorance or carelessness on the part of the operator, the operation was discontinued entirely in Scotland, and to a great extent also in London.

There are those now in the neighbourhood of Edinburgh whose experience of the operation at the time of which I speak has led them to declare, recently, that they would not again allow it to be performed on their animals on any consideration whatever.

In the Appendix to the Veterinary Report of the Privy Council Office for 1874, an instance is quoted where Pleuro-Pneumonia broke out in a stock of dairy cows which had been successfully inoculated by Mr. Priestman, who is, acknowledgedly, one of the most experienced operators in this country.

In the *Veterinarian*, October, 1878, the following note occurs:—"The Minister of Agriculture writes from Berlin to the President of the Central Agricultural Society of Saxony, stating that he is continuing his researches and experiments on the value of inoculation in Pleuro-Pneumonia, but that he is not as yet sufficiently convinced of its efficacy as a preservative measure to contemplate the introduction of any law rendering it compulsory."

Recently the matter has been again revived and brought into prominent notice in Edinburgh by the individual exertions and untiring and zealous efforts of an old pupil of the Dick Veterinary College, Mr. Richard Rutherford, of Bread Street. Having had the advantage of an extensive and personal acquaintance with the operation as practised by Mr. Graham Mitchell in Australia, Mr. Rutherford—who long ago expressed to me his determination of bringing it prominently forward at the earliest opportunity—has quietly and perseveringly been experimenting in the dairies of Edinburgh and Leith, and, so far as I can vouch from personal knowledge, with apparently gratifying success.

I have frequently remarked that this is a subject for imperial, not for individual consideration, and I repeat the remark here. A few experiments have been carried out in the Brown Institute in London, by Professor Duguid, under the auspices of the Royal Agricultural Society of England, and, so far as they have gone, the results have been positive in favour of inoculation.

Is it not worth the attention of the Highland and Agricultural Society of Scotland? The Society is ever to be found in the van of progress, and it might put its funds to many less worthy purposes than this.

Undoubtedly the operation of inoculation has some drawbacks, but can we find in anything, good unmixed with evil?

Mode of performing the operation.—Many methods have been adopted at different times and by different operators. The introduction of the virus under the skin, by the aid of a pipette, a grooved needle, or a woollen suppository;—incision and scarification of the skin, and bringing the inoculative matter in direct contact with the abraded surface, have each their advocates.

The most scientific and easy method of operating is by the use of the hypodermic (subcutaneous) syringe, and I am much surprised that this method has not been adopted by veterinary surgeons, as its advantages over others are very obvious, and all that is required is a small preliminary incision through the skin, in order to facilitate the introduction of the needle. Next to the subcutaneous introduction of the lymph by the syringe I most certainly prefer the *woollen suppository* (which is the method practised by Mr. Rutherford), viz., the introduction of one or two strands of ordinary stocking yarn, about two inches in length, underneath the skin; the yarn having been previously saturated in lymph, and introduced by the aid of a curved needle, and previous incision with a lancet or rowelling scissors.

If the latter method is adopted, the suppository may be moved backwards and forwards, once either way, in about twelve hours after introduction, and wholly removed in twenty-four. The moving of the suppository ensures the lymph coming in contact with an absorbing surface, and allows of the escape of any fluid which may be thrown out by the irritated vessels. If the yarn is left in for too long a period it undergoes decomposition, and may originate septicæmia.

Point of introduction of the virus.—Various parts of the body have been chosen by different operators in which to introduce the lymph, the parts usually chosen being those devoid of hair. The most convenient, and the most free from a variety of objections is the distal extremity of the tail, or rather about two or three inches therefrom, and for the purpose of future observation the hair should be previously removed close to the skin, for a distance of three or four inches.

The material for inoculation.—Great care must be exercised in the choice of this. The following particulars should be strictly observed:—

1stly, The lymph should be taken from the lungs of a healthy animal—*i.e.*, from one free from any other disease, particularly tubercle or cancer.

2ndly, And if possible, when the disease is in its very earliest stage.

3rdly, It must not be taken from the actually diseased part of lung, but from the surrounding healthy parts in which there is always more or less lymph which has infiltrated from the adjacent affected portions. Neither must it be taken from the effusions in the cavity of the chest, or from the bronchial tubes.

4thly, Great care must be exercised in the removal of the lymph from the lung-tissue. This can be done (*a*) by the aid of a pipette, or a subcutaneous syringe from the interlobular tissue; (*b*) by inserting small canulæ or grooved needles or teat syphons in the interlobular tissue, and allowing it to drain out into porcelain or glass vessels; (*c*) by puncturing the interlobular tissue with a lancet, and applying gentle pressure; (*d*) by cutting the lung into sections, and placing the sections on any ordinary drainer. Whatever method is adopted, the introduction of extraneous matter must be avoided, and every instrument or vessel used must be scrupulously clean.

5thly, The collection of the fluid should be accomplished in as pure an atmosphere

as possible, and where the fluid is not wanted for immediate use it should be preserved in stoppered bottles, or corked bottles effectually sealed. A small quantity (two per cent.) of common salt may be added to the lymph with the view of more effectually preserving it, without interfering with its activity. It may be collected in capillary tubes in the same way as in the collection of vaccine matter.

6thly, The lymph should be collected as soon after death as is practicable.

In the choice of animals for inoculation, and their treatment during the period of inoculation, some care is also required.

It were, manifestly, unwise to inoculate an animal whose appearance betokened the existence of any chronic disease, such as tubercle, and if evidence exists (though it must be borne in mind that the source from which this evidence—viz., the owner—is not always trustworthy, as whenever Pleuro-Pneumonia exists in a byre, and an animal shows any symptom of illness—even though it is only indigestion—the conclusion is at once arrived at that it is suffering from Zy. p. p.) that an animal has been the subject of Zy. p. p., inoculation is useless, as, if it were true that one attack gave immunity from others, the natural disease *must* be more protective than a localised artificial one.

Some operators—Mr. Rutherford amongst them—will not operate under these circumstances, neither will some inoculate if the animal is already the subject of disease, while others assert that the operation has a curative effect.

During the period of inoculation strict attention should be paid to hygiene, the tails kept as clean as possible, and in hot weather the byres should be kept cool, and the animals protected from the attack of flies, as they cannot, if the inoculation is successful, protect themselves owing to the great soreness of the tail. The inoculation of recently calved cows (as has been often proved in Mr. Rutherford's experience, and as I have had opportunities of seeing myself in one or two animals upon which that gentleman has operated) is injudicious, as the system is in an unhealthy state, and consequently untoward results are liable to follow. A fortnight is about the time allowed by Mr. Rutherford to elapse, after calving, before inoculating.

If the weather is favourable and not too hot, no harm can accrue from allowing the animals to go out to grass, particularly during the day; but exposure to inclement weather must be avoided.

General and Local effects of Inoculation.—The systemic effects are not always well marked, but in some cases a certain degree of fever—the thermometer indicating about 103° Fahr.—is observed; there may be, so far as my observation and inquiries are concerned, a little falling off in the quantity of milk in the case of milch cows, and occasionally a little interference with the appetite simultaneously with the elevation of the temperature, and slight excitement of the circulation—as indicated by quickened pulse. I am here assuming that the inoculation is successful, and its course regular. The general effects are developed with the advent of the local symptoms.

The local effects are as follows :—In a period varying, on an average, from ten to fourteen days, the inoculated part becomes swollen, hot, and tender, and the hair erect ; the incision may gape and discharge a serous or sero-purulent fluid, and when the inoculation is quite successful, a straw or amber coloured serosity oozes from the dermis, and collects in the form of more or less distinct, but small, vesicles ; in some cases no vesicles are formed, the serum simply oozing out and flowing evenly over the surface of the skin ; if vesicles are formed, they burst and discharge their contents in the course of a day or two ; in either case, the fluid becomes inspissated from exposure to the atmosphere, and forms, with the epidermis and hair, an adherent scurfy or scabby mass, which, in the process of time, falls off, leaving the skin more or less roughened, wrinkled, and hardened, and the hair somewhat erect.

The whole process from the day of inoculation to the period of desiccation occupies from four to six weeks.

If no local effects are produced within twenty-one days or thereabouts, the advisability of re-inoculation may be considered.

The evil results of Inoculation are—Extension of the inflammatory process along the tail to its root, and thence to the vulva, perinæum, vagina, pelvis, anus, thighs, udder, scrotum, and muscles of the haunch, back, and loins. This result is more likely to occur if the system of the animal operated on is unhealthy at the time, or if sufficient care has not been exercised in the selection of the lymph ; but occasionally it occurs where every precaution has been strictly observed, and in the hands of the most skilful operators ; doubtless in such cases the lymph possesses unusually virulent properties, or the system of the inoculated animal is peculiarly susceptible to the action of irritants. The inflammatory condition is allied in many respects to erysipelas, and we know how much more liable to this form of inflammation some constitutions are than others.

Although the inflammatory condition set up by inoculation with Zy. p. p. lymph is, on the whole, a circumscribed one, it is nevertheless an infective one, and as such—like all other infective inflammations—may pass far beyond its original boundaries, —mainly, of course, by the lymphatics.

When the inflammatory action is about to extend upwards, the local symptoms become intensified ; the heat, tenderness, redness, and swelling are increased, and the systemic symptoms are more pronounced.

If the inflammation terminates in sphacelus—either circumscribed or diffuse—the involved parts become of a dirty red colour, cold, and insensible, and a line of demarcation separates them from the healthy structures; moisture may, or may not, appear, and form phlyctenæ on the surface,—this will depend upon the form of gangrene—*i.e.*, whether it is wet or dry. In some cases large swellings are formed in the neighbourhood of the hips, which, on incision give exit to fluid or semi-coagulated lymph ; if undisturbed, this lymph may degenerate and form abscesses, or may become organised ; if abscesses are formed, they are sometimes succeeded, on eruption, by unhealthy, chancrous-looking, ulcers.

The inflammatory action may kill the animal by exhausting the vital powers (though it is wonderful to what length it may extend before alarming constitutional symptoms are developed), by involving the pelvic organs, by sphacelus, or by the absorption of septic matter-producing septicæmia.

Treatment of inoculated part, if untoward symptoms appear.—The majority of operators recommend (what indeed is the natural and most rational method of treatment) if there are any signs of gangrene or of extension of the inflammatory action, that the tail should be at once amputated, or, failing this, deeply scarified and freely dressed with antiseptic applications.

Constitutional remedies, depending on the character of the inflammation and the condition of the animal, should not be neglected.

Mortality of Inoculation.—The losses from inoculation are not great—at the outside, three to five per cent., where proper precautions are taken.

Rationale of Inoculation.—The advocates of inoculation hold that it renders animals proof against the contagion of Zy. p. p., if not for life, certainly for some months or even for years. How does it do this? It differs entirely in its results from all ordinary inoculative contagia, inasmuch—as already pointed out—as it does not produce the same visible effects as the natural disease, consequently we are not warranted in assuming that the virus enters the blood and circulates through the whole of the system—although the production of some febrile reaction would seem to indicate that it does so—and if it does not enter the circulation, it is difficult to understand how it is that it renders the animal proof against future attacks; if its effects were simply local, its action would not be in any way superior to a blister or seton, and of the wonderful change wrought—within a very short period—in the condition of the blood by the introduction of setons, we have good evidence in their power of preventing many charbonous diseases, such as black quarter, in cattle.

Inoculators also make at times two very contradictory statements—viz., that "the inoculation will not take if the animal is already the subject of Zy. p. p.," and that "inoculation is not only preventative but curative." How it becomes the latter, when the system is already saturated with the virus, is difficult to conceive—unless on the principle of counter-irritation.

However difficult it may be to understand the action of inoculation, it is certainly a fact that since its introduction into Edinburgh and Leith by Mr. Rutherford, Zy. p. p. has materially diminished; but time is, of course, required to prove that this diminution will be permanent, or that it is due to the operation in any great degree, as periods of decline in the disease are frequently observed in every infected district. It is an undoubted fact that those animals which have been successfully inoculated thrive very rapidly after recovery from its effects; and if it can be deprived of some of its objectionable features, and its prophylactic powers are permanent (though we can hardly hope this, seeing that the natural disease itself is not permanently protective),

it may yet prove a great blessing to stock-owners; and those who have so consistently and perseveringly advocated its introduction will deserve the best thanks of the profession and the public.

Pathological Anatomy of the Inoculated Part.—Whether we look at the structures at the immediate seat of inoculation in mild cases, or at the tissues in the pelvic region in virulent inoculative inflammation, we shall find a condition of matters closely allied to what is seen in the lung-tissue in the natural disease, at least so far as the pathological processes are concerned.

These processes are not confined to the skin and the subcutaneous tissues alone, they extend to the muscles and other adjacent organs.

If a section is made through the skin of the inoculated part it will be found that the dermis is greatly thickened, infiltrated with lymph of a yellowish or straw colour, and its vessels intensely hyperæmic; here and there hæmorrhagic spots will be detected.

The subcutaneous connective tissue, the structures of the vulva (occasionally the vagina, bladder, uterus, perinæum, and anus), and the pelvic fascia will be found in the earlier stages surcharged with the same yellow—fluid or semi-fluid—lymph, and in the later stages they are the seat of a new interstitial growth, which ultimately becomes organised, and, in case of recovery, forms a part of the system.

The lymphatic vessels are charged with lymph, the blood-vessels ultimately become involved in the process, and plugged; while the muscular tissue becomes pale, friable, infiltrated with lymph (moist), and its transverse striæ destroyed.

Miscroscopical examination of the tissues reveals extensive infiltration with cell elements in the early, followed by fibrillisation and organisation of the exudates in the later stages.

Micrococci, similar to those which I have described as existing in the lung-tissue, will also be found in large numbers. The affected parts possess a peculiar odour.

The lymphatic glands become inflamed and either indurated or caseous; in one instance recently, I found that hæmorrhage had taken place into the substance of a gland and the surrounding subperitoneal connective tissue to such an extent as to form a hæmatocele several pounds in weight.

If the process is not very destructive the glands become indurated and mottled, or undergo dry caseation.

Finally, in the prevention of Zy. p. p. (leaving inoculation for the moment out of the question) it is necessary that animals should be purchased from healthy districts and conveyed in clean and disinfected vehicles or carriages; that the general health and condition should be attended to—as depression or debility favour the lodgment and development of disease germs; that all byres or sheds should be built on sound sanitary principles, not inhabited (when newly built) before the walls are thoroughly dry and the drainage in perfect working order—be kept thoroughly free from accumu-

lation of matter of any kind and periodically cleansed and disinfected; and that inter-communication with infected places should be strictly prohibited.

In its suppression—slaughter, isolation, and external and internal disinfection can alone be relied on, with arrest of all movements to and from infected districts.

As to the working of the new Contagious Diseases (Animals) Act, sufficient time has not yet elapsed to enable us to draw any definite conclusion in the matter, but one thing of great importance I have already observed—viz., that where critical and frequent examinations are made of all animals in an infected place, and all those which are either actually diseased, or which present suspicious symptoms, are immediately removed, the number of attacks relatively diminishes and the lesions are of a much more circumscribed character.

I am satisfied that the pernicious system—which was allowed in some localities under the old Act—of keeping diseased animals about byres for weeks or months in the hope of their ultimately recovering has been the cause of Zy. p. p. remaining in our midst for a much longer period than it would otherwise have done.

FOOT-AND-MOUTH DISEASE.

SYNONYMS. — Murrain; Eczema Epizootica; Distemper; Epizootic Aphtha; Vesicular Aphtha; Vesicular Epizootic; Aphthous Fever.

All the above synonyms are applied, by different authors, to this well-known affection. Technically, at least in G. B., the term "Eczema Epizootica" is most frequently used, while the appellation of "Foot-and-Mouth Disease" is universal both with professional and laymen.

The word *Eczema* is synonymous with "boiling out" or "eruption," and signifies that a vesicular eruption accompanies the disease.

Epizootica has the same signification—viz., a disease raging over large tracts of country—here, as in all other diseases of an allied nature.

The term "*Murrain*," according to Dunglison, is derived either from the Saxon *mypphan*, "to destroy;" or from the Latin *mori*, "to die." It was probably applied to this disease from a conviction that it was identical with the murrains alluded to in scriptural history.

DEFINITION.—It is a vesicular eruptive, or exanthematous affection, due to a specific ferment, and having its lesions localised in the skin and mucous membranes.

CHARACTERS.—Eczema Epizootica is probably indigenous in the bovine tribe only, but there is no direct proof that it may not originate in the ovine species also. However this may be, there are few zymotic affections which, when once originated, attack so many animals, or which are so easily transmitted from one animal to another of a totally different species. It is indeed questionable if any animal is proof against the contagion; it readily attacks sheep, goats, swine, and poultry; it is easily transmitted to the human subject; it has been described as existing in the horse, the dog, wild fowl, deer, wild boar, &c.; I have seen it in the cat; my friend, Mr. Welsby, of West Derby, has described to me cases of it in the hare; while I have even heard it asserted that it attacks ferrets. With regard to the last assertion I may remark that the common sporadic affection "Eczema Plantigrada," which is produced in these animals by foul bedding, may be easily confounded with such an affection as the one under consideration.

Of the exact nature of the ferment of Eczema little or nothing is known. From the fact that a fungus, similar to the *oïdium albicans*, has been detected in the mucus of the mouth and the saliva, it has been conjectured that the ferment is of a fungoid nature; others, again, owing to these bodies having been found microscopically in the blood, muscles, and milk, have attributed it to some form of micrococci. Their presence here has no specific bearing; they are, as already remarked, concomitants of other zymotic affections.

In the case of one cow, which was slaughtered by my advice, suffering from multiple abscesses as a result of the disease, I discovered numerous micrococci in the milk—which was otherwise quite normal in character—but not a trace of them existed either in the blood or the contents of the abscesses. So far as the origin of micrococci in the milk is concerned, I think there can be little doubt that they have a local origin—viz., from the vesicles, and subsequent pustules, which so often form on the mucous membrane of the teats, and on the skin of their apices.

Foot-and-Mouth Disease is probably one of the most infectious and contagious maladies which affects domestic animals, and is the easiest of transmission. One very remarkable fact in connection with it is the effect which the milk of diseased animals produces on their young, and even in the young of other species.

In almost every outbreak of the affection in breeding districts, great numbers of young animals are found to die suddenly, and in most instances before anything abnormal is detected in the dams, and without the development of any characteristic symptoms in their own systems. Not only is the milk destructive of infantile life, but—as was proved in the practice of Mr. John Aitken, of Dalkeith, in 1872, and by whose kindness I had the opportunity of examining several of the stomachs of the dead animals (calves)—the whey is, though in a less degree, very deleterious also.

As the death of young animals under the above circumstances is a totally distinct pathological process from the natural disease, it will be advisable to consider it in this place.

At a period of from twenty-four to thirty-six, or in some cases forty-eight, hours prior to the development of external manifestations of the disease in the dam, sucklings (notably pigs) are observed to die without any premonitory symptoms of illness, and on a *post-mortem* examination being made, few or none of the usual signs of the disease are traceable.

The necroscopical alterations are confined mainly to the mucous surfaces—*i.e.*, the laryngeal, bronchial, intestinal, and—according to some authorities—renal. The changes are purely congestive, and, if the animal has survived a sufficient length of time, inflammatory; they are most concentrated and most intense in the stomach (fourth in the ruminant), the mucous membrane of which will be found, wherever the milk has lain in contact with it, intensely hyperæmic, and in some cases even hæmorrhagic—the milk is usually curdled. Aphthous patches have been described

as existing on the tongue. I have never seen such, and I imagine they do not exist except in those cases where the disease has been fully developed in the dam, and vesicles have formed on the teats or on their mucous membrane.

When the milk is possessed of very virulent properties, and the young animal does not die very suddenly, hyperæmic patches are developed in the mucous membrane of the small intestines, and serum is thrown out into the muscular tissues of the body and into the connective tissue of the more vascular glands.

Various theories have been promulgated with reference to the cause of death in these cases. So far as my own researches go, they incline me to the opinion that it is produced by the direct action of the virus of the disease upon the nerve centres, rather than by any direct local influence which it exerts on the thoracic or abdominal organs.

Eczema Epizootica in some outbreaks assumes very malignant characters, while in others it is equally as benign; it is, as a rule, much more severe at the commencement of an outbreak than when it has existed for some time. Exacerbations and declinations are very marked. Foreign—non-acclimatised—animals suffer very severely, and while it may linger in a district for two or three years, it may die, as it were, a natural death in the course of a few months. The outbreak of 1865 in Wales, and that of 1872 in England and Scotland, were the most severe and disastrous of any which has come under my observation.

As the disease is variable in character, so it is capricious in the choice of its victims, and in its local manifestations. In some seasons it expends the greater part of its force on cattle; in others, on sheep; in others, on pigs; while occasionally it attacks the domestic animals indiscriminately.

In different outbreaks it localises itself mainly in the feet, the udder, the mouth, the skin, and mucous membranes respectively; the more it is concentrated, the greater are its unfavourable results, and concentration in one organ or part renders it milder in others.

One attack does not give immunity from others; and not only may an individual animal suffer several times from it in the course of its life, but even twice or thrice in a season, though in the great majority of cases each successive attack becomes milder in its character.

The channels by which nature endeavours to eliminate the poison are—the salivary and mammary glands, the mucous glands of the bronchial and intestinal mucous membranes, and the skin—hence the secretions of the two former, the fæces, the expired air, and the cast-off epidermic scales all become the means of disseminating the virus. The effects of the poison on the skin are invariably well marked, desquamation of the cuticle being extensive; but while this is a common characteristic of many zymotic diseases, it nevertheless points to the necessity of encouraging the elimination of the poison by this channel; and, in practice, it is found that the more cutaneous elimina-

tion is encouraged the more quickly does the disease run its course, and the less is the severity of its results.

Eczema Epizootica claims a strong relationship with rinderpest and hog cholera, and, though it is only in very malignant cases that local congestions and extravasations are seen during its course and on necroscopical examination, it has many points of resemblance to both these diseases. These points will be fully considered hereafter in speaking of diagnosis.

HISTORY.—The descriptions given of different epizootic maladies by ancient writers most certainly lead to the conclusion that Eczema Epizootica has been an occasional scourge in different countries, and at different periods, for many, and probably remote, ages ; but, be this as it may, we have no definite proof of the affection having had a European existence prior to the seventeenth century, and all writers on the affection are agreed that, so far as Great Britain is concerned, at least in modern times, it was unknown until the year 1839, since which time it has been a tolerably frequent and persistent visitant.

Like many other affections of an allied type, it is exotic in its origin, and usually extends from the east to the west ; but outbreaks of it sometimes occur under such unusual and inexplicable conditions as to lead some authorities to assert, with some amount of positivism, that it originates spontaneously in our own country. On this point I have nothing more to say than what I have already said in reference to the origin of all zymotic diseases.

GEOGRAPHICAL DISTRIBUTION.—This disease is now acknowledged to have had existence in almost every cattle-raising country in the world, but while this is the case, it is nevertheless an undisputed fact that, although it has been introduced on several occasions into one or more of our colonies, it has not in these gained a very prolonged or very extensive footing.

VITALITY OF THE VIRUS.—Though its vitality is less than that of some other zymotics, and though it is generally believed to be comparatively short—thirty days, or thereabouts, after the recovery of the last animal—circumstances have come under the observation of practitioners which show that, in some instances at least, its vitality is great. Thus, Fleming, in his "Sanitary Science and Police," vol. i., p. 460, says :—

"Zundel has known instances in which stables have retained the virus for a very long time, either in their walls or in the air confined in them. The disease has been communicated to animals in such stables after they have remained vacant for fifteen days.

"The same excellent authority also states that he has known the virus to be preserved for a long period in forage, although this had not been impregnated by saliva from the disease, but only exposed to the atmosphere of the stable they had inhabited.

"One of the outbreaks in Australia in 1872 is said to have been due to the importation of a cow or bull from Britain, which had exhibited no symptoms of the disease

during the voyage. The virus is believed to have been present in the last truss of hay given to the animal, which sickened therefrom as it entered Sydney harbour.

"Many other cases are on record to prove the tenacity of the virus, but the following, which I have been fully assured are accurate by a competent observer of the facts, are perhaps the most striking, and afford an example of the subtle manner in which the contagion may obtain access to healthy animals, even when every care has been taken to guard them from it. These are the cases in which the disease is supposed to appear spontaneously, or to be due to 'something in the air.'

"About two years ago a farmer in the neighbourhood of Shorne, a village lying some distance from the highroad between Gravesend and Rochester, and isolated from all cattle traffic, bought a number of young cattle (twenty-four) at Kingston Fair. As he had suffered a good deal from the effects of aphthous fever, some months previously, he was determined to adopt every precaution to escape it for the future—consequently he had these cattle quarantined in a yard, where they remained for more than a month without showing any symptoms of the disease. At the end of that time he was desirous of feeding them out of troughs, and a number of these were brought from a field a long way off, where they had been employed to hold the mashes and other soft food given to the cattle which had been sick some four months previously. These troughs had been allowed to lie in the field from that time up till now, exposed to the snow and rain of the winter, and three days after the new stock had been feeding out of them, the first case of disease appeared, and soon the whole of the animals were affected. The troughs were not at first suspected, and the greatest mystery prevailed, as well as alarm, for such an outbreak seemed to put even quarantine measures at defiance. There was no disease in the neighbourhood, no droves of strange cattle had passed within miles of the farm, and no infected animals had ever been in this particular yard; and the mystery would have perhaps remained unsolved had the experienced veterinary surgeon (Mr. Martin, of Rochester), who was called in to attend the diseased stock, not been furnished with the history of the outbreak.

"The other case occurred in the same locality at another time. A farmer owned two farms in an out-of-the-way place, and some distance from each other. On one of the farms the disease had prevailed rather severely, and the animals had been fed out of the ordinary field hay-racks. The disease disappeared, and nothing more was seen of it until five months afterwards, when one of these racks was brought down to the other farm where the malady had not been, and very soon the cattle which fed out of it sickened. There was no traffic in the locality nor yet disease until the tainted rack was carried down."

Now, although no such instances of prolonged vitality have come under my own notice, I think those just quoted afford tolerably conclusive evidence on the point in question.

PERIOD OF INCUBATION.—Is, compared with other zymotic affections, short—viz.,

from twenty-four hours to three weeks, the latter being an exceptionally long incubatory period. The average is from two or three to five or six days.

INVASION.—Is, on the whole, rapid and pronounced, the manner of invasion depending upon the amount of poison received into the system, the condition of the host, and the surrounding circumstances.

DURATION.—Is variable, and is regulated by the intensity of the attack, and the care which is bestowed upon the patient; from ten to twenty-one days may be looked upon as the average period of duration where the disease runs a regular course and is not succeeded by important sequelæ. The duration of the results is extremely indefinite.

FATALITY.—Depends largely upon the character of the outbreak; in some seasons death in any animal is a rarity, while in others, great numbers succumb to the primary effects of the disease. More deaths from Eczema came under my notice in the year 1872 than in the whole of my professional experience besides. Though much of the mortality which sometimes characterises the disease may be looked upon as due to the virulent nature of the attack, much also may be attributed to the disgracefully careless manner in which some stock-owners treat their animals.

The results of Eczema Epizootica are productive of far more loss and suffering than are the primary processes, indeed in some outbreaks the direct and indirect losses so occasioned cannot (even approximately) be estimated.

PROPAGATION OF ECZEMA EPIZOOTICA.

No disease with which we are acquainted is capable of propagation in such a variety of ways as the one under consideration. It is propagated by Direct and Mediate Contagion, as the virus is both fixed and volatile, but it is only diffused through the medium of the atmosphere at comparatively short distances. As the saliva, the nasal, conjunctival, and intestinal mucus are highly charged with the virus, it is most readily spread by the conveyance of these secretions to healthy animals by many direct and indirect means.

Thus, food of every kind—as hay, straw, corn, mashes, roots, grass, which has been contaminated with saliva—becomes a splendid carrier of the ferment, and only requires to be ingested by other animals in order to propagate the disease.

It is a well-known fact that a wisp of hay saturated with the saliva of a diseased animal and introduced into the mouth of a healthy one is a ready and certain method of transmitting the malady; not only this, the exposure of contaminated dung, rugs, &c., in healthy byres is a sufficient means of propagation; and more than one instance has come under my immediate notice in which I was satisfied that the disease had been spread by affected animals wading and defæcating in streams which subsequently

coursed through healthy areas. This is, indeed, one of the reasons why Eczema is so often seen to follow the course of a stream.

Instances of propagation of the malady to pigs through the medium of the contents of the stomachs of cattle have repeatedly come under the notice of veterinarians; particularly has my attention been directed to this mode of dissemination by Mr. Welsby, West Derby; by Professor M'Call, and by the late Professor Fordie. The disease is also spread widely by animals in whose feet its lesions are localised, and by the feet of man, of birds, and of ground game. The general circumstances which favour the development and propagation of Eczema do not differ, in any material degree, from those which are in operation in the case of Zymotic Pleuro-Pneumonia.

Vagaries in the spread of Eczema Epizootica are not so marked as in the case of Pleuro-Pneumonia or Rinderpest; nevertheless, we occasionally meet with some remarkable ones, such as animals being surrounded on every side by diseased herds without themselves becoming affected in the slightest degree, and even some of those who are in close and daily cohabitation with diseased animals entirely escape the infection. Its vagaries are most observable in the peculiar manner in which, in different seasons, it singles out as its victims the different species of animals.

SYMPTOMS AND COURSE.

The Symptoms of Eczema Epizootica must be divided into General, or Constitutional, and Local.

THE PREMONITORY CONSTITUTIONAL SYMPTOMS are identical with those of other zymotic diseases, and are best observed when animals are at grass, and the weather is at all cold. These are—isolation, usually very marked; arched back in cattle; tucked-up abdomen; muscular twitchings or shiverings, more or less severe; erection of hair, the skin being hot and dry; and stiffness of gait, which is most pronounced when the feet—and particularly so if three or the whole—are affected. In all animals endeavours are made to seek shelter and warmth,—pigs burying themselves under their bedding, and cattle and sheep crouching in hollows or under hedges, walls, &c.

The bowels are usually a little constipated; the urine is sometimes scanty, high-coloured, and laden with solids, especially as the disease advances; at other times it is profuse and limpid.

There may or may not be mucous discharge from the eyes and nose, with increased lachrymal secretion from the former. In young animals, exposed to inclement weather, such discharge is very constant, and in the course of a few days that from the eyes forms a yellow accretion at the inner canthus of the lids and down the sides of the face, a similar accretion being formed, by the nasal discharge, round the edges of the nostrils. Under exposure, too, an irritable bronchitic cough is present; it is produced

by localisation of the lesions in the bronchial mucous membrane. The pulse and respiration may not be much disturbed, but there is always elevation of the temperature to the extent of 2° or 3° F.; this is indeed the best and most unerring guide to the existence of the disease, when other symptoms are but very slightly developed.

In nursing animals there may be a little indifference shown towards the offspring. In all animals the appetite may be indifferent; as a rule, it is not unless invasion is rapid and the fever high, or the stomach and bowels affected; indeed, in ordinary cases, a desire for food is evinced, even though the lesions in the mouth are extensive. In ruminants, rumination is performed naturally, unless the mouth is much affected, in which case difficulty in remastication is marked.

In milch animals interference with the lacteal secretion will be largely regulated by the localisation or non-localisation of the lesions in the udder; in the former case, not only is the quantity of milk diminished, but its characters are altered; in the latter, the diminution will depend upon the amount of fever and the intensity of the other local lesions. In the great majority of cases more or less decrease, for a few days, in the quantity of milk, is observed, and in severe cases it is often totally suppressed.

The alterations observed in the character of the milk during the course of the disease are—lowered specific gravity; diminution in the quantity of fat; tendency to rapid decomposition, especially in the most virulent forms of the disease, and when the udder is involved; aggregation of the globules; and the formation of granular cells, resembling colostrum corpuscles, as shown in Fig. 6, Plate XI.

When vesicles are formed on the mucous membrane of the galactophorous ducts, on the teats, or around their external orifices,—granular debris, bacteria, pus, granule, and epithelial cells, will be all present—more or less abundantly—in the milk, as are also, in some cases, lymph casts, and red blood corpuscles.

The milk, under any circumstances, possesses virulent properties. These are always, of course, much intensified under the above-mentioned condition.

The virulent effects are exerted to the greatest extent on the animal's own offspring; but even man does not always escape its deleterious influence. I have already referred to the effects of the milk on young animals; in man, cases are on record of the transmission of the disease through the medium of the milk, while other effects—as ephemeral fever, diarrhœa, and vomition—have, in some instances, been noticed.

The addition of a little salicylic acid to suspected milk, or the exposure of it to a moderate degree of heat—absolute boiling being most certain—and allowing it to cool before consumption; deprive it of injurious properties.

As the disease advances the phenomena above enumerated increase in intensity and continue to do so until the climax—which may be calculated at from the third to the seventh day—is reached, after which they gradually subside.

In addition, vesicular and pustular eruptions may appear on the skin of different

parts of the body; they are most often seen in the pig, and the primary may be succeeded by secondary and even tertiary crops.

Jaundice (*icterus*), as shown by yellowness of the skin, is a very frequent concomitant; it is most observable in those parts which are naturally devoid of hair, and more in some breeds than others—Herefords particularly.

Desquamation of the cuticle is an invariable accompaniment of convalescence, the skin being covered with abundant bran-like scales of a yellow colour; it is also extremely irritable (*pruritic*), animals rubbing against prominent objects vigorously.

If the lesions are localised in the gastro-intestinal mucous membranes, colicy pains —sometimes very violent—are induced, and (as first pointed out to me by my friend Mr. William Good, of Ludlow), under these circumstances, the mouth lesions are usually well developed.

In sheep, if the feet are much affected and the weather hot, the breathing becomes much accelerated—panting; and as the febrile action increases the pulse becomes very rapid and the temperature rises very high, up to 105-6°.

In malignant cases terminating in death, the constitutional symptoms are extremely aggravated; in cattle and sheep there is moaning and gnashing of teeth; in all animals great vital depression, lopping of the ears, total suspension of the natural functions, diarrhœa—often fœtid—foul breath, and weak, irritable pulse, are observed.

If animals survive sufficiently long the hair and wool fall off or are easily removed from the follicles, while ulceration of the eyelids, cornea, and nostrils, with profuse muco-purulent discharge from the eyes and nose, is occasionally present; emaciation is rapid and weakness extreme. Death is produced by coma as a result of blood depravation, or by asthenia consequent upon exhaustion.

Abortion in pregnant cows is very apt to take place in the latter stages of this disease when it assumes malignant characters.

THE LOCAL LESIONS are, so far as the skin is concerned, usually seen in parts devoid of hair, or where it is delicate, as the feet, the mouth, the udder, and in some cases the vagina of the female and the sheath of the male. They will be considered in the order laid down.

In all animals the earliest pedal sign is lameness—more or less sudden and severe—with uneasy movements of the limbs; and in cattle, frequent shaking of the affected foot or feet, as though a desire existed to rid the part of an irritant; if both fore feet are affected the hind legs are thrust somewhat forward in progression (as in laminitis in the horse), if all the feet, the limbs are drawn together under the body, the back arched, and the movements generally stiff and painful; in sheep and pigs a tendency to walk on the toes is evinced—the lameness is materially aggravated if the pasture is hard and dry or, contrariwise, muddy and stony; it is also aggravated on stubbles and hard roads or streets.

Sheep kneel, when the fore feet are most affected, in order to graze, and both sheep

and cattle drag themselves about, or raise themselves on to the sternum, for this purpose, when all the feet are involved; lying persistently in the intervals, showing great disinclination to move, and if forced to do so, tottering a few steps, and resuming the recumbent position with all possible speed. Pigs and sheep—especially the former—endeavour to walk on the toes; while cattle, if at pasture, show a great inclination, in the earlier stages, and when the lameness is not extreme, to stand in water, ponds, streams, or ditches, preference being given to those having muddy or clay bottoms.

If the affected foot is manipulated, the coronary band will be found more or less swollen and hot; and in white-skinned animals, the skin is increased in vascularity.

In the course of a few hours (no definite time can be laid down) subsequently to the advent of the lameness, the vesicular eruptions characteristic of the affection appear.

In the Ox the vesicles are located either in the front or the posterior part of the interdigital space, or in both positions, and in some instances around the base of the supernumerary digits (Fig. 12, *a*, *b*, Plate IV.) The cuticle in the two first-mentioned positions, being somewhat resistant (and the skin hairless), is raised, in the form of blanched, rounded, or pyramidal, elastic, or fluctuating—depending upon the amount of fluid—bladder-like elevations (vesicles), varying in size from a hazelnut to a walnut.

If allowed to remain undisturbed, the vesicles burst in from three or four to about twelve hours, and discharge a limpid, colourless, or pale-straw coloured fluid; the jagged edges of the lacerated epidermis become retracted and slightly everted, and form an irregularly-raised white boundary around the margin of the resulting sore (Fig. 12, *c*, Plate IV.) Frequently the vesicles extend inwards to the skin of the interdigital space, and in the case of anterior and posterior vesicles, become continuous (confluent); in a few cases, an independent vesicle forms on the interdigital skin, and, subsequently, continuity is established between it and those formed anteriorly and posteriorly respectively.

The cutaneous structure which is exposed by the eruption of the vesicle is of an intensely scarlet (hyperæmic) colour (Fig. 12, *b*, Plate IV.), this colour being more marked in the coronary than in the interdigital vesicle.

The exposed surfaces become rapidly covered by an abundant serum, or lymph exudate, which inspissates and forms a temporary covering, protecting them from the irritation of the atmosphere,—the hyperæmia disappearing as the lymph coagulates, and the epidermis becomes restored.

Gradually the lacerated marginal epidermis is cast off, and the eroded skin becomes covered by new cuticle, and unless the destructive process involves the true skin—which is rarely the case if the part has not been irritated or destroyed by caustic applications—no granulations are formed; neither is there any subsequent cicatrix, though the new epidermis is much smoother and finer for a time than that of the surrounding parts.

In Cattle, vesicles do not form so often on the coronary region as in sheep; neither, as a rule, does extension take place in this direction; if it should do so, however, the hoof separates from the coronet, and gradually exfoliates from above downwards.

Immediate exuviation of the hoof is not common in cattle, in fact, it is only seen in very aggravated cases, where the animals have been travelled for some distance during the active processes, or where much stress has been put on the feet in railway or sea travelling. The shedding of the hoof under these circumstances is due to extension of the inflammatory process to the laminæ (*laminitis*), the actual separation being produced by the pressure of the effused serum.

The exposed laminæ may be very red (*hyperæmic*), or, if effusion of serum has been rapid and abundant, pale in colour and macerated in appearance, rapidly, however, becoming intensely scarlet.

It need scarcely be observed that immediate exuviation renders an animal helpless and incapable of progression until new horn is formed—a comparatively slow process.

Foot Lesions in the Sheep are more constant and more severe than in the ox. They are seldom confined to one or two feet—more usually affecting three or the whole, the number affected regulating the amount of suffering, and, to some extent, the constitutional derangement.

The position of the vesicles differs materially in the sheep from that of the ox; probably the most constant site is at the heels, or the anterior part of one or both digits (1, Fig. 9, Plate III.; and *a*, Fig. 11, Plate III.) They may appear primarily around the coronet or in the interdigital space, and in either case may become confluent—occasionally, when located at the anterior part of the foot, extending into the orifice (*b*, Fig. 11, Plate III.) of the biflex canal.

The course of the vesicles is similar to that which has been detailed as occurring in the ox; when fully formed, a sense of fluctuation is imparted to the finger on manipulation, the hair is erect, and the coronets are hot. After eruption, or if the vesicle be violently burst, the subjacent coronary skin is seen to be intensely scarlet, as shown in Figs. 9-10, Plate III., the exposed structures quickly becoming bathed in pus, which rapidly inspissates, and imparts to them a brownish colour.

Immediate exuviation is far more common in the sheep than the ox, but, as in the latter animal, it more frequently takes place when they have been travelled by road, or conveyed long distances by rail or sea. Whether it be immediate or subsequent, separation of the hoof *always* goes on from above downwards.

Secondary vesication, or relapse, is not at all an unusual occurrence in the sheep; the vesicles, however, are smaller, more frequently single than multiple, show little tendency to extend, are developed in only one or two feet, and are usually situated at the anterior part of the digit (*c*, Fig. 4, Plate XI.)

Separation of the hoof is much more circumscribed, indeed it is usually limited to

the immediate area of the vesicle (*c.* Fig. 4, Plate XI.), and not infrequently a small granulation, as shown at *b*, Fig. 4, Plate XI., forms in the seat of the vesicle.

I have already indicated that exuviation takes place from above downwards. As the old hoof separates a new one forms beneath and above it—*i.e.*, continuous with the coronary band—a distinct line of demarcation (*a*, Fig. 4, Plate XI.) between the old and the new being formed, the lower border of the latter being overlapped by the upper edge of the former. The old hoof often clings to the new until the last vestige is worn away, and the extent of separation always serves as a good guide to the length of time which has elapsed from the date of attack. We may calculate that the time required for the growth of a new hoof is, on the average, from three to four months, and consequently the length of the old hoof enables us to form a fair estimate as to the date of the attack.

The Lesions in the Foot of the Pig are, as a rule, primarily located around the coronary band—*i.e.*, at the junction of the skin with the hoof; they may, however develop, either primarily or secondarily, in the interdigital space, and either one or the whole of the feet, as in the sheep, may be involved. The foot lesions in this animal run a rapid course and are often very severe.

Immediate exuviation is seen perhaps more frequently than in other animals, and it is not at all an uncommon occurrence for the deck of a vessel or the floor of a railway truck, in which they have been conveyed some distance during the active stages of the disease, to be strewn with the cast-off hoofs.

It also very often happens that the hoof slips off in the hand when the animals are caught by the leg.

The foot lesions in the pig do not call for any further comment, as their course is, on the whole, similar to that of the vesicles in the feet of the sheep.

In Birds the vesicles are located between the claws, and they occupy the same position in the dog, cat, and allied animals. The character of the vesicle will, of course, be materially altered in the fowl owing to the difference in the epidermis as compared with that of animals. Shedding of the nails sometimes takes place in these creatures and thus assists in establishing the identity of the malady.

THE MOUTH.—The advent of mouth lesions in cattle is marked by smacking of the lips, or rather the mouth; by dribbling of saliva, and by partial or total inability to masticate.

Smacking of the mouth is no doubt due to the peculiar sensation imparted, by the virus contained in the saliva, to the gustatory bodies of the tongue. It is heard sometimes several hours prior to the development of the vesicles, and is often the first sign which attracts the notice of the attendant or the professional man; it usually ceases after the vesicles have burst, and does not again recur.

Dribbling of saliva is due to the abnormal increase of that fluid, owing to the stimulation of the salivary glands by the excreted virus, these glands constituting one

of the channels through which nature endeavours to eliminate the poison of the disease from the system.

The saliva at the outset is normal in character and consistence, but it quickly becomes viscid, the viscosity increasing to such an extent as to render it very tenacious, and, as a consequence, to cause it to hang in ropy-like strings from the sides of the mouth. Micrococci and fungoid bodies have been detected in this secretion, and, as the disease advances it becomes largely intermixed with mucus, epithelial cells, and (after the eruption of the vesicles) with shreds of the destroyed epithelial lining of the mouth.

The degree of difficulty in prehension and mastication will depend upon the extent of vesication, its stage, and position; and the character of the food upon which the animal is feeding. If the vesicles are situated on the dental pad and tongue, or are large and numerous on the latter organ, difficulty in both actions is extreme, and as the greatest amount of pain exists at the maturation of the vesicles and immediately after their laceration, it follows that difficulty in prehension and mastication is most marked at these periods. The greatest difficulty is experienced in grazing, and in attempting to masticate roots, and if an animal is watched during an attempt to eat the latter it will be observed to twist the jaws uneasily about, elevate the nose with the object of passing the root to the posterior part of the mouth, and, after several ineffectual efforts to comminute it, will allow it to fall from the mouth thoroughly coated with viscid saliva and mucus—all food partially masticated by affected animals exhibits the same conditions.

The mouth may precede or succeed the other local lesions, and, as before remarked, they are always most aggravated if the disease is concentrated in the alimentary tract.

The mouth vesicles vary in size and character according to the part of the buccal membrane in which they are located. In point of time the dental pad, in point of size the tongue, takes precedence. The position of the vesicles on the dental pad is, in the great majority of cases, at the angles of that structure (Fig. 14, Plate IV.), but a vesicle may appear independently, or concurrently, in its centre, and if more than one vesicle exists they may—though at the outset perfectly discrete—coalesce, the resulting ulcers, in this case, being of course continuous.

The pad vesicles are easily detected by the experienced practitioner, as the normally flattened and firm appearance and feel of the pad are replaced by a bulging or elevation of the epithelium, which, on pressure, imparts a sense of fluctuation to the finger, and on puncture or forcible rupture allows of the exit of a pale yellow lymphoid fluid. The bulging sometimes extends to the loose fold of skin which connects the short upper lip with the pad.

There is no formation of areolæ around the base of the vesicles, neither is the buccal membrane—as would naturally be presumed—increased in colour in this situation; on the contrary, it is blanched, and contrasts markedly with the surrounding healthy parts.

On the tongue the vesicles usually occupy one or more of the following positions —viz., the tip, the borders, or the thick part of the upper surface (dorsum); primary vesication seldom, if ever, takes places in the membrane covering the lower (venter) surface of this organ.

The primary vesicles are here much larger than elsewhere; they vary in number from one to four or five—seldom more—and are, owing to the great thickness of the epithelium, very resistant; from the latter cause, and from the fact that the buccal membrane is often discoloured by grass or other succulent vegetable matter, the vesicles may easily escape detection by superficial examiners.

The vesicles present much the same characters on the tongue as on the pad, with the exception that the raised epithelium in the former position bristles with the horny coverings of the filiform papillæ; two or more may coalesce and form one very large vesicle.

The inside of the cheek, amongst the large conical papillæ, is one of the sites on which the development of the vesicles is least frequent, and in this position they are (on account of the papillæ) small; the epithelium being here more delicate than on the pad or tongue, the reddened condition of the mucous membrane is more discernible, and the resistance of the vesicles considerably diminished.

On the lower lip vesication, in ordinary cases, is comparatively infrequent, but is more constant in the malignant types of the disease; the vesicles are much smaller, in fact often escape detection, and are rapidly followed by an aphthous deposit which has a blanched appearance, and, from its very slight power of cohesion, readily cracks and allows of the intensely reddened (hyperæmic) membrane being seen in the chasms. It is in this position that the eruptions are so liable to be confounded with those of rinderpest, but as a rule they are more extensive and more pronounced than in that disease, and are not situated in such close apposition to the gum.

On the skin outside the lips vesicles are developed with comparative rarity; they are more frequently secondary than primary, are small, and succeeded by pustules and scabs. In the course of a few hours after full development the buccal vesicles burst or are forcibly ruptured by the movements of the tongue or by attempts at prehension or mastication; it will be readily understood that the character of the food—*i.e.*, as to whether it is coarse and hard or soft and fine—will materially influence the period of deflorescence of the vesicles.

After rupture the epithelium of the large vesicles on the pad or tongue presents a very characteristic appearance. Owing to its cohesive properties it does not, unless detached by violence, fall off immediately after rupture, but hangs in loose skin-like folds, which gradually become detached by the movements of the tongue, the circumferential portion, contracting somewhat upon itself, forming a thickened raised border around the original site of the vesicle (Fig. 15, *b*, Plate IV.)

The exposed mucous membrane is at first of a bright scarlet colour, hyperæmic,

(Fig. 15, *a*, Plate IV.), but the colour quickly diminishes in intensity owing to the exposed surfaces becoming covered by new epithelium, which assumes a brownish hue; and, from deficiency in cohesive properties, forms an aphthous crust; if the latter is scraped off, the hyperæmia will still be found to exist, as seen in Fig 13, Plate IV.

In the course of a few days the epithelium will be so far restored as to form a perfect coating over the inflamed tissues; it is, however, much smoother, more delicate, devoid of papillæ, and depressed below the level of the surrounding parts (Fig. 15, *c*, Plate IV.), but unless ulceration has succeeded the bursting of the vesicle, no cicatrix is formed. Suppuration is seldom seen in connection with the buccal vesicles, as although pus may be formed, it is immediately removed by the saliva and by the movements of the tongue and lips during prehension and mastication.

The buccal eruptions do not, as a rule, show much tendency to appear in successive crops, though secondary, or even tertiary, crops are occasionally seen. The secondary vesicles frequently present totally different characters from the primary ones; they are more circumscribed, involve the mucous membrane to a greater depth, usually situated on the borders of the tongue, are surrounded by a tolerably deep and extensive inflammatory area, and are succeeded by ulceration; the ulcers presenting, in fact, much the same characteristics as those which are formed in the mucous membrane of the stomach: they are usually oval or circular in shape, and segregated from the surrounding tissues by a livid line of demarcation; and, as shown in Fig. 8, Plate XI., necrosis commences in the centre—pitting being more frequent than after the primary vesicles.

Mouth Lesions in the Sheep are far less frequent and less extensive than in the ox; thus out of a flock of 194 merinos, thirty or forty of which presented well-marked foot lesions, only two were found affected in the mouth.

The vesicles in this animal may form either on the tongue or on the pad; in the former position they are more frequently than otherwise circular in shape and very circumscribed; in the latter they present all the characteristics common to the pad vesicles in cattle, but owing to the small size of the pad in sheep the vesicle more frequently involves its whole width than is the case in the ox.

External lip lesions are sometimes seen in the sheep.

In the Pig the vesicles are more often situated on the snout and around the free borders of the lips than on the buccal membrane.

In Birds the eruption is situated in the mouth, around the nostrils, and on the crest.

In the other domestic animals buccal vesication follows much the same course as in the ox, but owing to the more delicate character of the mucous membrane the vesicles are smaller and more easily ruptured, and the congested condition of their bases is more visible and pronounced. The horse, cat, and dog suffer much more than the ox—croupal exudations sometimes forming on the mucous membrane, and the lesions occasionally extending to the pharynx.

THE UDDER.—When the lesions are located in this organ in milch animals, the quantity of milk is found to decrease, the udder—or one or more quarters—becomes swollen, hot, and sensitive; but the extent of these conditions is largely regulated by the state of activity of the organ itself—being always greatest shortly after parturition.

In the Cow, the vesicles, which usually appear in a few hours after the premonitory signs above mentioned, are formed most frequently around the base, on the body, and around the apex of the teat, but they may be developed in any part of the udder: at the outset they are discrete, but from various causes, as irritation in rising, the attrition of milking, or the sucking of the young, they frequently become confluent; they vary in size, from a threepenny piece upwards, and in delicate white-skinned animals show a somewhat inflamed base. The period of vesication will vary from one or two to about thirty-six hours, depending upon the amount of pressure and friction to which the vesicles are exposed: if they do not burst, the fluid contents become absorbed, and the separated epidermis subsequently desquamates.

The exposed dermis after rupture of the vesicles is intensely hyperæmic, but if the parts are undisturbed it quickly becomes covered by inspissated pus, coagulated lymph, and epidermic cells—the hair, when present, assisting in forming a coherent brown-coloured scab which has usually irregular edges and varies in thickness. If the parts are much irritated ulcerative action is extensive, the wounds become confluent, as seen at *d*, Fig. 16, Plate IV., and frequently extend over the end of the teat and even into the duct, blocking it up and causing retention of milk.

Forcible and premature removal of the scab reveals the hyperæmic condition of the dermis beneath (Fig. 16, *d*, Plate IV.), but if the former is left undisturbed the latter quickly becomes covered with new cuticle, and the scab is cast off in the course of a few days. The epidermis on the site of the vesicle is more delicate in texture than that of the surrounding parts, but in the course of time it regains its normal condition: cicatrices are only formed when the ulcerative process has extended through the cutis.

The skin around the seat of ulceration remains somewhat corrugated (Fig. 16, *a*, Plate IV.), and thickened for a short period after convalescence.

Udder lesions are much more rare in the sheep than in the cow. They are more frequent in the pig than the sheep, and extend more or less over the whole of the glands.

Secondary mammary vesication is sometimes seen in each of the animals mentioned, the vesicles being smaller and more discrete than those of the primary eruption.

The eruptions on the external generative organs are much less constant than those of the mouth, feet, or udder; in fact, in many outbreaks, they are not seen at all, while in others they are so slight as scarcely to attract attention.

If located in the vagina and vulva the mucous membrane becomes swollen, red, and hot; and viscid mucus is discharged, which adheres to the hair on the lower commissure of the vulva. The vesicles are smaller here than elsewhere, and either run on to

pustules or give place to aphthous deposits. Urination gives rise to straining, owing to the irritating effects of the urine upon the eroded membrane.

In the male the eruption is usually situated on the anterior part of the sheath, internally and externally, and may involve the extremity of the penis. The vesicles here, as in the vagina, are small, and the irritation which accompanies their formation frequently produces balanitis, causing a free discharge of pus, which mats together the hair at the end of the sheath.

Eruptions are stated by some authorities to take place occasionally around the base of the horn, which may become loose and fall off. I have never seen such an occurrence.

DIAGNOSIS.

The diagnosis of Eczema Epizootica is not very difficult to the experienced practitioner, but under certain circumstances it is—by inexperienced persons, and by a superficial and careless examination only having been made—easily confounded with a number of other diseases. Those for which it is most frequently mistaken are Aphtha, Blain, Ranunculus Poisoning, or poisoning by caustic chemical agents; Rinderpest, Cow-Pox, Foot-Rot, and occasionally, Sheep-Pox and Hog Cholera.

Aphtha (thrush) seldom occurs as an epizootic, usually attacks young animals only, the buccal vesicles are smaller, more numerous, and appear in successive crops—the feet are never affected.

Blain (gloss-anthrax, stinge) is a sporadic affection, attacks cattle only, the vaginal vesicles are large and few in number, and there is considerable swelling (œdema) of the eyelids, head, vulva, and anus. Foot lesions are never seen, though vesicles may develop on the udder. The disease is evanescent and non-infectious.

In *Ranunculus Poisoning* the mouth eruptions partake more of the character of ulcers from the outset, they are few in number, and there is entire absence of foot, vaginal, or udder lesions; there is frequently, however, much discharge of saliva, grinding of teeth, and smacking of the lips; a number of animals, too, may be poisoned simultaneously, and more or less fever is always present.

In *Poisoning by Corrosive Agents* stomatitis is more extensive than in Eczema, the buccal membrane is uniformly reddened, swollen, hot, tender, and frequently livid; the discharge of saliva is profuse, and the epithelium peels off in large patches.

The lesions are confined entirely to the mouth, and, as a rule, the number of animals affected is small.

Rinderpest.—Foot-and-Mouth Disease is more likely to be confounded with this malady when it assumes a malignant type; when eruptions take place in the vagina, sheath, and lower lip; when diarrhœa and catarrh are present, and, above all, when enteric fever is a complication. The distinctive characteristics of Rinderpest, if

carefully studied, will always suffice to prevent mistakes in this direction. Under any circumstances, Rinderpest has no local manifestations in the feet.

Cow-Pox.—This disease, so far as the general febrile condition of the system and the udder lesions are concerned, may readily be confounded with Foot-and-Mouth Disease; it seldom assumes epizootic dimensions, is peculiar to the cow, is never accompanied by foot, mouth, or vaginal lesions, and the character of the udder eruption is totally different.

The mammary vesicles of Cow-Pox are as often developed on the gland itself as on the teats, they are umbilicated (depressed) in the centre, and in desiccation the scab presents different characters to those of Eczema, as shown in Fig. 17, Plate IV.; the eschar is usually circular, more circumscribed, thicker, composed largely of hair, clings tenaciously to the skin by a well-defined pedicle, and is followed by temporary or, in bad cases, permanent pitting.

The cutis, on the site of the vesicles, is less hyperæmic, though the swelling, redness, and tenderness of the skin may be greater; and as a rule the latter is more corrugated.

With *Foot-Rot* and *Foot-Sore* Eczema Epizootica is probably—either in its earlier or later stages—more frequently confounded, in the sheep, than with any other affection.

The circumstances in which mistakes in this direction are more likely to arise are —1stly, When animals have been changed from fine grass lands to turnip or other fields, the condition of which is highly favourable to the development of Foot-Rot, and, as a consequence, a great percentage of animals contract the disease in a very short space of time, and many of them in several feet simultaneously.

2ndly, When they have been driven a long journey over rough or hill roads, or over lava beds, producing wearing away of the toe by attrition, denudation of the laminæ, insinuation of dirt and laminitis, with the burrowing of pus and its escape at the anterior part of the coronet. Both cattle and sheep are very liable to suffer from inflammation of the coronet (coronitis) when travelled under these conditions, and if the road journey is succeeded by a rough sea voyage, or if the animals are conveyed some distance by rail; the state of the feet resembles very closely that of Foot-and-Mouth Disease, as the coronet is hot, swollen, tender, and red, the interdigital skin lacerated and inflamed, and the animals show great signs of suffering, with more or less fever, especially if extremes of weather, as great heat or cold, or cold and wet, respectively, prevail.

3rdly, When sheep, especially lambs, are suffering from the so-called *dew-scald*, or that form of interdigital inflammation which is produced—often in tolerably large numbers of animals simultaneously, and in a comparatively short space of time—by hoar-frost or cold dew on tough or long grass.

In these cases the inflammation is superficial, affects the interdigital skin only, and does not extend round the coronet, though it may involve the bulb of the heel posteriorly, and extend into the biflex canal anteriorly; no true vesicles, however, are

formed, there is no loss of hoof, fever is entirely absent, and the affection is non-contagious.

In cattle the disease known as *Foul*, especially if attacking several feet of three or four different animals at the same time, is occasionally mistaken for the foot lesions of Eczema, but in this disease there is a total absence of mouth and udder lesions, and—except in extreme cases, or where other and independent causes exist for its production—of fever; the odour of the discharges in foul is also very characteristic.

The distinguishing differences between Foot-Rot and Foot-and-Mouth Disease are numerous, but they require to be closely studied in order to guard against mistakes, particularly where a hurried examination under unfavourable conditions is alone possible.

The main distinguishing features of the two affections are as follows:—Foot-Rot is not generally contagious, and under no circumstances is it infectious; and while certain forms of it are thought to be contagious by some authorities, there are others who fail to attribute contagious properties to any of its forms, and I must confess that I am not, as yet, fully convinced that Foot-Rot is under any conditions—except direct introduction of the purulent fluids into wounds on the coronet or interdigital skin—contagious.

Foot-Rot is not associated with fever, except from some independent and coexistent cause, the heat and swelling of the coronet are less than in Eczema, the appetite is not interfered with, and in milch ewes the diminution in the quantity of milk is only very slight.

Foot-Rot, except under the circumstances before detailed, does not attack three or four feet or a great number of animals suddenly, and its invasion is much slower than that of the foot lesions of Eczema.

Foot-Rot is not accompanied by mouth, skin, or udder lesions, though slight scratches and pustules may be present in these positions from many causes, as, for example in the case of the mouth, thorns, glass, or other sharp objects in the food, or injuries inflicted by the molar teeth; the non-specific character of these lesions is alone sufficient to distinguish them.

Foot-Rot commences in the great majority of cases in the interdigital skin, the inner surface of the wall, or the toe.

The changes in the interdigital skin are, from the outset, of an ulcerative not of a vesicular character, and extension goes on laterally, so that by-and-by the hoof commences to separate towards the posterior and anterior part of the digit, exposing the laminæ in these directions, and if the ulcerative action continues extending outwards and finally upwards, the separated hoof clings tenaciously to the skin of the coronet for a considerable period, and the reproduction of horn goes on underneath it.

Foot-Rot, too, is frequently accompanied by extensive granulation (so-called fungoid growths) of the laminæ. This is well shown in Fig. 3, Plate XI.

When Foot-Rot begins at the toe from attrition and insinuation of dirt, the inflammation, as already indicated, extends upwards, and the fluids collecting underneath the epidermis at the junction of hair and hoof give rise to the production of an apparent vesicle which on being lanced or forcibly ruptured gives exit to a sanio-purulent fluid, but not to the limpid fluid characteristic of foot-and-mouth vesicles.

In very wet pastures moisture follows, by capillary attraction, the course of the laminæ, and elevates the epidermis at the coronet in the same way as does pus, but in either case the separation of the hoof, if the inflammation and suppuration goes on, extends laterally.

In Foot-and-Mouth Disease, separation—either as the result of primary or secondary vesication—always goes on uniformly from above downwards (Fig. 4, Plate XI.), the new hoof commencing to form at the coronet, and in its descent displacing the old one in a downward direction, the latter clinging (even to the last vestige) tenaciously to the former.

If the old hoof is prematurely or violently detached, and the new horn and laminæ exposed to dirt and moisture, extensive inflammation of the latter is established, and a troublesome and painful form of Foot-Rot induced.

Sheep-Pox can only be mistaken for Foot-and-Mouth Disease when the eruption is in the vesicular stage, and when sheep are, coincidently, very lame from Foot-Rot.

Body eruptions in Eczema are rarely seen in the sheep, and when developed are not preceded by redness (*Roseola*) or papulæ as in Sheep-Pox; in the latter disease coronal vesication does not take place.

Hog Cholera.—A wrong diagnosis in this direction is only likely to be made when the redness or lividity of the skin has passed away; when secondary vesico-pustular eruptions are developed, and when stiffness and cramp of the muscles are extreme.

The gastro-intestinal ulcers—as will be shown in speaking of the pathological anatomy of the disease—present certain common peculiarities.

Finally, in diagnosing Foot-and-Mouth Disease, it must not be forgotten that cattle are occasionally the subjects of localised pustular eruption, and various forms of scratches and incisions, on the skin of the udder and teats; and that the excoriations of the upper lip and dental pad produced by the incisor teeth when associated with simple cold or catarrh are apt to mislead. A careful estimate of the concomitant circumstances will always prevent wrong conclusions under these conditions.

PROGNOSIS.

The favourable termination of an ordinary attack of Foot-and-Mouth Disease may always be prognosticated; but when the elimination of the poison by the usual channels has been suppressed, when the fever runs high and is accompanied by manifestations of extensive internal complications, and when the disease is of an unusually malignant type, a guarded opinion as to the probable termination should be given.

PATHOLOGICAL ANATOMY.

In considering this part of the subject we will first glance at the condition of the carcase. If an animal, in ordinary attacks, is slaughtered—as many thousands are—during the height of the fever, little or no alteration, either texturally or otherwise, can be detected in the condition of the flesh or fat, and we have no evidence that it is, when ingested by man, in the slightest degree deleterious or unwholesome. In malignant cases the carcase may present three very opposite conditions: *i.e.*, it may be tolerably dry but of a very dark red, or of a mahogany hue—flabby and soapy to the feel; it may be moist throughout, from albuminous effusion, and of a magenta hue,—these conditions are identical with those which I have described as existing—under different circumstances—in Zy. p. p.; or extensive watery (serous) effusion, combined with exudation of lymph and extravasation of blood, may exist in different parts of the body, and in the various cavities and organs. Either of these conditions, when present, warrant the unhesitating condemnation of the flesh—on the part of the inspecting officer—as human food.

The state of any milk which may remain in the udder in milch animals will be similar to that which has been drawn during life, and need not be further alluded to.

In pregnant animals no material change will be detected, in ordinary cases, either in the uterine membranes or the fœtus, but in malignant cases effusions and hæmorrhages exist—more or less extensively—in the tissues of both fœtus and membranes.

On examining the parts of the skin or mucous membranes in which vesicles exist it will be found on section through their structures that the lesion is superficial, though, in each case there may be a little subjacent and circumferential effusion or exudation.

In malignant cases ulceration is oft-times very extensive and deep, involving not only the structures of the skin and mucous membrane but the underlying tissues also.

The substance of the tongue is usually much more flaccid than normal, and if the papillæ are carefully examined it will be found that in the earlier stages of vesication they are much congested, subsequently becoming atrophied and shrivelled; so much

is this the case, that in the healing process the surface of the tongue, in the involved parts, is smooth and glistening.

In addition to these external *post-mortem* lesions very important changes will be discovered in the bronchial, gastric, and intestinal mucous membranes.

On the bronchial membrane numerous ulcers may be developed; they are usually small—florid and healthy in appearance.

The First Stomach (*Rumen*).—The mucous membrane of this organ frequently presents characteristic lesions. These consist in the development of large oval spots of a dusky-red colour, on the pillars or the smoother parts of the membrane adjacent thereto, which are ultimately succeeded by the formation of ulcers. The spots are comparatively large in size (reaching, in some cases, a measurement of one by one and a-half inches), vary in number from three or four to twenty; frequently become confluent, and, at the outset, are not raised above the level of the membrane. In the course of a few days molecular death of the mucous membrane takes place, and simultaneously with this change there is rapid proliferation and degeneration of the epithelium of the immediately surrounding parts.

The involved portions of membrane become thickened, and consequently the surface of the ulcer is raised considerably above the adjacent parts. Owing to degeneration commencing in the centre, solution of continuity of the destroyed tissue is first noticed here.

The ulcers, which are of a brown colour, become surrounded by a red or purple line of demarcation, and in the healing process a cicatrix—owing to the great loss of substance—is formed.

If a section is made through the involved structures it will be seen, as shown in Fig. 18, Plate IV., that the diseased action does not extend deeper than the mucous membrane, but if the dead tissue is scraped off, the muscular fibres beneath are observed to be somewhat widely separated. Perforation of the rumen is never seen.

The ulcers of Foot-and-Mouth Disease in this situation present some characters analogous to the intestinal ulcers of Hog Cholera, but the analogies do not extend beyond the fact that the substance of the ulcer in each case seems to be formed much in the same manner. Solution of continuity commences in the centre, both show a tendency to coalescence, and they are both raised above the surrounding healthy surface.

In Hog Cholera the thickened and raised condition of the membrane is preceded by loss of substance; the ulcer is usually circular, and the subjacent tissues become involved in the inflammatory process, leading to considerable thickening and subsequent puckering and constriction—see Figs. 7-9, Plate XI.

The Third Stomach (*Omasum*).—In some severe and protracted cases of Foot-and-Mouth Disease the folds of the omasum are bestudded with spots varying in size from a sixpenny piece to a shilling, more or less oval, quadrilateral, or irregular in shape,

with a dusky-red and finally brownish-coloured centre and a scarlet (*hyperæmic*) circumference—the scarlet colour extending through the substance of the folds. These spots resemble those which are seen in this viscus in Rinderpest (Fig. 26, Plate VII.), and in poisoning by the ranunculus; they are not pathognomonic, and are not often succeeded by sloughing.

The Fourth Stomach (*Abomasum*) also presents characteristic lesions which are more frequently developed in the antrum pyloris than over the general surface of the organ.

These lesions consist—1stly, Of a number of discrete, dusky-red coloured spots; oval or slightly irregular in shape, and about the size of a pea or a horse bean; they do not, at the outset, extend deeper than the mucous membrane, but after a time loss of integrity and sloughing result, followed by ulceration—the ulcer having jagged scarlet edges, and being about double the size of the original spot. In some respects these ulcers resemble those of Cattle Plague, but they are never associated with cracks —rhagas. 2ndly, On the general mucous surface of the stomach, patches (sometimes very extensive, at others, circumscribed) of congestion or extravasation, followed by erosion of the superficial portions of the membrane, are observed.

The Intestines.—The only lesions which have come under my observation in these organs are ulceration, similar to that described as existing in the abomasum; and more or less extensive inflammation (mucositis), congestion, and hæmorrhage.

TREATMENT.

The first thing to be thought of—at least in certain periods of the year—is to protect the affected animals against the injurious influences of wet and cold.

Exposure checks skin (cutaneous) elimination, determines the lesions to the internal mucous surfaces, retards recovery, and frequently leads to great losses by intensifying the results. In fine summer weather both cattle and sheep do better at pasture than in sheds or byres.

When the mouth is very sore, food requiring little mastication should be allowed. If the animals are grazing it is a wise plan to mow down the luxurious patches of grass, which are usually abundant in most pastures, two or three times a day; failing this, the animals should be allowed a judicious supply of freshly-mown young grass, clover, or other succulent vegetable matter. In the winter and spring a plentiful supply of mashes of various kinds—bran, oatmeal, linseed, malt or oil-cake, or a judicious admixture of these with steamed cut hay—must be provided.

There can be no objection to a little long hay if it is soft and good, but roots—as carrots, turnips, mangolds, or beets—except they are pulped, should be avoided as they are very apt to lead to choking, and produce much pain in the act of mastication.

In the case of the pig, boiled roots, meals, and milk form the best foods.

In all animals, when the disease is of a malignant type or protracted, stimulants—as beer, porter, and wine—should be judiciously administered, with such easily assimilable matter as oatmeal or linseed gruel, hay tea, raw eggs, glycerine, molasses, and milk.

Strict attention must be paid, under all circumstances, to general hygiene, and a plentiful supply of pure water must be provided.

Medicinally.—In all instances, where practicable, a gentle laxative—Epsom salts in preference—should be administered at the outset; excessive purgation is inadvisable, as it may lead to extensive intestinal disease.

In ordinary cases, nothing further than this—except the addition of some antiseptic saline, as nitrate of potash, chlorate of potash, sulphite or salicylate of soda or potash, to the drinking water—is required.

If the disease assumes an unusually severe type, and fever runs high, sedative medicines are called for, as aconite and tartar emetic—the latter being especially indicated if shivering is very marked; it may be beneficially combined with bitartrate of potash.

In all protracted cases, and where great weakness is manifested, tonics—as iron, gentian, cinchona, the mineral acids, and nux vomica, with such cordials as ginger, caraway, or aniseed—should be ordered.

In the case of very heavy cattle, bruising must be carefully guarded against by putting them into loose boxes (if possible), and bedding them with a thick layer of chaff, short straw, soft hay, saw-dust, or spent hops.

The young of all nursing dams should be removed, immediately any symptoms of the disease are manifested, and fed artificially until the disease has declined.

I am quite aware that in the case of the ewe this precaution is impracticable, but fortunately it is not so much demanded in this animal as it is in the cow and sow, owing to the fact that mammary lesions are less frequent and less severe.

Local Treatment.—Under no circumstances should the vesicles—either in the feet, mouth, or udder—be prematurely ruptured. I have heard the advice given by eminent veterinary surgeons to secure a piece of flannel, tow, or other material on a stick, and rub it backwards and forwards in the mouth till all the vesicles are broken. I need scarcely observe that this is both unnecessary and cruel—the vesicles will burst naturally (and without laceration of the underlying tissues) in due course, without exposing the exquisitely sensitive membrane or skin to the visitation of the atmosphere or of foreign matter before it has become ensheathed by lymph.

Mouth.—After the bursting of the vesicles in this situation all that is requisite is the application—by the aid of a soft sponge attached to a stick—of a solution (one to sixteen), twice or thrice a-day, of any of the following agents: viz., potassa chloras or nitratis, ferri sulphas, any of the sulphites—except lime—alum, borax, or salicylate of

soda. The addition of a little carbolic acid (five or ten drops to each ounce) dissolved in glycerine to either of the above solutions is beneficial. Some authorities object to the use of alum, considering that it is too harsh, but from an extensive acquaintance with this agent I have no hesitation in asserting that it is superior to any other application, as although it may cause a little temporary smarting, the subsequent relief is both rapid and marked.

If deep or unhealthy ulceration takes place, a few applications of such agents as nitrate of silver, solution of perchloride of iron, or dilute mineral acids will be required; but, as a rule, no stronger applications than those above enumerated are needed.

Udder.—The same solutions which I have recommended for the mouth may be used for the udder and teats; and, in order to protect them from the irritation of the atmosphere, and to favour rapid healing, powdered gum tragacanth or powdered resin may be freely scattered over the exposed surfaces, two or three times a-day.

Every precaution should be taken to prevent irritating the wounds in the operation of milking; in fact, when the teats are much involved, it is better to use syphons to withdraw the milk than run the risk of having subsequent, and sometimes deep ulceration, or extension of inflammation to the gland itself.

If scabs form around the points of the teats they must be softened by glycerine or cream, and the orifices be kept open by the occasional insertion of the syphon, previously dipping it in a little almond or olive oil. If the teats are allowed to become occluded the milk is retained, and, acting as an irritant, induces mammitis.

Feet.—The alum or sulphate of iron lotion answer admirably here; but if there is any tendency to unhealthy action, as indicated by fœtor or suppuration; carbolic, creasote, chloride of zinc, or salicylic applications should be substituted. If the inflammation has extended into the deeper structures, and exuberant granulations (proud flesh) are formed, a little caustic, dry alum, or a strong solution of sulphate or chloride of zinc may be applied with the object of reducing them.

When sheep are attacked in large numbers it would be impossible to dress the feet in each individual case; under these circumstances it is better to drive them every day through a shallow wooden trough, containing a solution of the agents recommended.

It is necessary to remove any loose horn or surrounding hair, to keep the feet perfectly clean, and if the sensitive structures are much exposed, or the hoofs cast, to envelope them in fine carbolised tow, or tow and tar, which may be secured in position by the aid of canvas bags. In speaking of feet symptoms, I made the remark that cattle showed a great tendency to stand in pits, ditches, or streams; there can be no possible objection to this, providing the water is not charged with decomposing organic matter; clay or salt-water are especially beneficial, and in any case there can be no doubt that the cold and moisture afford great relief, and moderate the inflammatory action. Of course, it would be unwise to allow animals to stand in water under these circumstances if the weather is cold and changeable. Poultices and fomentations in ordinary cases

are not necessary, but when the feet lesions are severe they are very beneficial, particularly in the case of heavy cattle—and if there is fœtor, yeast, charcoal, or malt cummins may be added.

Vagina and Sheath.—No applications are, as a rule, required to these parts; they should, however, be cleansed, and if it is thought desirable a little alum or other lotion may be applied twice a-day.

Eyes and Nostrils.—Unless the skin of these parts becomes excoriated, no attention is demanded; if there is excoriation a little alum or liquor plumbi lotion is all that is required.

RESULTS.

The Sequelæ of Foot-and-Mouth Disease, in some outbreaks, are productive of far more loss and suffering—especially in cattle—than the primary active processes. They may be divided into Local and General.

The Local Sequelæ are seen in the parts in which the lesions are primarily localised; and are most severe and destructive in the feet.

Feet Sequelæ are seen most frequently in fat, heavy cattle and sheep, in cases in which the lesions have been concentrated in these parts, where animals have been neglected, or dirt allowed to gain access to the exposed structures.

The inflammatory action extends not only to the deep layers of the skin of the interdigital space and the coronet, but to the subcutaneous tissues, the coronary substance, veins, ligaments, tendons, joints, and bones. Secondary inflammation also takes place in the sensitive sole and laminæ.

When the inflammation is extending, the skin of the coronet and pastern becomes very red (the redness being best seen in white-skinned animals), hot, swollen, tense, tender, and shining; the digits are widely separated; lameness is extreme, the animal is often, in fact, incapable of bearing its own weight, and usually lies persistently, not even rising for the purpose of grazing or performing the natural functions until it is forced to do so.

Sympathetic fever runs high, and the animal quickly loses flesh, especially if several feet are involved. If the inflammatory action is not checked, a variety of unpleasant and often intractable lesions result—as chronic interstitial inflammation, anchylosis, suppuration, the formation of sinuses, mortification, sloughing, casting of the hoof, open joint, caries of bone, and phlebitis.

If the inflammation becomes chronic the parts become thicker, for a time, and harder, subsequently reducing in size as the inflammatory products become organised and contracted. If bony matter is deposited on the periosteum permanent stiffness (anchylosis) of the joints usually takes place.

If suppuration ensues the inflammation becomes intensified, and the suffering

aggravated; and on the approach of maturation of the abscess or abscesses, the skin becomes thinned and fluctuating.

If exit is not given to the pus surgically the abscess bursts, and unless the resulting cavity becomes filled up by healthy granulations, sinuses (fistulæ) are established, in which case the lameness continues, and there is a constant discharge of glary agglutinous pus, which has a sickly unpleasant odour. Where more than one fistula exists examination with a probe frequently reveals intercommunication; and, in addition, extensive undermining of the coronary substance. If ligamentous or bony structure are involved the discharge contracts a distinctive fœtor, and is sometimes black in colour—involvement of these structures always retards the healing process and intensifies the suffering.

If open joint results, the characteristic synovial discharge—*i.e.*, yellow, thick, transparent, and adhesive—makes its appearance, or entirely replaces the sinual pus—it is a grave complication.

If mortification ensues, the red blush of the skin disappears; it becomes livid, cold, and insensible; evolves a fœtid odour, and discharges a bloody serosity, which may either collect under the cuticle in the form of circumscribed bladders (*phlyctenæ*), or be poured over the surface; the process is usually accompanied by the evolution of sulphuretted hydrogen gas, which may also collect, in the form of bubbles, under the epidermis, and produce emphysema. The sphacelated structures become separated from the living by a red line of demarcation, and are ultimately cast off by the agency of the inflammatory action which is established in the surrounding tissues. Sloughing is most frequently seen in the interdigital skin, but in very malignant cases the whole of the structures of the foot, and even the pastern, are involved in the destructive process. Dry gangrene is less frequent than moist, but when it does take place the dead structures become shrivelled and tough (leathery), and cling tenaciously, and for a tolerably long period, to the surrounding parts.

Mortification (unless circumscribed) is usually accompanied by grave constitutional symptoms—as vital depression, quick weak pulse, hurried respiration, fœtor of the mouth, fœtid diarrhœa—and interference with all the normal functions. If it is at all extensive, death is an inevitable result; if circumscribed, the chasm which is left after the removal of the slough becomes filled up with granulations, the wound cicatrises, and the animal may be restored to perfect health.

It occasionally happens that the whole of the structures below the fetlock are amputated by the sphacelating process, but even if this does take place, the animal (if of value for stock purposes, or otherwise worth keeping) may ultimately recover—though minus part of a limb—and do well.

Secondary exuviation of the hoof may be produced by extension of the inflammation to the laminæ, and if the coronary band is extensively disorganised, imperfect restoration, only, can be expected.

The last result of coronitis is inflammation of the veins (*phlebitis*), forming the coronary plexus, and unless the inflammation is circumscribed, and the veins become rapidly occluded by the formation of coagula (*thrombi*), and by the pressure of the surrounding exudates, there is great danger of small portions of the clots becoming detached and being carried away by the circulation, and lodged in the vessels of some of the internal organs—producing embolism or plugging, with all its consequences.

If the phlebitis extends upwards, numerous (multiple) abscesses form in the affected limb—they may be superficial or deep-seated.

Treatment of Feet Sequelæ.—Unless some valid reason exists for the preservation of life, or the diseased processes are not severe, it is very unwise to attempt treatment, particularly if the animal is fit for slaughter, as in almost every instance recovery is tedious, the loss of flesh great, and, in addition, there is always a probability of death resulting.

If neither sphacelus or multiple abscesses exist, the flesh is perfectly wholesome, and even putting the question of profit aside, it is far more merciful to put an animal, suffering from extensive feet lesions, out of pain than allow it (as I have more than once seen done) to drag out a miserable existence for an indefinite period.

If treatment is determined upon, the first care of the practitioner is to endeavour to moderate and circumscribe the inflammatory action. This is best accomplished by free and deep scarification, immersion in hot water, poultices, the application of belladonna, aconite, or arnica lotions or liniments, and the internal administration of cathartic and febrifuge medicines—pain may be relieved by the subcutaneous injection of morphia or atropia. If the inflammatory action stops short of suppuration, but still persists, the application of a blister will often prove beneficial; and, at a subsequent period, when the superficial inflammation is reduced, the actual cautery or setons should be had recourse to—their use is also indicated in anchylosis.

If suppuration is threatened, efforts should be made to expedite the suppurative process by fomentations, poultices, and the inunction of stimulating liniments—such as lin. sapon. co.—while the application of a blister, in some cases, hastens suppuration materially.

After the evacuation of the pus, premature healing of the wound or wounds must be carefully guarded against, otherwise secondary suppuration may take place, or sinuses form. If the orifice of the wound is small it should be enlarged, and antiseptic dressings freely applied two or three times a-day.

If fistulæ are produced they should be fearlessly laid open—much greater liberty can be taken in this respect with the foot of the ox than with the foot of the horse—and treated as common wounds. If there are several communicating sinuses, setons may be tried.

If evidence exists of bone ulceration, endeavours should be made to expose and scrape the diseased surface, subsequently applying dilute mineral acids or solution of

chloride of zinc. It must be borne in mind that all attempts at healing will be rendered futile if carious bone or necrosed ligamentous structures are present.

If a joint becomes opened, the practitioner may adopt one of two courses, either persevere with his efforts to reduce inflammatory action and apply antiseptic liniments to the wound, in the hope of its becoming filled up by granulations; or destroy the synovial membrane by the injection of an irritant. Anchylosis will of course follow the latter method of treatment, but it is a matter of little moment in the ox or sheep.

If mortification is threatened, measures must be adopted with the view of circumscribing it—as deep scarification and the free application of a stimulating antiseptic liniment, with a blister to the skin of the surrounding parts. Subsequent separation of the dead parts must be favoured by the use of antiseptic poultices and stimulating liniments—the resulting chasm being treated in the ordinary manner. If there has been sloughing of the interdigital structures, care must be taken in the subsequent healing (cicatrisation) to prevent the digits becoming firmly and closely united, by inserting a piece of cork or wood between them.

Immediately signs of mortification are evidenced, the patient should be allowed good nourishing food and stimulants. Ammonia, malt liquors, and spirits, with tonics and antiseptics—as cinchona, iron, gentian, &c.—should be ordered. If multiple abscesses form in the limb, the treatment hereafter advised for pyæmia must be adopted.

In very severe and intractable cases of feet sequelæ—as in sphacelus, disease of the bones, joints, or ligaments—the question of amputation may be considered. If determined upon, it must not be forgotten that the bones of the lower phalanges are duplex, and consequently, in some cases, those of one side only may require to be removed, the others being left intact. Sheep, and very often cattle, do much better on three legs than most people imagine, and it is not a difficult matter to apply a wooden leg—even in the case of the ox.

MOUTH SEQUELÆ.—The results of Eczema, as observed in the mouth, are few, and are always due to extension of the inflammatory action to the deeper-seated structures, in the case of the tongue producing *glossitis*. The degree of inflammation will be regulated, to a great extent, by the amount of irritation to which the mucous membrane has been subjected by coarse food or premature rupture of the vesicles. Concentration of the lesions in the mouth increases the liability to deep-seated inflammation.

The symptoms of glossitis are, continued inability to masticate, persistence of salivation, and fever—the tongue becoming swollen, hot, red, and tender.

The inflammation may subside in the course of several days, or emerge into a chronic form (interstitial)—in severe cases it occasionally terminates in the formation of abscess or in mortification.

If extensive suppuration is going on, fever runs high, the inflammation increases

in intensity, the tongue becomes enormously swollen, fills up the oral cavity, and ultimately, from its size, hangs pendulous from the mouth, greatly congested, and of a livid colour.

The parts in which the abscess is forming are at first hard and resistant, subsequently becoming soft in the centre as maturation proceeds—swallowing is very difficult.

If mortification is threatened, the organ becomes livid, cold, and insensible; phlyctenæ form on its surface, and a red line of demarcation develops between the living and dead parts; in some cases, the sphacelating action is not thus circumscribed, and the dark colour shades gradually off into the adjacent healthy structures. The usual constitutional symptoms of mortification will also be present, and if the animal lives a sufficient length of time the involved parts of the tongue fall off, naturally amputated.

If the inflammation takes on the interstitial form (as it does more frequently than any other) the symptoms, and consequently the suffering, are less acute; the tongue, however (usually the whole of the organ), gradually enlarges, becomes harder, less mobile, and less sensitive; it generally regains its normal temperature and colour, but loses the sense of taste. Prehension, mastication, and deglutition become more difficult every day, and as a result the animal's body visibly emaciates—death ultimately taking place from inanition.

If life is prolonged, the hyperplasy gives place to atrophy, and as a result of this change the organ daily diminishes in size but increases in hardness—while the immobility remains.

The condition of the tongue here described is known as *induration* or *scirrhus*, and it is readily detected after death by the resistance which the tissues offer to the action of boiling water, if attempts are made to cook the organ.

The treatment to be adopted in glossitis depends upon its stage. At the outset, every endeavour should be made to limit the inflammatory action by the administration of saline febrifuges and sedatives; by scarification, in order to relieve the tension and pain; by general blood-letting, if the condition of the system warrants it; and by withholding all solid food—allowing only nutritious fluids. If the animal evinces a desire to drink (it is often very thirsty), cold water containing a little nitrate or chlorate of potash may be allowed. The tongue should be suspended by the aid of a bag secured to the horns on either side, the bag containing sponge, cotton wool, or other porous material, upon which hot water should be constantly poured. Arnica and belladonna lotions may be applied directly to the tongue.

If the inflammation continues, it will be necessary to introduce nutrient fluids by means of a canula and double-action syphon, directly into the rumen, as enemata are seldom retained by ruminants, and it is very unwise to irritate the tongue by administering large quantities of material in the form of drenches.

If an abscess forms, it must be evacuated as quickly as possible, even though it is necessary to cut somewhat deeply for the purpose. If extensive mortification takes place, the animal had better be destroyed, but if it is circumscribed the separation of the slough should be favoured by scarification, hot fomentations, and the application of turpentine liniment.

If the inflammation takes on the interstitial form, scarification, with the local application of solution of potassa iodidum, and the internal administration of the same salt, is all that can be done. It is very intractable and unsatisfactory to treat, consequently the animal should be slaughtered if its condition is at all good.

UDDER SEQUELÆ.—Are due to extension of the superficial inflammation of the skin and mucous membrane—usually the latter—to the glandular structure of the organ; or to inflammatory action set up by the neglect of the attendant to withdraw the milk (which acts as an irritant) during the course of the disease.

Mammitis is usually most severe in heavy milkers, and its intensity is greater in those cases in which it immediately succeeds the primary processes than when some time has elapsed after their subsidence, unless the animal has been allowed to lie on cold wet pastures. Parturition and previous disease of the udder always act as predisposing causes to the extension of inflammation.

The symptoms of mammitis are identical with those of inflammation elsewhere, and its results are the same as in glossitis. If the inflammation is not very acute, indolent abscesses may form; I have seen a great number of them in one gland, and in some cases have been satisfied that their formation has been secondary to, and dependent upon, the existence of feet sequelæ.

The course of treatment to be adopted is the same as that recommended for glossitis, but constant friction and the inunction of castor oil, in addition to belladonna and aconite liniments, can be here had recourse to with benefit; any milk that may be secreted must be removed, and a warm mild solution of bicarbonate of soda injected into the teats. In suspending the udder it will be necessary to pierce the bandage so as to allow of the protrusion of the teats.

Some authorities recommend local blood-letting from the milk (superficial abdominal) vein. It is an unwise procedure, as the operation of phlebotomy, while the system is in such an unhealthy condition, is very apt to produce phlebitis or thrombosis, with such systemic results as pyæmia.

If abscesses form at the base of the teat, they may be evacuated by the aid of a syphon or a small concealed bistoury without making an external incision.

If the udder becomes indurated and enormously enlarged, excision may be necessary; and, in case of sphacelus, the mortified structures should be got rid of as quickly as possible, in order to prevent absorption of the infective fluids which are formed in the process.

The other Sequelæ of Foot-and-Mouth Disease are—Abortion, Diffused External Cellular Abscesses, Cysts, Thickening of Joints, and Internal Abscesses.

ABORTION.—May take place in malignant or suppressed cases during the active stages of the disease, or it may not occur until after the subsidence of the primary processes. It is produced by several causes.

1stly, By extravasation or effusion into the tissues of the placental membranes.

2ndly, By the fœtus being supplied with impure blood.

3rdly, By the fœtus itself contracting the disease.

With reference to the latter cause, I may remark that no instance of congenital transmission has, as yet, come under my personal observation, though a few instances of the phenomenon are on record.

Abortion, in this, as in other zymotic affections, may be favourable to recovery; on the contrary, it may become an independent cause of death, owing to the great rapidity with which the placenta, uterine tissues, and blood coagula undergo liquefaction and decomposition—absorption of the fluids taking place through the open mouths of the uterine vessels. Independently of absorption of infective matter, death may be produced by the breaking up of thrombi in the uterine veins—leading to embolism, and its results in distant organs.

DIFFUSED OR UNLIMITED EXTERNAL CELLULAR ABSCESSES.—These—with the exception of foot lesions—are the most frequent results of Foot-and-Mouth Disease. They may be *superficial* or *deep-seated—i.e.*, located in the subcutaneous connective tissues, the intermuscular tissues, or the ligamentous structures surrounding joints.

The predisposing causes to the formation of these abscesses are—neglect to supply proper bedding, or allowing affected animals to lie on hard and uneven ground; great weight of animal's body and imperfect nutrition of the tissues from over-feeding—hence, such abscesses are most frequently seen in high-bred, grossly-fed animals; persistent recubation, as the result of inability to bear weight in severe and protracted foot lesions; and, probably, interference with the nutrition of the blood and the walls of the blood-vessels, from prolonged and excessive fever, and from the action of the poison on the blood.

The actual causes are—1stly, Injuries received in recubation, leading to rupture of blood-vessels, and extravasation of blood, the extravasate subsequently undergoing degeneration; and 2ndly, Localised zymotic cellular inflammation.

Injury, received in recubation, is, without doubt, the most frequent determining cause of the formation and localisation of these abscesses—hence, they are most frequently developed in the knees, hocks, stifles, shoulders, sides, and hips.

The abscesses vary in size, from a few inches to one or two feet in diameter; in number, from one to twenty; and in capacity, from one or two ounces to two or three gallons. Their contents are, primarily, exuded lymph or extravasated blood; secondarily, purulent matter mixed with masses of degenerated muscular or cellular tissue, or blood coagula. The matter may or may not be fœtid, but the presence or absence of fœtor will depend upon the length of time the abscess has been in existence.

Characters.—If located in the limbs, great stiffness and, usually, lameness, are observed; the affected part is swollen, and, after a day or two, hot and tender, the swelling being, except in those cases where the lymph or blood has been thrown into comparatively circumscribed sacs—as the superficial bursæ of the knees, hocks, stifles, and hips—diffused and puffy. In a day or two, pain on pressure is evinced, and the swelling shows a tendency to diffuse, owing to the occurrence of fresh hæmorrhage, or to the extension of the degenerative or suppurative process, from the non-formation of a limiting membrane. Diffusion is sometimes very rapid and extensive. I have seen the whole of one costal and the whole of one scapular region involved in the course of a few days.

The puffy character of the swellings rapidly gives place to imperfect, followed by perfect fluctuation, thinning of the skin, and pointing; but, owing to the non-limitation of the abscess, pointing, especially on the sides, shoulders, and haunches, is often delayed and imperfect, the suppurative action continuing to extend in every direction, until the animal sinks from exhaustion, fever, or absorption of the purulent fluids.

The constitutional disturbance attendant upon the formation of these abscesses is sometimes very great, at others astonishingly slight. In some cases fever runs high; there is great prostration, loss of appetite, arrest of lactation in milch animals, suspension of rumination, rapid emaciation, moaning, and gnashing of the teeth, with constipation of the bowels; the urine being scanty and high coloured.

In other cases all the natural functions are performed regularly, there is no fever or loss of flesh, the animal is bright and cheerful; and in microscopical examination no material alteration can be detected in the blood or milk, even though abscess after abscess may have been formed, and the process has extended over a period of months.

It occasionally happens that after a number of diffused abscesses have been evacuated, grave constitutional symptoms are suddenly developed; these are due either to pyæmia or septicæmia, and are usually followed by death.

Treatment.—Early maturation should be favoured by fomentations and the frequent application of stimulating liniments, the compound soap liniment being the best. If no signs of pointing are observed in the course of a few days, and extension of the abscess is manifest, no time should be lost in having recourse to surgical evacuation. In lancing, a tolerably free vertical incision should be made at the most dependent part of the swelling, and the whole of the contents, whether solid, semi-solid, or fluid, should be carefully evacuated, the resulting cavity thoroughly washed out with a warm solution of any antiseptic—as Condy's Fluid, carbolate of glycerine, or salicylic acid—and the mouth of the wound plugged with a little fine tow, saturated in carbolic or creasote liniment. Where practicable, the tow should be retained *in situ*, and the walls of the sac compressed by the application of bandages. Folds of antiseptic linen may be laid over the wound, in place of plugging, if it is thought preferable.

The enormous size of these abscesses, and the extent of undermining of the skin,

sometimes leads to the fear that evacuation will be followed by extensive superficial sloughing. This, however, seldom takes place, as the skin is so thick as to retain its vitality perfectly.

If several abscesses are formed in close contiguity and subsequently intercommunicate, it may be advisable to introduce setons or drainage tubes after evacuation.

In the case of the knees and hocks, every precaution must be taken to prevent bruising by the use of soft bedding and the application of soft bandages—poultices are sometimes very useful where they can be retained in position.

Slings, in the case of cattle, are—in my experience—more productive of harm than good; but where an animal is incapable of rising it will be necessary to attend to the regular evacuation of the fœces and urine, to turn from side to side twice or thrice daily, to remove all wet or dirty bedding, and, in the case of milch cows, to prevent accumulation of milk in the udder.

Constitutionally, every means should be adopted with the object of strengthening the system, such as the allowance of nutritious, easily-digested food—milk, eggs, and malt liquors, if necessary—and the administration of vegetable and mineral tonics, particularly iron compounds, sulphuric acid, and cinchona.

If the milk is microscopically and otherwise normal, and there is no evidence of constitutional disturbance, it may be utilised; but it is better to check lactation than to favour it.

In order that the course of these cases may be better understood, I have here introduced the notes of two which occurred in my practice in 1873.

Secondary Diffused Cellular Abscesses in a Cow as Sequelæ of Eczema Epizootica.

Subject.—A red and white shorthorn cow.

History.—About three weeks prior to my attention being called to this animal, a fresh cow had been introduced into the byre, and retained there for three or four days; but as she had an unhealthy appearance she was returned to the vendor, and in a few days after the subject of this history was noticed unwell, the most prominent symptom being a very violent cough, which continued a few days, when the attack appeared to have subsided. Another cow, which stood in the same byre, was also unwell, and ultimately one quarter of her udder became indurated. Eczema, however, was not suspected in either case, and it was not until the formation of abscesses was noticed that my advice was sought—viz., on the 1st of April, 1873. The symptoms then present were those of slight fever, natural functions not interfered with; appetite, on the whole, good; the body well nourished, the skin sleek and healthy. The right thigh, from the hip to mid-way between the hock and stifle, presented one enormous round swelling, readily observable as I entered the door of the byre—on the left hip a swelling also existed, more regular and defined than that on the right. From the character of

these enlargements, and from finding on manipulation that they contained semi-fluid matter, I at once inquired if the cow had suffered from Eczema. I was answered in the negative. I then examined the feet, and found the hoofs exfoliating; a large quantity of furfuraceous material also lay on the skin of the haunches and back. From a consideration of these facts, coupled with the state of the other cow's udder and the history above given, I had no hesitation in declaring that both animals had suffered from Eczema Epizootica.

Treatment.—Evacuated the abscesses, both of which contained a large quantity of pus, fœtid and of the consistence of cream. The abscess on the right hip could not have contained less than from four to five quarts; the pus from that on the left, however, was the thickest, and in each case it was mixed with large masses of what appeared to be disintegrated fibrinous tissue. The abscesses were well washed out and syringed with lotio carbolici acidi twice a-day; good food and good bed ordered; at the same time, administering tonic medicines, with an occasional saline purge. On 3rd, she was progressing favourably; I had, however, to lance the off-side in a fresh place; both legs were œdematous and hot, but otherwise, appetite, &c., normal, and animal appeared much relieved. The abscesses closed in about a week, and I entertained the hope that all would go well. On 27th June, I was again asked to attend, when I found that an abscess was forming in the near hind foot,—the whole coronet and pastern being enormously swollen, tense, red, and tender, and the digits wide apart; the animal was also suffering extreme pain, but still she ate well, although she lay much, and the respiration was very hurried. I scarified the diseased parts freely, and had ungt. hydrarg. biniodidi well rubbed in; two days after yeast poultices were applied, and in a few days the pus found exit inside the leg about three inches from the coronet. This was followed by a sinus three to four inches deep and taking an inward direction, though always discharging healthy pus: it was simply treated with poultices and the insertion of stick nitrate of silver, and ultimately did well. 16th July—I was again asked to see the cow; this time I found an abscess immediately under the left hip-joint, which I evacuated and treated as before. 29th—Another large abscess had formed in the right tibio-femoral region and one above it, covering the whole of the gluteal region—the latter evidently being more superficial than the former and its contents more fluid. On evacuating these the following day I found that the contents of the inferior one was of the same character as the others—the contents of the superior one being quite fluid and very fœtid. Mr. Matthews, a student, who was with me at the time, discovered another abscess on the left side an inch or two below and in front of the antero-inferior spinous process of the ilium; and on further manipulation I found two others in the course of formation—viz., one on the extremities and superior surfaces of the last two transverse processes of the lumbar vertebræ, and the other above and behind the anterior spinous process of the ilium. I at once determined upon opening all these, and in doing so I found they contained comparatively

fresh blood-clots, with portions partially disintegrated and undergoing conversion into pus—there was no fœtor. The one below the spine of the ilium, and the one above and behind it, communicated with each other through a round aperture (I should have mentioned that this hip had been much crushed in lying, as she always lay on this side), and in making a deep incision in a backward and downward direction from the latter, I found a large quantity of extravasated blood, which I at once removed. All these abscesses were treated in a similar way to the first, especial care being taken to prevent too early closure, and to do away with the fœtor, a seton being also passed between the two communicating ones. The animal was liberally fed on cake treacle, sugar, &c., cinchona, potassa chloras, and gentian being administered in ale twice a-day; in ten days her condition had materially improved. 10th August—All the wounds doing well, except the upper one on the off-side, the suppurative action in which had extended to the ischium, and the discharge was still thin and fœtid. I made an incision over the region of the superior spinous process of the ilium, passed a seton through it and the original wound, and dressed with lint. creasotin co. She milked well, the milk was of excellent quality, and microscopically pure. 22nd—Old abscesses were progressing favourably; an irregular swelling about the size of the hand had appeared a little to the right of the umbilicus; I suspect the formation of another abscess. Another cow standing in the same byre, and suffering from indurated udder, had two abscesses of a similar character in the off-hind quarter of that organ. 30th—The old abscesses were still progressing favourably; the enlargement on the abdomen was reduced in size, tender to the touch and hard, but no signs of suppuration existed. In the udder of the other cow a third abscess had formed at the superior part of the off-fore quarter, the teat also contained a large quantity of pus. She was, however, in good condition, and fattening fast. 12th September—The first cow had progressed favourably, the coronet and pastern becoming callous, smaller, and losing the inflammatory blush, while the discharge was also diminished. The abscesses on the right haunch were still discharging, but the pus was less in quantity and less fœtid. The tumour under the abdomen remained hard, but was smaller, and appeared like pointing. The abscesses in the udder of the other cow had burst at the upper part of the right anterior quarter, and also at the base of the teat.

In the last week of September, up to which time she had been progressing favourably, the first cow suddenly commenced to breathe rapidly, and this continuing for some days, the owner, under the impression that she was suffering from Pleuro-Pneumonia, had her slaughtered without my knowledge; but I was informed afterwards by Mr. Reid, V.S. (the Inspector), that a number of small abscesses existed along the inferior part of the abdomen, and a large one in the dewlap, but none internally—he did not examine the glands. The rapid breathing would probably be due to fever consequent upon the formation of fresh abscesses, or it may have been due to septicæmia.

ABSCESSES IN JOINTS.—Are usually the result of pyæmia, extravasation of blood, exudation of lymph, or metastatic inflammation. They are more serious than those we have just considered, and their formation is accompanied by a greater amount of suffering. Their course and treatment are identical with suppuration elsewhere, only that the measures to be adopted, in the way of treatment, must be energetic and prompt. I prefer the early application of a blister, and its subsequent repetition if necessary; the joint, both before and after evacuation of the abscess, must be as far as possible immobilised by the aid of splints, bandages, &c. In almost every instance where the animal recovers, the joint becomes stiff (anchylosed), and if a *post-mortem* examination is made, it will be found that the articular cartilages have been removed by softening and attrition, and the articular extremities of the bones more or less inflamed or carious.

The surrounding connective tissue and the muscles are largely infiltrated with serum or lymph, and in long-standing cases the muscular tissue is replaced by fibroid —fibroid substitution.

CYSTS.—Are usually formed in the superficial bursæ of the knees, hocks, stifles, and hips, and are produced by effusion of serum, as the result of pressure or of the altered condition of the blood and tissues (dropsy); or, from extravasation of blood—organisation of its viable portions and expression of the serum into the cavity of the bursæ.

These cysts may be small or may attain a very large size; they are always circumscribed, and although they do not, as a rule, give rise to lameness, they produce stiffness, and the skin becomes abraded and thickened, and, in some cases, ulcerated. Secondary inflammation, too, may be set up, organisable lymph thrown out, and the yielding, fluctuating, comparatively painless swellings become converted into hard and painful ones, rendering the joints stiff, and probably ending in the deposition of bony matter, with true anchylosis as a result.

Distension of the sheaths (thecæ) of the tendons may also take place either in conjunction with, or independently of, the bursal cysts just described; it is due to increase in their normal secretion (synovia) and the resulting swellings—following the course of the tendons—are elongated.

Treatment of Cysts.—The first care of the practitioner is to determine, as accurately as possible, the precise nature and localisation of the contained fluid—*i.e.*, whether it is synovial or serous, and whether it is contained simply in the superficial bursæ or communicates with the interior of the joint or joints. The first point is determined by the use of the exploring needle, as is also the second to some extent, *i.e.*, by observing the character of the synovia, if present, as to whether it is arthrodial or bursal: manipulation will also materially assist in arriving at a conclusion on this point.

If it is found that the cyst is confined entirely to the superficial bursa, no doubt need exist as to the adoption of radical measures for its reduction.

Two courses offer themselves—1stly, The introduction of a seton through the bursa; 2ndly, Boldly opening it by a vertical incision. If the first course is adopted, the seton should be passed through the whole length of the bursa, and the tape drawn backwards and forwards twice a-day, cleansed, and saturated with creasote or carbolic liniment—it may be retained in position for about ten days or a fortnight, or until the serous and the subsequent purulent discharge have become replaced by lymph. After the withdrawal of the seton, premature closure of the inferior aperture resulting from its introduction must be prevented.

If the second course is adopted, every drop of the fluid contents of the cyst should be expressed by gentle manipulation, all loose or partially loose lining membranes removed by the finger, the sac washed out with carbolic or mild iodine solution, and the mouth of the wound protected by a pledget of carbolised tow—a bandage being applied where practicable. Serum will be discharged from the cyst for a day or two, and will then give place to pus, the latter being followed by lymph: the sac ultimately becoming obliterated by adhesive inflammation.

THICKENING OF JOINTS.—May be due to secondary causes (considered above), or to primary exudation of lymph into the connective tissue surrounding the joint. At the outset the swelling is somewhat doughy, painful, and hot, subsequently becoming painless and unyielding. The amount of attendant lameness and stiffness will be regulated by the extent of the swelling, the rapidity of its formation, and the subsequent changes in the exudates.

Treatment.—The application of refrigerant or iodine lotions, with cold water and cold bandages, should be tried at the outset, but if they fail in effecting reduction repeated blistering should be had recourse to—protecting the blistered surfaces from friction by the use of canvas caps or bandages.

The compounds of iodine, with an occasional laxative and diuretic, should be administered internally; and, so long as there is evidence of inflammatory action, dieting should be attended to.

INTERNAL ABSCESSES.—May be primary or secondary, primary when resulting from the throwing out of lymph or extravasation of blood into the tissues of organs; secondary when resulting from pyæmia.

Evidence of the formation of internal abscesses is afforded in the occurrence of rigors with high fever some time after the subsidence of the active symptoms of the disease; the natural functions becoming partially or totally suppressed, and the animal showing pain by moaning, gnashing of the teeth, and uneasy movements, and, as time goes on, becoming emaciated and weak.

Internal abscesses may be formed in any of the abdominal or thoracic organs; and the localisation of the suppurative process in any particular organ will lead to the production of pathognomonic symptoms in addition to those above mentioned.

In the case of the intestines and lungs, these abscesses may be evacuated by the natural channels, but as a rule they terminate in death.

Treatment is of little avail; the general comfort, however, of the patient should be attended to, pain relieved by anodynes, fever moderated by febrifuges, and the vital forces sustained by easily assimilable, but non-stimulating, nutritive materials.

PREVENTION AND SUPPRESSION.

Owing to the multitudinous channels through which the virus of this disease is disseminated, prevention and suppression are very difficult, and any measures which may be devised—having these objects in view—must be promptly and vigorously carried out.

Looking at the fact that the virus exists in the mucous discharges of the eyes, nose, and urino-genital organs, in the saliva (probably in the expired air from the lungs and the cutaneous exhalations), and in the local vesicles; and that all these fluids, either in a dry or moist condition, become dispersed far and wide, and lodge in the exposed mucous surfaces of healthy animals, or ingested by them with water or food; we need not be surprised when we find that suppressive and preventive measures are often only partially successful.

The prophylactics which I have hitherto found most effectual have been—perfect isolation of diseased animals; suppression of all movements; destruction or thorough disinfection of all contaminated porous materials, whether food, litter, ingesta, dung, clothing, or feeding utensils; and of dead animals, hides, hoofs, &c.: the administration of antizymotic medicines, as the sulphite or sulpho-carbolate of soda, or carbolic acid; and the thorough disinfection of tainted buildings or fixed fences with sulphurous acid, chlorine, lime-wash, disinfecting paints, and gas-tar.

With regard to isolation, I am perfectly aware that rigid internment of a large number of animals is a very difficult matter, nevertheless, every endeavour should be made to carry it out. I have succeeded, beyond my most sanguine expectations, in protecting single and small numbers of animals, by placing them (so far as was practicable) in isolated buildings; attaching to the horns (of horned animals) pieces of cloth saturated with strong carbolic solutions—taking care to prevent the fluids running into the eyes and mouth—suspending sacks or canvas over the doors and windows, and bedding with sawdust mixed with carbolate of lime.

In the case of *Pigs* there is no more certain method of disseminating the disease than by the adoption of the, too common, foolish plan of feeding them on the rejected foods, the internal organs (with their contents), and the blood of diseased animals.

Amongst *Sheep* the disease is very often spread by the shepherd, in the performance of his ordinary duties, in the dressing or examination of the feet of affected

animals, and subsequently dressing the feet of those suffering from foot-rot, and in the lambing of ewes when the skin of the hands and the clothes are contaminated with the virus of the malady; hence the necessity of providing separate attendants for the healthy and the diseased portions of the flock.

In dealing with sheep, too, the fact—previously pointed out—of their being subject to relapses of the disease, must not be forgotten, as there is every probability that they are just as capable under these circumstances of propagating the disease—though in a less degree—as when they are suffering from a primary attack; I say there is every probability, because I have no positive arguments or proofs to advance either for or against propagation in this way.

In the case of *Cattle* I have usually insisted upon the necessity of occasionally anointing or sponging the skin during the active and convalescent stages—the latter especially—of the disease, with some simple disinfecting pomade or wash, with the view of rendering the exhalations and desquamated epidermis innocuous.

Vaccination has been suggested as a preventive measure in Foot-and-Mouth Disease. It is utterly valueless, indeed it is inconceivable that it could be otherwise, seeing that the disease itself is not protective.

Inoculation—i.e., in a very primitive, but oftentimes very effectual way—has been had recourse to largely by farmers and dealers, particularly the latter, the method usually adopted being the saturation of wisps of hay with the saliva of infected animals, and introducing them into the mouths of healthy ones—using a little friction in the introduction.

The great advantage of this plan is, that the animals of the herd or flock are affected as nearly as possible together, and consequently the disease is got rid of much earlier than by allowing it to run its natural course.

In reference to the utility of the provisions of the new Act, as applying to Foot-and-Mouth Disease, it would of course be premature to hazard an opinion until the disease has again made its appearance amongst us on a large scale. As in the case of Pleuro-Pneumonia, one step has been made in the right direction—viz., the extension of the restrictions to fourteen, or, if it is thought necessary, to twenty-eight days; but, as with the Pleuro-Pneumonia rules, the great flaw exists of allowing all animals which have been exposed to the infection, in markets, auctions, &c., to go scot-free. I have no doubt, however, that, as large powers are now given to the Privy Council for the regulation of markets, fairs, &c., advantage will be taken of these powers to issue more stringent orders in this respect, should the disease again become rife.

CATTLE PLAGUE.

SYNONYMS.—Rinderpest; Steppe Murrain; Contagious Typhus.

The term Rinderpest is derived from the German "rinder" (cattle) "pest" (plague).

Steppe Murrain—from the supposition that the disease originates in the steppes of Russia and Russian Poland.

Contagious Typhus has been applied by foreign veterinarians from the belief that the disease was closely allied to typhus fever, and from its contagious nature.

The term Cattle Plague is the most significant, and the one most largely employed in Great Britain.

DEFINITION.—A specific eruptive fever, peculiar to cattle, produced by a special zymotic poison, and having its lesions localised in the skin and mucous membranes.

NATURE AND CHARACTERS.—Cattle Plague, though indigenous in the bovine tribe alone, is propagated to sheep, goats, and other ruminants, but we have no evidence of its having been conveyed to other than ruminating animals; and even in sheep, goats, and allied creatures its symptoms and course present important modifications, as compared with those usually seen in the bovine species. The nature of the disease has afforded much matter for inquiry and reflection, consequently many and very diverse conclusions have been arrived at in reference thereto. That it is purely zymotic, and not a septic affection, all authorities are agreed, but in reference to the point, as to whether it should be classed under the head of typhoid or variolus affections, there is, and must necessarily be, a great diversity of opinion. Like typhoid fever, it runs a definite course, is of a low type, is marked by important intestinal lesions (and these, too, in the small intestines), and by characteristic skin eruptions; while its catarrhal, and intensely infectious and contagious characters, with the cutaneous complications, lead to its association, in the minds of observers, with smallpox. Even in Thibet, according to the report of the Indian Cattle Plague Commissioners, this idea is entertained; and much stress was laid on its striking resemblance—in some particulars—to smallpox, by the Cattle Plague Commissioners of Great Britain in 1866; but I fail to see that the points of identity are so great as to render it necessary to accord to the matter more than a passing notice.

Very close analogies have been observed to exist between Rinderpest and Foot-and-Mouth Disease, and it has often occurred to me, when contemplating malignant cases of the latter affection, that the points of divergence between the two were very slight, and that although these divergences were sufficient to enable us to differentiate the two diseases, they were, at least in their causes and pathology, intimately allied—were, as I have more than once remarked, own cousins; and that probably very slight forces would cause the one to develop into the other.

The striking general resemblances which exist in the nature of the two diseases were never before so forcibly impressed on my mind as in 1872, when Rinderpest was introduced into Leith Roads by the s.s. *Benachie*. To these analogies which exist between the diseases in question I shall refer more at length when speaking of diagnosis of Cattle Plague.

Another class of diseases to which Cattle Plague somewhat approximates in some of its characteristics are, those which are known as Aphthous; but, while certain points of resemblance undoubtedly exist—especially in the localisation of the lesions in the mucous membrane of the alimentary tract—the points of divergence are so great as to enable us at once to draw a wide line of demarcation between them.

Aphtha is, more generally, an ovine disease, and in this species of animal alone does it assume anything like an enzootic or epizootic type; it is, too, usually confined to young animals, is not—except it is of a malignant type—destructive to life, and is purely local (usually dietetic) in its origin.

That Cattle Plague is, like allied affections, due to a specific poison there can be no reasonable doubt, however much we may be in the dark as to its precise nature. Equally certain is it that this poison when introduced into the blood of an animal sets up a high degree of fever, which originates important changes in the mucous and cutaneous surfaces and in the circulatory and nervous systems; and although serious tissue changes are always observed to result from a high temperature—no matter how it may be produced—the structural transformations and phenomena of Cattle Plague possess very distinctive and pathognomonic characters. The question as to whether the viruliferous principle of Cattle Plague possesses recognisable physical properties, or whether it is ultra-microscopical, may be left open, but one of the best writers on the subject, Professor Semmer, of Dorpat, seems to entertain little doubt on this point, and ascribes it to a form of bacteria which he has always found associated with the disease.

As Professor Semmer has had opportunities of studying the nature and characters of Cattle Plague in its home, and under such favourable conditions as can scarcely ever be obtained by British or Continental veterinary surgeons, I do not hesitate to introduce a short summary of his views on the subject, extracted from a publication of his (printed in 1875) by Mr. George Fleming, and quoted by him, in a review on this little work, in the *Veterinary Journal* for January, 1876.

The summary is mainly the result of a large number of experiments by inoculation, in reference to which Professor Semmer says :—"The ten *post-mortem* examinations (fully detailed in the *Journal*) alluded to, prove that the anatomo-pathological alterations in inoculated animals which do not show any marked symptoms of the disease thirty-six hours after inoculation, are yet quite characteristic.

"The intensity in the appearances increases with the duration of the disease. The most notable lesions were remarked in the last two cases, which did not die until thirteen and fourteen days after inoculation.

"The microscopical examination of the inoculation matter (nasal and mouth mucus, tears), of the blood, and of the glandular organs, yielded the following results : In the blood the colourless corpuscles were always increased in number, and were often heaped together in ball-like masses, very granular, and stellate-shaped. Globular bakteria (*Kugelbakterien*) were found in the colourless, as well as in a few red corpuscles, and in the serum. In the blood of a calf thirty-six hours after inoculation, the white blood-corpuscles were to the red as one to thirty. They were extremely granular, and contained bakteria, surrounded by masses of protoplasm. This condition is only to be observed in fresh-drawn blood, which must be examined before coagulation takes place. Otherwise, the white blood-corpuscles become broken up and mixed with the bakteria in the coagulated fibrin.

"In the nasal mucus of the calves, seven hours after inoculation, globular and chain bakteria had made their appearance. The number of colourless corpuscles had increased in the blood, and in the serum were found individual bakteria. The latter increased with the duration of the disease, and decreased in number when amendment took place.

"In the nasal mucus of sick animals, granular epithelium, with granular roundish mucus-cells, blood-corpuscles, and numerous globular and chain bakteria, were observed.

"In the mucus from the mouth, as well as in the tears, similar appearances were noted.

"The inoculating material was procured at Carlowka, by introducing a piece of clean sponge into the nostril of an animal undoubtedly diseased. The sponge, being thoroughly saturated with the nasal mucus, was squeezed out into small glasses, which were then carefully closed. In this manner the viruliferous material retained its potency for several days.

"When this nasal fluid was examined about twenty-four hours after it had been obtained from the animal, the white and red blood-corpuscles were found to be unchanged, and the matter itself without odour. But the number of globular and staff bakteria had, in comparison with the freshly-obtained fluid, much increased. Out of the globular bakteria, short chain and staff bakteria had issued, the latter with distinctly discernible articulations. These bakteria were, some of them, motionless, and others in active movement.

"The movements may, however, be hindered by adding glycerine and vinegar to the

fluid they are in, without any further alterations appearing. The staff bakteria are recognised under a very high magnifying power as articulated chains.

"The bakteria increased until the third day, and were in active motion. On the fourth day, no increase had taken place, and the movements were less lively than on the preceding day. On the fifth day appeared *Vibrionæ* and *Infusoria*.

"Inoculation material which had been buried for two years in small glass tubes smelled strongly of sulphuretted hydrogen, was slightly troubled in appearance, contained decomposition bakteria, and was found to be impotent to produce the disease.

"In the liver and cortical structure of the kidneys were found the alterations in the cells already noticed—viz., enlargement, troubled contents, fatty degeneration, and shedding; bakteria were also present.

"Whereas in the blood and secretions of healthy cattle, bakteria are not met with in such numbers as these control examinations or inoculation experiments yield; and as the inoculation material only remains potent so long as it contains globular and chain bakteria, its potency disappearing with the advent of decomposition bakteria and putridity, so does Professor Semmer maintain that the globular and chain bakteria have an undeniable importance in the pathogenesis of Cattle Plague. He also asserts that the corpuscles which Beale discovered in the bodies of plague-stricken cattle, and which he named modified germinal matter, are nothing more nor less than 'globular bakteria.' As in other contagious or inoculable diseases (Splenic Apoplexy, Septik-æmia), Semmer considers the globular bakteria to be the active agents—not the staff bakteria. The latter most frequently first appear in the last stage of the disease and after death, whilst the globular bakteria always exist from the first. On account of their exceedingly minute size, essential differences cannot be distinctly made out between individual globular bakteria, even with the hightest magnifying powers.

"Their presence in very dissimilar diseases, however, is strongly in favour of their diversity; as well as the circumstance that they are penetrated by decomposition bakteria and are destroyed, and, finally, the different forms into which they may, under certain conditions, be developed.

"The Cattle Plague bakteria are not essentially different from other bakteria; in their further development they form chains of various lengths. After their immigration or transference into living bodies, they multiply in the blood and lymphatic vessels, penetrate the white and also the red blood-corpuscles, inducing division and amplification of the former, and producing obstruction, congestion, and hæmorrhage in different organs. Further, they bring about degeneration and breaking-up of the tissue cells, such as the epithelium and living cells.

"It will be seen from all this that Semmer attributes the production of the disease we know as 'Cattle Plague' chiefly, if not entirely, to the globular bakteria—a very important result to have arrived at, certainly, and one which, if further researches

fully substantiate it, marks an important step in advance, and adds another disease to the list of those whose exciting agent is pretty accurately determined.

"Semmer, in recapitulating the main points of his observations, sums up as follows: The bakteria are created from transformations of the blood and tissues, and notwithstanding the apparent similarity in their form, they are really diverse in their characters; so that every contagious disease has a specific kind of bakteria.

"They always obtain admission to the blood; and, firstly, bring about primary alterations in that fluid, which, secondarily, induces changes in the tissues and organs. By the entrance of large numbers of bakteria into the blood and their increase there, fever constantly ensues. After the epizootic kind of malady which each peculiar sort of bakteria develops, has led to certain blood-elements being consumed, and has engendered certain alterations in the constitution of that fluid, the bakteria perish, or at least cannot further multiply, because they no longer have a favourable soil; in this way they behave like some kinds of seed which, when they have exhausted the soil on which they grew, can survive no longer.

"When the specific soil has thus been permanently and thoroughly altered through the action of the specific contagium, the organism cannot be affected by the same agent a second time; and thus we have immunity conferred from future attacks of the disease: as happens in Cattle Plague, Variola, Distemper, &c.

"But should the alterations, on the contrary, not be permanent, then repeated attacks may occur, as in Splenic Fever, Septikæmia, Glanders, &c.

"Should the secondary effects of the bakteria (Congestion, Infiltration, Degeneration of the internal organs) not be very serious, then convalescence will follow after the expulsion of the infecting agent through the skin, lungs, intestines, and kidneys. If, on the contrary, they are serious, then death will ensue.

"Semmer particularly insists on the recognition of the fact that, in Cattle Plague, bakteria have the same significance as in Anthrax, Septikæmia, Distemper, &c. The contagium of Cattle Plague always engenders a primary blood-disease and fever, before there is any localisation in the mucous membranes and the glandular structures and the production of the secondary changes."

Cattle Plague attacks the bovine species indiscriminately; old and young, fat and lean, male and female, coarsely-bred and finely-bred, each alike fall victims to the scourge; but, while this is the case, certain local influences—as in all other zymotic affections—predispose animals to an attack of the disease. Debility (no matter how it may have been produced), exhaustion, change of climate, bad sanitary arrangements, and unhealthy conditions of the atmosphere, all render animals exposed to their influence more prone to contract the malady.

A certain proportion of animals will in every outbreak be found to withstand the influence of the contagium, but in no other zymotic affection is the percentage of refractory subjects so small as in this. Exceptional cases of non-susceptibility are

occasionally met with by every practitioner, but the few exceptions prove the universal applicability of the rule. In 1865-6, numbers of herds were entirely swept away, and under the most favourable circumstances the percentage of animals which escaped the scourge was extremely small. In the majority of instances which came under my own observation the proportion of refractory animals did not exceed three or four per cent.; certainly some herds, though surrounded at comparatively short distances by the disease, escaped without the loss of a single animal. In one instance only, in which the malady gained a firm footing, was the percentage of escapes large, and the details of this particular case are, I think, worthy of being fully recorded in these pages.

Two small farmers in Lancashire (J—— and G——) occupied neighbouring farms, the steadings of which were only separated by a road of about six yards wide. The plague made its appearance first in the stock of J——, which consisted of four middle-aged dairy cows, in good condition, a yearling and a two-years'-old bull, and one young calf. The yearling bull was the first victim, and he succumbed in about two days after the disease declared itself, the symptoms and *post-mortem* appearances being fairly well developed. It was the first case I had seen, but from what I had learnt of the nature of the disease from Professor Simonds, I had no difficulty in arriving at a correct diagnosis, which was subsequently confirmed by Mr. Welsby, M.R.C.V.S., who was inspector for the district, and who had consequently become well acquainted with the peculiarities of the malady.

I strongly advised the farmer to slaughter the two-years'-old bull as he was in very good condition, but as vaccination was just then the rage, he preferred to try its protective powers, and after some urging I consented to vaccinate the whole of the small stock except the calf. The operation in each case was successful. I did not content myself with resorting to it alone, but in addition administered arsenic twice daily in the food of each animal.

The bull and the young calf fell victims to the disease in due course, while the whole of the cows remained perfectly healthy.

The stock of G—— consisted of nine mixed—old and young—cattle, and, so far as my memory serves me, the whole of them were attacked by the disease, one only of the number recovering.

Cattle Plague in those countries in which it is enzootic is usually of a very benignant type, and not only do many animals run the gauntlet of infection quite scatheless, but the majority of those which are attacked recover, and the symptoms are comparatively but slightly developed. This is probably one of the worst and most deceptive features of the malady, and not only so, but, in addition, an animal which does not show the slightest sign of the existence of the affection in its own system, frequently becomes the means of propagating it in its most virulent form to every animal with which it is brought in contact.

British experience of the disease has hitherto been such as to lead every competent

observer to stamp it as the most virulent and malignant malady to which cattle are liable, and this verdict is endorsed in every country in which the disease is not indigenous. Unlike other zymotic affections, it does not lose—at least to any appreciable degree—its virulent properties by continued residence in the country into which it is imported ; and though it may appear for a short time to decline and to stay its ravages, it only gains strength by the treacherous repose, and anon breaks out with increased fury.

In Rinderpest—as in Foot-and-Mouth Disease—the characters, localisation, and intensity of the lesions vary materially in different epizootics: thus at one time it seems to be concentrated in the nervous tissues ; at others, in the mucous membrane of the alimentary tract, the air-passages, and the urino-genital organs respectively.

In some outbreaks cutaneous eruptions are constant; in others they are seldom seen. In those cases which came under my notice in 1865-6 and 1872, skin eruption was rarely present, and only in a mild form; undoubtedly, this can be explained, to a certain extent, by the fact that many animals died or were killed before cutaneous exanthema had time to develop.

Cattle Plague is highly contagious, and is propagated in a greater variety of ways than any other zymotic affection. Every secretion and excretion, all the fluids and solids of the body, are charged with its viruliferous principle. Hence it can be readily conveyed by the blood, milk, flesh, offal, hides, and hoofs ; by the nasal and lachrymal discharges, the saliva and the buccal mucus, the contents of the stomachs and intestines of dead animals, and by excreta. The contagium being both volatile and fixed, or, assuming these conditions indifferently, it is readily disseminated by *mediate* and *direct* means, and animals which are themselves insusceptible to the influence of the virus readily convey it to those which are naturally its victims.

These facts have been proved by numerous experiments and by the extensive observations of natural facts in every country in which the disease is known.

It has been propagated by inoculation with various fluids of the body, as saliva and nasal and conjunctival discharges; and, during the brief visitation of 1872, Professor Simonds seized the opportunity of setting at rest the question of transmission by the imbibition of water contaminated with the mucus and exudative matter from the mouth and throat. The subject of his experiment was a yearling heifer which had never been in contact with diseased animals, and the symptoms were well marked in four days after the last draught of the contaminated water ; the temperature rising on the 3rd day to 105·2° F. The animal died on the ninth day after the first draught.

E. Semmer, of Dorpat—*Veterinary Journal* (January, 1876)—used nasal mucus for the purpose of inoculation, and he found that it retained its virulent properties when kept in closed glass vessels for a period of seven days, even though vibriones had developed in it ; and when carefully preserved it remained efficacious for years.

Various influences—as in other zymotic affections—retard and favour its spread.

Cold is a preservative of the poison, humidity causes it to hang for a long period about an infected district, while a free circulation of pure air, by dilution, reduces its virulence, though it may disseminate it more widely. Under ordinary circumstances it is not spread to very great distances through the medium of the atmosphere; hence, animals which are perfectly isolated, even within a comparatively short range of those which are diseased, escape the infection. Raupach says that even in the Steppes a wide ditch is sufficient to arrest its spread. In 1865-6 several valuable herds were saved, though the malady raged with great violence all round them, by housing in under-ground sheds; and in the neighbourhood of Market Drayton—as I am informed by Mr. Kettle—a lot of eight or nine young cattle which were grazing on the banks of the river Tern, and removed only a short distance from affected animals, but carefully preserved from contact with them or with contaminated matter of any kind, entirely escaped the disease.

The virus is disengaged from the body of an infected animal from the earliest stage of the disease to convalescence, and probably, through the agency of the desquamated and dried epidermis and the hair, it is propagated after complete recovery. It readily gains admittance to the blood of healthy animals through all exposed mucous surfaces, and probably by cutaneous inhalation.

One attack usually gives immunity from the disease for a long period—six to twelve years, it is asserted by some authorities; for life, by others.

I have before referred to the fact that a certain percentage of animals in every epizöoty of Cattle Plague enjoy immunity from its ravages. It is an interesting inquiry to make as to how far the prior existence of another zymotic affection protects animals from an attack of the disease. In 1865-6 the malady was often found associated with two of its congeners—Foot-and-Mouth Disease and Zymotic Pleuro-Pneumonia—and animals which had recovered from one or both of them were not protected. The prior existence of variola vaccina, artificially propagated by vaccination, though greatly vaunted and extensively practised as a protective, failed to render the systems of bovine animals proof against the more virulent virus of Cattle Plague.

In Leith, however, the escape of several dairies of cows was attributed to the fact that most, if not all, of the animals had passed through a natural attack of cow-pox.

History.—In considering the history of Cattle Plague, I shall confine my remarks mainly to what is known of its existence in Great Britain. As it is purely exotic in its origin, every outbreak in this country has, at least since 1745, been traced to a foreign source.

In 1745 the disease — according to Professor Simonds — was introduced into England, either by two white calves which were purchased in Holland, by a farmer residing at Poplar, near London, or by a quantity of contaminated hides which had been purchased by a tanner at Zealand. Be this as it may, the disease, after its introduction, made rapid strides, and remained for a much longer period amongst us than in any subsequent visitation. It was not exterminated until the year 1757.

The second outbreak, and the most destructive in our day, occurred in the year 1865, and continued its ravages through the greater part of 1866. The disease at that time was introduced by a cargo of cattle from Revel, brought by the *Tonning*, and landed at Hull on the 29th of May. The original intention of the owners being—according to Mr. Gamgee—to land them at London.

These cattle, it was stated, had been purchased in Esthonia, but it was known that they had been mixed at Revel with a lot of infected cattle from the interior of Russia, and it was even thought that a few of the latter were imported along with them.

From Hull the infected animals, as also a number of sheep which were brought over with them, were sent in the usual course of trade to London, Leeds, and Manchester, and from these—and probably several other—centres the disease was widely disseminated. Curiously enough, it was conveyed from the South to the North by cattle which had been forwarded from the neighbourhood of London to Aberdeenshire.

The loss which was inflicted by this outbreak upon British farmers was incalculable. One estimate, that of Professor Gamgee, placing it as high as 233,629 head of cattle.

The third visitation of the disease was in 1872, and it was introduced into three ports—viz., Deptford, Hull, and Leith—almost simultaneously, from Cronstadt. The *Joseph Soames* arrived at the Humber Dock, Hull, with fifty-eight oxen, on the 25th July; the s.s. *Benachie* arriving in Leith roads with fifty oxen, early on the morning of 23rd July.

The cattle on board the *Joseph Soames* were allowed to stand in the dock for two days, within a short distance from the cattle market, and with constant communication shorewards; they were subsequently killed, and the carcases placed in a lighter, the hatches of which, it is stated, were improperly battened down, and as a result of this negligence they were set at liberty and washed ashore. Another and very different explanation of this matter is, that after the lighter was scuttled, no provision was made for the escape of gas from the interior, and consequently when the carcases decomposed the accumulated gases burst the lighter open. Whichever of these may be the true explanation, they equally teach an important lesson in the manner of dealing with the carcases of diseased animals.

No cattle were landed at Hull, either alive or dead, yet the disease spread by mediate contagion to no less than three districts—viz., Bridlington, Patrington, and Pocklington—before it was stamped out.

The *Benachie* was boarded at 5 A.M., on the 23rd, by the late Mr. Romanes, M.R.C.V.S., ex-inspector for the Port of Leith, who found that three of the animals comprising her cargo were suffering from Rinderpest, and who immediately took measures to prevent the vessel being brought into the harbour, and to obtain confirmatory evidence of his opinion.

On landing from the tug which had conveyed him to the *Benachie*, Mr. Romanes put himself in telegraphic communication with the Veterinary Department of the Privy

Council, with the Local Authority, and with the Custom House Authorities; but it was not until the following day—Wednesday—that he received any definite instructions as to how he was to dispose of the unwelcome visitors. About 2 P.M., on the 23rd, I proceeded on board the vessel with Mr. Romanes, Professor Williams, and Mr. Baird, and after making a collective examination of the animals, we confirmed the opinion of Mr. Romanes, as to the nature of the malady from which they were suffering; at that time three oxen were badly affected, one in fact was dying, and a number of others showed premonitory symptoms.

On Wednesday afternoon Mr. Romanes received definite instructions as to the method of dealing with the cargo—viz., to slaughter all the living animals on board the vessel, to disinfect the healthy carcases, and bring them ashore for the purpose of sale, and to throw the diseased animals, with the hides and offal of the healthy ones, overboard at a distance of not less than fifty miles from the port.

The vessel steamed out to a point off the Isle of May, with Mr. Romanes, Mr. Bird (then my assistant at the Dick College), and several fleshers—the work of killing being proceeded with *en voyage*. One animal died on the 23rd, and another at 6 A.M. on Thursday, by which time thirteen others evinced unmistakable signs of the malady.

The diseased animals were knocked on the head, their abdomens ripped open, their skins slashed, and, with the hides and offal of those which were healthy, thrown overboard; the carcases of the healthy animals were brought back to Leith, with the object of landing them for sale, but owing to a subsequent order from the Privy Council Office they were again sent out to sea and thrown overboard.

A similar occurrence to that which took place with the Hull cargo happened with this also. The carcases were washed ashore, at different points, for many miles along the north and east coasts of the Firth, and caused much alarm amongst the occupiers of the neighbouring lands; but the disease was fortunately checked, though the occurrence here mentioned took place within a comparatively short space of time after the animals were slaughtered; indeed they were observed, by Mr. Bird, floating with the head uppermost—owing to the neglect of the precaution to slash the diaphragms—on the return voyage.

The particulars of the fourth visitation of Cattle Plague, occurring so recently as 1877, must be fresh in the mind of all persons interested in the subject, nevertheless I have thought it advisable—in order to preserve the links in the chain of its history—to introduce here a short account of the outbreak—extracted from the Annual Report to the Veterinary Department of the Privy Council Office for 1877, by Professor G. T. Brown:—

"In the first week of January, 1877, Cattle Plague was detected in Upper Silesia among some oxen which had been smuggled from Poland. The official veterinary surgeon recognised the first case of the disease on 5th January. Immediate action was taken by the authorities to arrest its further progress. Sixty-nine cattle were

slaughtered in the two villages where the disease had appeared; but from the fact of five animals having died before the slaughter could be completed, it was evident that the outbreak had occurred some time before it was discovered. It subsequently appeared that infected cattle had stood in the cattle market at Breslau on 8th January, and probably on a previous market day (30th December). On 10th January infected cattle were in the market at Altona, and on 15th January Cattle Plague had extended to the market in Dresden. From these great centres of cattle traffic the propagation of the disease continued in spite of the most stringent precautionary measures.

"The Hamburg-Altona market was, it appears, infected by cattle brought from the Berlin market, and some of the animals which were exposed in Hamburg market on Wednesday, 10th January, were taken to the stables of Gorris and Wieck, where Professor Köhne detected Cattle Plague on Saturday, 13th January, the day after cattle from both these stables had been shipped to England in the two ships *Castor* and *Hansa*.

"A telegram announcing the outbreak at Altona was received at the Veterinary Department on Monday, 15th January, at one o'clock in the afternoon, three hours after the cattle brought over in the *Castor* had been landed at Deptford, and twenty hours after the landing of the cattle from the *Hansa* at Hull.

"Information of the outbreak of Cattle Plague at Hamburg was telegraphed from the Veterinary Department to the inspectors of the Privy Council at the various ports, directly the existence of the disease was known, in order that they might exercise extraordinary care in the inspection of cattle and sheep from Germany; but owing to the delay which occurred in the transmission of the intelligence from Germany, the inspectors did not get the warning in time to enable them to intercept the vessels, and inspect the animals on board. In fact the two cargoes which introduced Cattle Plague into this country had been landed before the Privy Council was informed of the existence of the disease in Hamburg, and the facts relating to the shipment of the infected cattle were not ascertained until some time afterwards.

"The *Castor*, with forty cattle on deck, left Hamburg on Friday night, 12th January, and arrived in the Thames on Sunday, 14th January. Instead of proceeding at once to Deptford, the vessel lay off Gravesend all night.

"On the arrival of the vessel at Deptford on Monday, 15th January, Mr. Holmans, the inspector, was present; and his attention was attracted to the animals in consequence of the extremely wretched appearance which they presented. One of them indeed died on the landing-place, and others were in a dying state.

"On a cursory inspection during the landing it was ascertained that some of the cattle were suffering from Foot-and-Mouth Disease in the advanced stage, and this added to the effects of a prolonged voyage accounted for the prostrate appearance of the cattle.

"After the animals were placed in the lairs in block No. 1, the inspector made a further examination of them, and found distinct indications of Cattle Plague.

"Steps were immediately taken to isolate the animals. The block in which they were placed was locked, and orders were given that no persons should be admitted.

"On the following morning Mr. Holmans reported the facts of the case to the Veterinary Department. The Chief Inspector accompanied him to Deptford, and confirmed his opinion as to the nature of the disease. Arrangements were accordingly made for the slaughter of the animals, and the destruction of the carcases in the iron digesters which are provided in each block for this purpose. And in order to provide against the reintroduction of Cattle Plague from Germany by means of sheep, an Order was passed, on 16th January, to apply the regulations of the Fourth Schedule to sheep and goats brought from any German port, and similar provisions were afterwards extended to sheep and goats from Belgium and France. These restrictions on the importation of sheep and goats from Germany, Belgium, and France remained in force during the remainder of the year 1877.

"When I visited the lairs at Deptford for the purpose of ascertaining what steps were being taken to dispose of the animals, I found that the one digester in the block where the cattle were confined was being used to destroy the carcases of several cattle which had died; but on being informed that the use of the three digesters would enable the men to effectually destroy all the animals in thirty-six hours, I gave Mr. Holmans instructions to adopt this plan. In the meantime the Markets Committee had decided, rightly according to law, that the carcases could not be moved out of the infected place (block No. 1), and therefore the other digesters in blocks 2 and 3, although within easy reach, and so placed that they might have been employed with perfect safety, could not be made available without infringing the provisions of schedule six of the Contagious Diseases (Animals) Act. Under these circumstances, the process which might have been completed in a few hours, occupied several days.

"During the time that the slaughter of the diseased cattle was being carried into effect, every precaution was taken to prevent the conveyance of the poison outside the boundaries of the market; and my own conviction is, that the means employed were effectual, and that the real mischief was done before the nature of the disease was known.

"First, there was the unfortunate but quite unavoidable circumstance of the removal of the vessel in which the diseased cattle had stood for many hours, from Deptford to another landing-place for the purpose of landing passengers; and then a further removal to another wharf to land miscellaneous cargo. Next, the passengers, crew, and cargo had been dispersed in all directions, carrying the poison with them; further there were the drovers and other persons who had been engaged about the cattle before and after they were landed. All the people had gone to their homes and occupations in utter ignorance of the deadly powers which they had unwittingly acquired.

"The danger, indeed, was evident enough, the means of averting it not quite so

clear. No measures which would have interfered with the cattle trade in this country would have been tolerated at this period. Cattle affected with plague had been landed at Deptford before, and no harm had happened; and it would have been deemed quite unnecessary to impose onerous restrictions to meet an emergency which might never arise.

"All that could be done was to have the ship cleansed and disinfected as soon as possible. For this purpose the Customs were asked to decline to give the final clearing note until the inspector reported that the disinfection was finished.

"In justice to the owners of the ship it must be stated that they afforded every assistance to the officers engaged on the work, and voluntarily gave an undertaking not to carry cattle in the vessel for a month.

"With the view to meet the possibly coming disease, the Local Authorities were informed, cautioned, and advised in the terms of the following circular, which was at once despatched from the Veterinary Department: —

"'17*th January*, 1877.

"'SIR,—I am directed by the Lords of the Council to inform you that Cattle Plague has broken out at Hamburg, and that a cargo of animals affected with the disease has been landed at Deptford, in the port of London.

"'I am to suggest that the inspectors of your local authority should be instructed to make strict inquiries as to the existence of any unusual form of disease among animals in their districts.

"'In the event of any doubtful case occurring, it is desirable that the advice of a qualified veterinary surgeon should be obtained without delay.—I am, SIR, your obedient Servant, (Signed) C. L. PEEL.'

"To what extent the Local Authorities acted on the suggestions contained in this circular cannot be accurately stated. In a few instances the receipt of the document was acknowledged, and the Veterinary Department was informed that the inspectors had been instructed to report the existence of any unusual form of disease in their districts; but nothing in the direction of a systematic inquiry was ordered, and in no instance was an original outbreak of Cattle Plague detected in any district in consequence of any action which was taken by a Local Authority.

"While measures were being taken to prevent, as far as the circumstances allowed, the extension of Cattle Plague from Deptford, an inquiry was proceeding in reference to the removal of the infected cattle from the premises where the disease existed in Hamburg to the ship in which they were conveyed to England. A document dated Hamburg, 12th January, 1877, officially sealed and signed by the inspector at Hamburg, certified that the forty cattle intended for shipment to London in the ship *Castor* had been that day examined and found healthy; on the following day Cattle Plague was declared to exist among cattle in the sheds from which the forty animals had been

taken. On the third day after shipment all the animals landed from the vessels were suffering from Cattle Plague, some of them were dying of the disease, one had died on the voyage, and another fell dead on the landing-place. All the evidence was opposed to the idea that the cattle could have been free from indications of disease when they were put on board the vessel. Great things had always been predicted of the veterinary police organisation in Germany, it was not therefore unfair to ask for an explanation of the palpable failure which produced such disastrous results.

"Owing to the representations of the English Government, the German Government made an investigation respecting the shipment of the animals, and the result was the removal of the Hamburg inspector from his post.

"At the time of the landing of the *Castor's* cargo at Deptford no suspicion was entertained that cattle from the same premises at Hamburg had been sent to Hull. It was known that cattle from Hamburg had been landed at Hull and at Hartlepool before the landing of the *Castor's* cargo, but the inspectors at those ports did not detect any signs of Cattle Plague among them. It happened, however, that the inspector who had charge of the *Castor* while it was being disinfected heard one of the officers express his surprise that nothing had been heard of the other lot of animals which went to Hull. On this hint a further inquiry was made at Hull, but nothing was discovered beyond the fact that two cargoes had been landed from Hamburg on 12th and 14th January respectively. Foot-and-Mouth Disease was detected among the cattle in both cargoes, and consequently the sheep as well as the cattle had all been slaughtered within the defined part of the port. The investigation was now extended to the port of shipment, and the information was obtained that the *Leopard*, which landed her cargo at Hull on 12th January, brought forty-four sheep from Wieck's stables, and the *Hansa*, which arrived on 14th January, brought eighteen oxen from Gorris's stables. With the fact before us that Professor Köhne had declared the existence of Cattle Plague on both premises on 13th January, it was at least satisfactory to know that all the cattle and sheep had been slaughtered in the defined part of the port a few days after being landed.

"Beyond the indications of Foot-and-Mouth Disease, from which some of the beasts were suffering, the inspector at Hull observed nothing of importance until the second day after they were landed, when two of the cattle were found to be ill, the chief symptom being a loud grunting or roaring. On *post-mortem* examination no characteristic lesions were detected, but there is now little doubt that the animals were affected with Cattle Plague, the peculiar grunt being the special symptom of the early stage of one of the many phases of the disease. With the slaughter of the infected cargoes it was hoped that the danger of an extension of the disease had been averted, and attention was chiefly directed to the prevention of its reintroduction from abroad.

"On 27th January, owing to the continued spreading of Cattle Plague in Germany, an Order of Council was passed prohibiting the landing of cattle, fresh meat, hides, and

other animal products from Germany and Belgium, and subsequently, on a report of the appearance of Cattle Plague in the town of Emden, the regulations of the Fourth Schedule were applied to cattle, sheep, and goats from The Netherlands. These Orders remained in force at the end of the year.

"OUTBREAK OF CATTLE PLAGUE IN THE METROPOLIS.

"During the time that precautions were being enforced to prevent the reintroduction of Cattle Plague from the Continent, the disease was already existing in this country. The two infected cargoes which were landed in London and in Hull had done their work most effectually, although for some time the mischief was not discovered; and had it not happened that the owner of the diseased cows in Gill Street, Limehouse, was foiled in his endeavour to dispose of his stock, the disease might have assumed serious proportions in London before it was detected.

"The first intimation of the existence of Cattle Plague in a cow-shed in the Metropolis was received on 30th January, fifteen days after the landing at Deptford of the diseased cattle from Hamburg.

"OUTBREAK OF CATTLE PLAGUE IN HULL.

"After the expiration of a month from the time of the landing of suspected cattle from the *Hansa* at Hull, there was good reason for believing that the slaughter of the whole of the cargo of cattle and sheep in the defined part of the port had prevented the extension of the disease; but on 17th February a rumour was current that Cattle Plague had appeared in a dairy in the town. On the following day a telegram was received from the Inspector of the Privy Council stationed at Hull confirming the report, and the Chief Inspector of the Veterinary Department was immediately instructed to proceed to Hull for the purpose of making inquiry as to the origin of the outbreak, and advising the authorities in the emergency.

"Information of the existence of Cattle Plague in Hull was received on Sunday, 18th February, and as the cattle market in Edwards Place is held on Monday, it was most important that some steps should be taken to prevent the distribution of the animals exposed in that market over the country.

"On the representation of the Chief Inspector the Local Authority at once decided to have all the cattle in the market marked as foreign cattle, and sent into the defined part of the port for slaughter. The store stock market to be held on the following day was prohibited. An inspection of all the dairies in the town was ordered, and the movement of cattle in the district except with a licence was forbidden. This prompt and decided action on the part of the Local Authority probably prevented an extension of the disease.

"On the discovery of Cattle Plague in a dairy in Hull, it was apprehended that

the disease might have been recently introduced from Holland. An inquiry into the circumstances, however, soon led to the abandonment of this idea, and removed the difficulty which was experienced in endeavouring to connect the outbreak in the middle of February with the cattle which were landed from the *Hansa* in the middle of January. It will appear from the Chief Inspector's report that there is good evidence of the disease having invaded the dairies at a much earlier period.

"In the Hull outbreak, as in the Metropolis, the channel through which the virus was conveyed to the dairies was not discovered, but it may be safely asserted that some of the cattle which were landed from the *Hansa* were the subjects of Cattle Plague when they were slaughtered, and that some person who had been in contact with them, probably one of the butchers or drovers, carried the infective matter to the cow-sheds in the town, while in the act of removing a cow for slaughter.

"As soon as the existence of the disease was established the diseased animals and those in the shed with them were destroyed, a considerable area round the infected premises was defined by the Local Authority, and the movement of any animals out of the cow-sheds in the area was prohibited.

"Further outbreaks occurred in the Hull dairies up to 22nd March, when the disease ceased for a considerable period and was believed to be extinct, but on 4th May a report was received that Cattle Plague had appeared in a dairy in Lincoln Street, Sculcoates, some distance from the previous outbreaks.

"As the district had been free from Cattle Plague for about six weeks, and there was no reason to suspect that it had been reintroduced, it was reasonably doubted if the affection which had appeared in Sculcoates was really Cattle Plague; and an inquiry was immediately instituted. Mr. Duguid, of the Brown Institution, whose services had been placed at the disposal of this Department by the Royal Agricultural Society, was instructed to proceed to Hull and investigate the case. He reported that one of the animals was affected with Cattle Plague, and the Local Authority at once acted on his report, and ordered the slaughter of the diseased animal and the four which were in the same shed, a course which was certainly most prudent under the circumstances, although the destruction of the healthy cows which had been exposed to infection rendered it impossible to prosecute the inquiry further, or to obtain the absolute evidence which is afforded by the progress of the disease through a herd.

"In the entire absence of any evidence as to the origin of the outbreak in Sculcoates after a long interval, it was a matter of scientific importance to identify the disease, but the Local Authority could hardly be expected to incur the risk of the spreading of infection, while a problem in veterinary pathology was being worked out.

"At the suggestion of the Veterinary Department another inspection was made of all the dairies in Hull, in order to ascertain if any animals had been removed since the last inspection, or if anything had occurred to lead to the conclusion that Cattle

Plague had existed in the interval between 22nd March and 4th May, but nothing was discovered to give rise to a suspicion that undetected cases of the disease had occurred during that period.

"No extension of Cattle Plague occurred from the infected premises in Sculcoates, and on 2nd June the district was declared free from the disease, and the restrictions which had been imposed by the Local Authority on the movement of cattle and the holding of markets in the town were revoked."

GEOGRAPHICAL DISTRIBUTION.—Cattle Plague is a well-known disease in Russia, Asia, most European countries, and India. According to Mr. Fleming ("Sanitary Science and Police," Vol. I.), it appears in Mongolia, China (south and west), in Cochin China, Burmah, Hindostan, Persia, Thibet, and Ceylon.

So far as I know, it has never been seen in the Colonies.

VITALITY OF THE VIRUS.—In Great Britain no opportunities have occurred to enable veterinarians to arrive at any definite conclusion on this point, but from Continental practitioners we have some reliable information as to the length of time which the virus is capable of retaining its viruliferous properties. Thus, in "Veterinary Sanitary Science and Police," Mr. Fleming says:—"In Belgium, in 1868, Cattle Plague reappeared in two localities in the province of Antwerp, eight months after the last cases had been observed."

"Kept in carefully-closed capillary tubes, the virus will retain its power for a long period, or, lodged in closely-packed hay, it may remain potent for months. Salchow gives an instance in which the disease appeared after the use of hay that had lain in an infected stable for a year. Muller cites an outbreak due to giving hay which was contaminated five months before; and the remains of carcases (skin, flesh, &c.) buried for thirty days, and even three months, have preserved their contagiferous properties.

"A temperature of 131° destroys it, but putrefaction up to a certain point does not seem to have any effect on its vitality, as was shown by Vicq d'Azyr, who successfully inoculated with the remains of cattle which had been interred for three months."

PERIOD OF INCUBATION.—It is, I think, universally agreed amongst professional men—home and foreign—that the average incubatory period of this disease, when contracted naturally is about five or six days; a few veterinarians extending the period, in occasional cases, from ten or twelve to twenty-four days.

When artificially propagated, the period of incubation is usually shortened by about twenty-four to forty-eight hours.

INVASION.—Is usually, at least in exotic attacks, marked, though in some instances it is insidious, and notably so in those countries in which the disease is indigenous. In my experience, invasive indications have been pronounced, although I have never seen them so violent as they would appear to have been observed by some authorities.

The violence of invasion is no doubt largely regulated by the power of individual

animals to withstand the effects of the poison, by the virulence of the virus, by the amount received into the system, and by the method of introduction.

In some animals the vital force is so strong as to enable it to keep in abeyance the multiplying and disturbing powers of a zymotic poison for a very long time, while in others, again, no opposition is offered by the *vis vitæ* to the action of such virus.

Duration.—In the most malignant forms of the malady, death takes place within a very short period—sometimes thirty-six or forty-eight hours after the first symptoms have been observed.

On an average, duration may be stated at from two to eight or nine days, and, as a rule if an animal rallies over the fourth or fifth day, more favourable hopes of a recovery may be entertained.

Fatality.—No disease affecting cattle is so fatal as this, particularly in some outbreaks, and amongst animals which, from causes already considered, are peculiarly susceptible to the action of the poison, and whose systems are incapable of withstanding its effects; and the fatality is rendered greater by the fact that there is such a liability to *relapse*.

In many cases animals progress favourably for a few days, and have all the appearance of recovering, when, quite suddenly, the symptoms become aggravated, and death follows within a few hours.

ORIGIN AND PROPAGATION OF CATTLE PLAGUE.

It is supposed to originate spontaneously in the Asiatic steppes, at least all European scientific traditions lead to this conclusion, though it is combated by Russian veterinary surgeons, some of whom have, in the past, made strenuous efforts to trace the disease to its home; indeed, even now the Russian Government, with the aid of competent veterinarians, are endeavouring to discover from whence the disease comes, but, as yet, their efforts have not been rewarded. As I have already said, tradition points to the steppes as the original home of the disease, but, whether tradition is right or wrong, it is a well-known fact, and one which has been recently enlarged upon by Mr. Fleming, that most of the extensive outbreaks of the disease in Europe have been associated with, or have followed upon, the mobilisation of Russian troops, and the scourge of most wars in which Russia has been engaged has been added to by the spread of this virulent malady.

By some authorities it is believed the disease occasionally originates elsewhere than in the Asiatic steppes, but whether this belief is well founded or not, it is a fact that in a few countries the malady is, pretty well, a permanent guest, and it is well known that from these infected localities it frequently extends over the frontiers of neighbouring States. When once originated, or introduced into a district,

Cattle Plague, owing to its being propagated in such a variety of ways, makes rapid and certain strides.

The extraordinary methods of extension or propagation are—1stly, Through the medium of the offal, flesh, or blood of affected animals; 2ndly, By means of the excreta, saliva, and buccal mucus gaining access to streams or water, or contaminating food or litter; 3rdly, By inoculation with the catarrhal, nasal, or conjunctival discharges; 4thly, By the injection of the blood of diseased into the veins of healthy animals; 5thly, By the conveyance of milk from infected byres; and 6thly, Through the agency of living fomites.

VAGARIES IN THE SPREAD OF THE DISEASE.

As I have already indicated, remarkable divergencies from ordinary rules of propagation are occasionally seen in this as in all allied maladies.

It is a curious, but well-known fact, that during the visitation of 1865-6 Ireland—in spite of the direct and indirect communication constantly kept up between that country and Great Britain—entirely escaped the malady. Only one or two counties in Wales were visited by the disease, although it was extremely prevalent in most of the adjoining counties of England.

While Ireland was preserved from the ravages of the disease, cattle from that country readily contracted it, and in their subsequent journeyings spread it far and wide; a drove of these animals—depastured on the banks of the Tern by a local dealer, within a short distance of Market Drayton—being accredited with its introduction into Shropshire and one or two adjoining counties.

During the two days in which the s.s. *Benachie* lay in Leith Roads in 1872 constant communication was kept up by drovers and others between the vessel and cattle grazing in the vicinities of Leith and Edinburgh—and cattle, too (Icelandic and Danish), which were in a highly favourable condition for the reception of the virus, owing to many of them being weak and debilitated, and suffering from the effects of a rough and protracted sea voyage—and yet the malady did not spread beyond the confines of the infected steamer; neither, in the case of the Leith and Hull cattle, did any ill effects follow the washing ashore of the infected carcases. Doubtless the latter fact is easily accounted for when we consider the disinfecting properties of sea water.

SYMPTOMS AND COURSE.

In considering the general characters of Cattle Plague, I enlarged somewhat upon the fact of its presenting such a variety of aspects with regard to the particular sets of organs attacked, and its manner of comporting itself in different outbreaks. Those

remarks apply equally to the manifestation of symptoms, not only in any single epizöoty of the disease, but also in individual cases.

It is, no doubt, owing to these peculiarities of Cattle Plague that so many different opinions as to its precise nature have, from time to time, been expressed, and that so much difficulty has arisen—in its early stages—in arriving at positive conclusions as to its identity.

The PREMONITORY SYMPTOMS are not always observed, or are not attended to, as they are often but slightly developed, and do not differ from those of other contagious diseases.

Elevation of temperature—from two to three degrees—is the most common and, if the disease already exists in a herd, the most reliable premonitory sign; it has been observed in so short a period as from thirty-six to forty-eight hours after inoculation.

Symptoms are both CONSTITUTIONAL and LOCAL, and, for the convenience of description, are divided by most authors into stages—usually three—but in some cases the disease runs such a rapid course, as to render such division arbitrary if not impracticable.

In the first stage the symptoms are mainly constitutional. Elevation of temperature is constant; the pulse is slightly increased in frequency, and may be somewhat irritable; the respiration is a little quicker than normal, though, in many cases, this is more apparent than real, and is produced by the jerking action communicated to the flanks by involuntary—occasionally violent—muscular twitchings and shivering.

The external temperature, especially of the extremities, horns and ears, is variable; the back arched; the legs more or less approximated under the abdomen; the bowels constipated; the lacteal secretion in milch cows diminished; in nursing mothers there is indifference to, or absolute loss of affection for the offspring; rumination is partially or totally suspended; the appetite fickle; the spirits depressed; and the hair erect.

In those cases in which the primary force of the poison is expended on the nervous system, there may be more or less delirium, or involuntary and spasmodic muscular movements. Some animals bellow loudly (though they do this under other circumstances) and evince great excitement and irritability. In other instances, colicy pains, with uneasy movements of the hind limbs are shown, and there may or may not be increase in the normal mucous secretion of the nostrils, eyes, and external generative organs. A slightly irritable or husky cough may be noticed in this stage with general stiffness in motion and great unwillingness to move.

Positive local symptoms are not usually developed in the first stages of the disease, though, if the mucous membrane of the external generative organs and the rectum is examined, it will be found somewhat heightened in colour; there may be also a little increase in the salivary secretion.

In the second stage the symptoms, both constitutional and local, are considerably more developed. The excitement of the first stage will have given place to marked

depression, but if the nervous centres are much interfered with and delirium has been present it may still persist; all the natural functions are more or less suspended; the milk is diminished in quantity—if not actually suppressed—thicker in consistence, and, while its salts are lessened, the fatty matter is increased, the oil-globules being aggregated; considerable thirst may be evinced; the head, eyelids, and ears are drooped, the tail lax; pulse increased in frequency, 70, 80, 90, and weaker; respiration quickened but not laboured, though the expiratory effort is sometimes prolonged and accompanied by a peculiar grunting or roaring sound, which is also occasionally heard in the earliest stage; temperature, 104-7°, being higher at night than in the morning; the bowels are usually relaxed if there is not actual diarrhœa, and, in bad cases, the discharges possess the peculiar fœtor so characteristic of the disease, and, though usually of their natural colour, they may be much darker than normal and even tinged or streaked with blood; their voidance, though voluntary, is sometimes accompanied by tenesmus, and colicky pains, more or less severe, may be evinced; the urine is generally diminished in quantity, and high coloured, it may contain blood, albumen, or lymph casts, and, owing to the increased combustion, is usually loaded with solids.

The throat is frequently very sore; a cough of a low, husky (bronchitic) character is usually present, and results from exudation of lymph on the laryngeal and bronchial mucous membrane; there is a flow, more or less profuse, of saliva from the mouth; a peculiar agglutinous discharge, of a fibrinous character, issues from the inner canthus of the eyelids, and frequently collects or coagulates in the form of jelly or synovial-like masses—sometimes of a considerable size—of a yellow colour, with similar coagula prolonged from them down either cheek; discharge of a less fibrinous character, however, than that from the eyes is seen from either nostril, and there may be excoriation of the skin and mucous membrane around the nasal apertures, and of the eyelids. The mucous membranes generally are much injected, and if cutaneous eruption is about to become developed during the course of the disease, it may make its appearance in this stage. It is characterised by the formation of roseolous patches, which vary in extent (sometimes attaining to a dimension of many inches), in situation, and in depth of colour. The cutaneous eruptions, which are often developed on the skin of the inner surface of the thighs and on the udder, consist, according to some pathologists, of inspissated exudates from the glands and follicles, and of pathological changes in the more superficial structures. According to the Third Report of the Cattle Plague Commissioners, 1866, the eruption is first roseolous, secondly vesicular, followed by the formation of crusts and dessication; and, as shown in Fig. 29, Plate VII., frequently localised in cows around the base of the teats.

I am informed by Mr. Hallen, commissioner of the stud department at Hapur, that in India cutaneous eruption is considered favourable, as when it is profuse dysentery seldom prevails, and recovery is more rapid and frequent.

The local signs of Rinderpest are very pronounced in this stage, and are developed in the mucous membrane of the mouth, vagina, and sheath.

I have already indicated that the buccal membrane is, in the first stage, and especially where it is thin, increased in vascularity; this pink colour gives place to blanching of the epithelium—notably of the papilla of the cheeks—and to the formation of the characteristic eruptions, the most frequent and earlier sites of which are the inner surface of the lower lip, on a line with the junction of the lip and gums, and on the papillated inner surface of the cheeks—subsequently, in prolonged and severe cases, the dental pad, hard palate, and tongue are involved. (See Plates.)

The aphthous patches are formed by the rapid proliferation, degeneration, and desquamation of the epithelium; are irregular in shape, and variable in extent. They are sometimes described as vesicular in character. I have hitherto failed to associate them with such eruptions.

The first noticeable alteration in the buccal membrane, after blanching, is thickening of the epithelium, quickly followed by solution of its continuity and elevation in minute scaly masses, discrete at first but subsequently coalescing to a greater or less extent. The aphthous products bear a very close resemblance to minute particles or flakes of blanched oatmeal, and, as they crack, extremely red or scarlet lines are formed in the interspaces owing to inflammation of the underlying membrane: as the epithelium becomes removed, hyperæmic patches are formed (Fig. 19-21, Plate V.) If not removed by the movements of the tongue or lips, a little friction with the finger nail or the back of a knife easily detaches the aphthous collections.

Vaginal eruption is nearly as constant as buccal, and is—with the exception that there is a more marked tendency to miliary vesication—identical in character. The aphthous patches in this situation are usually developed close to the junction of the mucous membrane with the skin of the vulva (Fig. 22, *b*, Plate VI.) They are arranged in a vertical direction, and are associated with great increase of the mucous secretion, which is abnormally tenacious, and frequently depends from the inferior commissure of the vulva as a glairy rope-like mass (Fig. 22 *c*, Plate VI.)

Eruptions in the sheath are much less frequent than in the vagina; they are, however, of much the same character, developed on the extremity of the sheath, the prepuce, and the penis; and are associated with a mucous, which is rapidly replaced by a muco-purulent, discharge, possessing, like that from the vagina, a distinctive odour.

In the third stage.—The constitutional symptoms are increased in intensity and gravity. The pulse frequently runs up to 100-120, is very weak, often tremulous or indistinct, and irregular; the respiration is usually laboured and quickened, and accompanied by a peculiar action of the flanks, the production of which has been variously attributed to disturbance of the respiratory nerve-centres, to swelling of the mucous membrane of the glottis, and to pulmonary emphysema; it may be due to either of these causes individually or to the whole collectively, but, in my opinion it is

more frequently due to emphysema of the lungs than to any other single cause—respiration is almost always accompanied by a peculiar hollow grunt, or a characteristic miserable moan.

The temperature will vary with the tendency to death or recovery; if the former, it lowers—after attaining its greatest elevation—rapidly; if the latter, gradually.

The breath becomes very offensive—the odour being pathognomonic of the disease. The mucous discharge from the mouth is viscid, largely mixed with aphthous productions, and often streaked with blood from rupture of the extremely congested superficial capillaries; gnashing of the teeth is, in many cases, incessant; the expression of the countenance miserable in the extreme; the ears completely lopped; retraction of the eyes in the orbital cavities marked; the cough more weak and husky; the skin is tight, and exhales a fœtid odour; the hair erect, harsh, and loosened in its follicles, the cutaneous eruptions becoming—according to some authors—pustular in character, the pus inspissating and forming scabs, while others describe it as consisting simply of profuse desquamation of the cuticle, with dried serous effusion.

In every case there is great disinclination or unwillingness—often total inability—to move—simulating paralysis; emaciation is very rapid, and is due to a combination of circumstances—viz., to the great chemical changes going on in the system from the accompanying high fever, to the outpouring of fibrin by the mucous surfaces, to the anorexia, and to the destruction of the intestinal villi, and, consequently, non-assimilation of nutrient material. The latter cause is also very frequently in operation to produce the prolonged emaciation which is such a grave accompaniment of convalescence.

The intestinal discharges are often expelled involuntarily, but sometimes tenesmus becomes so extreme as to lead to eversion of the mucous membrane of the rectum, which now possesses a characteristic mahogany hue, and its surface is more or less ecchymosed, eroded, and excoriated (Fig. 23, Plate VI.); excoriation of the skin of the anus and perineum also results from the acrid nature of the fœces.

The odour of the alvine fluids is extremely—and pathognomonically—offensive. They are sometimes dysenteric, at others partake of the characters of rice-watery dejections, and are often mixed with the aphthous products of the mucous membrane.

The eruptions on the skin of the nasal apertures frequently extend to the Schneiderian mucous membrane, but in many instances independent ulceration of this structure takes place, converting the nasal discharges into muco-purulent matter, which is sometimes tinged with blood; on examination, too, ecchymoses are frequently seen in this membrane.

If auscultation of the chest is had recourse to, dry crackling, emphysematous sounds, with sonorous mucous *râles*, are heard; while on percussion, hyper-resonance is marked, and where the emphysema forms subpleural tumours a cracked-pot sound is elicited.

As the animal stands, the legs are drawn together under the body, and the hind fetlocks knuckle over.

Emphysematous tumours are developed in the lumbar region, the character of which is easily discernible on manipulation; the production of emphysema is variously explained, some authors attributing it to the development of gas in the connective tissues, others to infiltration of atmospheric air from the lungs.

I cannot comprehend the possibility of gas being formed *ante-mortem* in the tissue of an animal except in those blood diseases which are characterised by the rapid decomposition of the vital fluid, and, even in these, local extravasation precedes the evolution of gas.

I am firmly convinced that the sublumbar emphysema is mainly, if not entirely, due to the infiltration of air from the lungs, owing to laceration of the tissues at the root of these organs.

Abortion, in pregnant animals, is more often seen in this than in the preceding stages.

Death is produced in a variety of ways—from exhaustion of the vital forces, failure of the heart's action, and asphyxia, the latter frequently being a result of pulmonary emphysema.

In some cases dissolution is accompanied or preceded by convulsive symptoms, followed by coma; in others, no such conditions are produced, the life of the diseased animals gradually and slowly ebbing away without the slightest sign of struggling or resistance.

The local symptoms in this stage are simply an aggravated condition of those which have been described in the second. If no relief is afforded, the exposed mucous membranes ulcerate or remain intensely hyperæmic.

If the case tends towards recovery, the local as well as the constitutional symptoms ameliorate; the hyperæmia of the injured membranes disappears; the exposed parts become covered by new epithelium, and the mucous secretion is restored. Desquamation of the cuticle is profuse, and the skin gradually regains its usual sleek condition. The action of the heart becomes firmer; the pulse fuller, slower, and more regular; the respiration more natural; the mucous membranes regain their normal colour; the animal becomes more lively; and, in milch cows, the secretion of milk returns. If a small quantity of blood is abstracted it will coagulate, the fœcal discharges lose their dysenteric or rice-watery character and return to their usual consistence, colour, and odour. The appetite is restored, and, after a time, rumination is re-established.

Relapses are tolerably frequent, and in many instances are attributable to neglect or exposure, and to the injudicious and too early allowance of solid food; they are nearly always fatal.

In sheep and goats the symptoms and course of the malady differ from those in the ox only in being less intense. The disease, too, is not nearly so fatal.

PROGNOSIS.

Looking at the fact that practitioners are not now permitted to treat this disease, it will perhaps appear at first sight a somewhat superfluous proceeding to devote a single line to the consideration of prognosis; but, remembering that some veterinary surgeons may be located in countries where treatment is permissible, I have thought it best to make a few remarks on the point.

In the first place, it is necessary to observe that prognostications cannot here be founded on the rules which usually guide us in other zymotic affections. It has been already stated that some cases of Cattle Plague in which invasion is sudden and violent and the symptoms unusually well marked, are more likely to recover than where the opposite conditions obtain, and, further, that cutaneous eruption is looked upon—at least in some countries—as favourable to recovery.

In these respects Cattle Plague simulates Distemper in the dog and Foot-and-Mouth Disease.

Marked attacks—*i.e.*, those in which the visible pathognomonic manifestations are but slightly marked—are often the most treacherous and the most fatal. Doubtless, the use of the thermometer and a careful examination of the pulse in such cases will show that the fever is high, and that, too, in inverse ratio to the pronounced symptoms.

The amount of interference with the natural functions and cerebro-spinal system of nerves, the presence or absence of pulmonary emphysema, the character of the fœcal discharges, the amount of fever, the general intensity of the attack, and the condition of the victim, form tolerably good bases upon which to found our prognosis; to this may be added the length of time the disease has been in existence, whether we have to deal with an enzoötic or exotic outbreak, and the kind of care and attention bestowed upon the patient.

DIAGNOSIS.

I think I need scarcely observe that the proper diagnosis of such a malady as the one under consideration is one of supreme importance, not only to the practitioner, but to the public and the State. The ultimate consequences of an error in this direction cannot be calculated, and bad though it may be to condemn animals where condemnation is unnecessary, it is infinitely worse to be guilty of the slightest laxity, whereby diseased or contaminated animals may escape detection, and be allowed to spread the malady broadcast.

In inland districts the risk of a wrong diagnosis may be thought to be less than in parts where foreign animals are landed, but such, I imagine, is not really the case.

The urban practitioner—especially if he has never seen a case of Cattle Plague—

has not his suspicions aroused by the same train of circumstances, perhaps slight in themselves, which would claim the attention of the seaport inspector. Moreover, unless he is a close reader of daily newspapers, the former does not often know from what point or points danger may be apprehended, while the latter is always, or should be so, posted up in the most reliable and recent intelligence as to the whereabouts of the dreaded enemy.

Undoubtedly, under very exceptional circumstances, diseased animals may escape the notice of the inspector and attract the attention of the general practitioner who may be called upon to examine them on account of the development of suspicious and unaccountable symptoms.

The danger of a wrong diagnosis is not so much in connection with any particular zymotic or sporadic disease, pure and simple, but with complications of two or more diseases.

The collateral circumstances which may bias the mind of the port inspector are—1stly, Discrepancies between the manifest of a cargo and the actual number and characters of the animals composing it. 2ndly, The loss of several animals of a cargo *en voyage*, with signs of illness amongst those which have been landed. 3rdly, Doubts as to whether the animals composing a cargo have been brought direct from the port named in the manifest, or been in contact with other animals between the point of departure and their ultimate destination. 4thly, The fact that Rinderpest is manifested in such a variety of ways, and that its symptoms are sometimes so deceptive. The suspicions of any practitioner may, of course, be aroused by the prior existence of the disease in a district. The conditions under which this affection is so liable to be confounded with others of a less serious nature are, so far as my experience goes, as follows :—

(*a.*) Foot-and-Mouth Disease, in which foot lesions are absent, but in which eruptions exist in the vagina, sheath, or lower lip.

(*b.*) Foot-and-Mouth Disease, in which foot lesions are absent, broncho-catarrhal or muco-enteric—or both—symptoms present.

(*c.*) Foot-and-Mouth Disease complicated with enteric fever.

(*d.*) Malignant catarrh, especially if complicated with emphysema of the lungs.

(*e.*) Broncho-nasal with intestinal catarrh, and accidental buccal abrasions.

(*f.*) Sea-sickness with intestinal catarrh.

(*g.*) Variola ovina.

(*h.*) Acorn poisoning.

With regard to Foot-and-Mouth Disease, the well-known fact that it may coexist with Rinderpest, and that the two maladies are frequently introduced from the same country, will tend, in some degree, to bias the mind and render a proper decision more difficult.

Of the three conditions—*a.*, *b.*, *c.*—which I have mentioned as associated with

Foot-and-Mouth Disease, the latter is the most misleading; but, in either case, the exercise of a little patience, with close observation, will soon settle the question, as the milder nature of Eczema—with the rapid development of pathognomonic signs in the same or other animals—will quickly become apparent.

The exact circumstances under which Foot-and-Mouth Disease, complicated with enteric fever, will be best understood by the relation of a case in point.

In 1872, when the s.s. *Benachie* lay in Leith Roads with her cargo of rinderpest animals on board, a number of Iceland cattle—recently imported—were depastured on Bruntsfield Links, Edinburgh—intercommunication being kept up by the attendants in charge.

Early one morning, several days after the *Benachie* cargo had been disposed of, one of these animals was found dead, and several others showed symptoms of severe illness; the carcase of the dead animal was removed to the slaughter-house, and examined by the city inspector and the inspector of the abattoir, both of whom considered the appearances presented so suspicious as to cause them to seek my aid. I found all the *post-mortem* characteristics of enteric fever, complicated with miliary vesicular eruption on the extremity of the sheath and the penis.

On examining the animals on the Links I discovered a bull with miliary vesicular eruptions on the sheath and aphthous eruption on the mucous membrane of the lower lip. Several other animals exhibited similar conditions, but in a less degree.

As I thought it highly probable that the disease would develop into Eczema Epizootica, the expression of a positive opinion was deferred until the following day—the suspected animals being carefully isolated—when the usual symptoms of that disease were pronounced in a number of animals.

About the time of the decline of Cattle Plague in 1866 two mistakes—one at Liverpool, the other in Cheshire—were made in the diagnosis of enteric fever, and gave rise to much local alarm and interference with trade.

During life the two affections are most likely to be confounded with each other when enteric fever attacks several animals simultaneously, and when the cutaneous eruptions which sometimes accompany it are developed; but inasmuch as the disease often runs it course so rapidly as to preclude the possibility of the practitioner seeing its victims during life, I have thought it best to introduce a brief sketch of its pathognomonic necroscopical signs.

As in Rinderpest, the lesions are to be sought for in the mucous membrane of the stomachs and intestines. They are diffused and circumscribed hyperæmia and extravasation, with ulceration and sloughing.

The colour of the membrane varies from a simple inflammatory blush in the early to a dark mahogany hue in the later stages, the congestion and inflammation being most intense in the prominent borders of the gastric and intestinal folds. It may be

confined to the fourth stomach and small intestine, or may extend through the whole length of the alimentary canal.

The extravasations vary in extent from the size of a vetch seed to several inches in circumference.

The ulcers and sloughs are characteristic; they commence on the site of an extravasation or quite independently of it, and, from being at first discrete, rapidly extend, become confluent, and assume an irregularly elongated outline; their edges become everted and of a light-brown hue, the sloughing membrane being of a darker colour, closely resembling cassel leather, and separated from the surrounding structures by a line of demarcation composed of a belt of hyperæmic vessels and by a segregating chasm. In some instances the sloughing process does not extend deeper than the submucous tissue; in others, it involves the muscular and even the peritoneal coat, in which case the latter contracts a dark hue and becomes thick and opaque.

Perforation of the intestine sometimes results, while at others the integrity of the organ is preserved by the deposition and organisation of successive layers of lymph, which may, however, in their turn, degenerate and produce secondary perforation.

The ulceration in enteric fever is deeper and more extensive than in Cattle Plague; sloughing is more pronounced; and there are usually no bronchial complications, though, as in all affections of the alimentary mucous membrane, there may be interference with the nutrition of the buccal epithelium, and, as a consequence, superficial erosions with hyperæmia of the mucosa.

The muscular tissue and the blood are usually dark in colour in severe cases, and the contents of the intestines are of a blackish hue, fœtid, and often tinged with blood. The structure of the spleen and the mesenteric glands may be engorged, pulpy, and granular, and the latter dark in colour.

Malignant catarrh complicated by pulmonary emphysema has many points, both *ante* and *post-mortem*, of resemblance to Cattle Plague, particularly when aggravated by the lowering effects of a long journey, and by impure air in a ship's hold.

The particulars of a case of this kind which came under my notice in 1876 will illustrate the difficulties sometimes experienced by inspectors, as also the modifications produced in the characters of a disease by the influence of adverse circumstances.

The s.s. *S*—— arrived at Leith early one Tuesday morning from Copenhagen, with a cargo of cattle and sheep; the former, according to the manifest, numbering 201, the latter 133. The cargo was landed at 8 A.M., the number of cattle being 199, and sheep 132. One bullock had died on the voyage and had been thrown overboard, and one sheep was in such a condition as to necessitate its removal to the abattoir for immediate slaughter.

On proceeding with my assistant, Mr. Jones, M.R.C.V.S., to make my final inspection at 7.30 on Thursday morning, we found one ox so ill as to render it neces-

sary to have it removed, in charge of a constable, to the slaughter-house. The *post-mortem* examination—made a few minutes after death—in conjunction with the symptoms during life, showed strong evidence of the existence of Rinderpest amongst the animals. On the evening of Wednesday another animal, a cow, was observed to be very ill, and on Thursday morning she was no better.

Several circumstances combined, made matters seem very suspicious.

In the first place, The number of cattle landed was short of the number in the manifest, and the same occurred with regard to the sheep.

2ndly, Many of the animals were very lean, had a miserable appearance, and did not appear to be Danish cattle at all.

3rdly, The consignee afterwards voluntarily told me that some of the cattle in the cargo had been brought from Sweden *via* Malmo and Copenhagen.

The animal which I had slaughtered on Wednesday was first noticed by me when landed on Tuesday morning; he was on his way to the lairs, and he then had a haggard appearance, and was breathing rapidly. I examined him, and found that he had been abused and beaten, and to this fact I attributed the rapidity of the respiration. I saw all the animals on Tuesday evening and observed them closely, but, with the exception of a general unhealthy condition of many of them, there was nothing to attract special attention.

On Wednesday morning the ox above mentioned presented the following symptoms—quick, weak pulse, spasmodic breathing, dejected countenance, cold extremities, tremors, peculiar fœtor of breath, extensive ulceration and sphacelus of the Schneiderian mucous membrane, superficial ulceration of the lower lip, temperature 101°, and, on auscultation, emphysema of the lungs. All the symptoms were indicative of speedy dissolution.

By consent of consignee's representative, the ox was taken to the abattoir and slaughtered.

The *post-mortem* revealed extensive interlobular emphysema of both lungs—patches of ulceration in reticulum, numerous hyperæmic patches in the omasum, a general dark blush of the mucous membrane of the abomasum, with numerous hyperæmic circles and intracircular ulceration; ecchymoses, great prominence of mucous follicles, with bands of yellow lymph on antrum pylorus and ecchymosis of pyloric orifice; diffused inflammatory blush of cœcum, which contained a quantity of dark-coloured fluid and fœtid excreta; congestion of rectum, with bands of viscid mucus and numerous small masses of coagulated blood; fœces black, fœtid, semi-fluid, pasty, and mixed with scibulæ; muscles dark and ecchymosed in patches; blood grumous, red corpuscles crenated; aphthous collection in nostrils—the epithelium undergoing granulo-adipose degeneration.

On Wednesday night a cow was observed breathing rapidly, eyes sunk, pulse seventy-six, temperature 104$\frac{2}{5}$° F., mucous discharge from vulva, and ecchymosis of the

mucous membrane of the labia; grunt in expiration, and bulging of left chest. This animal was slaughtered on Thursday night, the *post-mortem* examination revealing extensive suppurative peritonitis, diffused patches of inflammatory blush in the intestines, and œdema of mucous membrane of fourth stomach, with thickening of the coats of the reticulum, through which a nail was found protruding, and which satisfactorily accounted for most of the symptoms and *post-mortem* appearances; but the symptoms in conjunction with previous circumstances, gave matters a very suspicious appearance.

During Wednesday and Thursday several animals were discovered breathing quickly, temperature 103°, and in one case 103⅔°, with arched back, staring coat, superficial muscular tremors, and fœces black and pasty. On Friday morning I made a final examination in company with Inspector Wilkinson, and, finding that no fresh symptoms had developed, and that those that had existed had passed off, I released the cargo.

I may remark that the head, stomach, &c., of the ox were shown to my colleague Mr. Baird, and to the inspectors of the Local Authorities of Edinburgh and Leith—all having large experience in Cattle Plague—and they each agreed that the case was extremely suspicious.

I may also add that I incidentally discovered that the ox which was thrown overboard was ill when shipped, and that within a few minutes after death the odour which emanated from the carcase was unbearable.

Broncho-nasal and intestinal catarrh with accidental buccal abrasions—particularly in foreign animals, and in those which have been exposed and allowed to drink sea-water—may cause the tyro to make a wrong diagnosis.

Under these circumstances, there is frequently profuse nasal and conjunctival discharge, injected conjunctivæ, diarrhœa, tremors, arched back, and bronchitic cough; the discharges, however, are purely catarrhal, the pulse is only increased about ten or fifteen beats per minute, the respiration—though somewhat hurried—is never spasmodic, the depression of spirits is less than in Rinderpest, the temperature seldom exceeds 103° or 104° F., the normal functions are not interfered with to any great extent, and the fœcal discharges are nearly natural in colour and odour. The buccal abrasions are readily referrable to their true cause by the characters already alluded to in speaking of Foot-and-Mouth Disease.

In sea-sickness with intestinal catarrh the depression is often extreme; the fœces possess a somewhat sickly odour; the membrane of the mouth is more or less injected, hot, and furred, and the mucus imparts an unpleasant odour to the skin of the examiner's hand; there is much shivering, the pulse is increased in frequency and weak, and the appetite impaired; but a few hours' rest and a little careful nursing restores the patient to health.

I have already remarked that, in so far as the general characters and nature of Smallpox in sheep are concerned, such a wide difference exists as to render it unnecessary

to dwell on the subject at any length ; here I need only remark that although the constitutional symptoms are closely allied to those of Rinderpest, the local lesions are of a very different nature, the eruptions of sheep-pox passing through several defined stages, viz., the roseolous, papular, vesicular, pustular, ulcerative, and desiccative ; and extending over a period of about twenty-eight days.

Sheep-pox does not under any circumstances attack cattle, while it is highly improbable that Rinderpest would exist in sheep without the cattle of the district being likewise affected.

Acorns—acting as an irritant poison—produce a train of symptoms and a few *post-mortem* appearances somewhat akin to those of Cattle Plague. Thus we have gastro-enteritis as evidenced by primary constipation, secondary diarrhœa—the discharge being fœtid and of a dirty colour—offensive breath, and abdominal pains ; the pulse is frequent and weak, the eyes glazed, the normal functions suspended, and there is extreme depression, with moaning, gnashing of teeth, and, in some cases, brain and spinal disturbance. The *post-mortem* lesions are those characteristic of muco-gastro-enteritis with patches of extravasation, ulceration, and even gangrene—the presence of acorns among the ingesta sets all doubts at rest.

PATHOLOGICAL ANATOMY.

The necroscopical characters of Cattle Plague vary, perhaps, more than those of any other zymotic affection, hence in the description of them by different authors, marked, and sometimes almost irreconcilable, discrepancies exist.

As with the symptoms, so here, authors have taken the characteristics of particular outbreaks as the type of the disease under all circumstances, and have dogmatised on this false basis.

Thus, Peyer's patches have been variously described as being the seat of ecchymosis, as having lost their integrity, as sloughing and ulcerating, and as pale, contracted, and more depressed than usual. The solitary glands again have been referred to as being congested, enlarged, and even sphacelated by some authorities, while others look upon any change observable in them as a purely negative one, and one which may be seen in the glands of many healthy animals.

Conflicting statements of this kind, as also the further contradictory ones in reference to the existence or non-existence of true ulceration in the gastro-intestinal mucous membrane, can only be reconciled by the facts that none of the characteristics of the disease—either *ante* or *post-mortem*—are constant, that they vary with the different stages of the affection, the condition of the blood and system generally at the time of the attack, and, to a certain extent, with the character of the food ; that they are modified by the subjection of the animal to treatment, by the abstraction of

blood prior to or at the time of death, by the time the carcase has lain after dissolution, by the state of the weather, by the use of disinfecting agents for the purpose of preservation, by the age of the victim, and by the prior existence of organic intestinal or gastric disease.

Hence, as regards the gastro-intestinal mucous membrane, we find the organic lesions varying from simple loss of epithelium to erosions, ulcerations, and sloughs; and in depth of colour, from a slight, superficial, inflammatory blush, to a deep mahogany hue, which may extend even to the submucous tissue.

In considering the *post-mortem* appearance, it will be as well to glance at the general condition of the systemic tissues, organs, and fluids in the first place, and at the state of the organs which are more particularly involved in the second.

Although the condition of the muscular tissue, the blood, the milk, and the serous membranes is tolerably characteristic, it is by no means pathognomonic—is, in fact, to a great extent, and in particular instances, purely negative, and may be observed in many affections in which the nutrition of the blood is primarily or secondarily interfered with.

The muscular tissue may (owing to the modifying circumstances already mentioned) present the magenta hue and albuminous glaze referred to in speaking of Pleuro-pneumonia; it may be nearly or quite normal, or of a dark (carbonaceous) hue with an iridescent glint on section, as shown in Fig. 8, Plate X. Its firmness and state of moisture will also vary considerably according to the influences which have been exerted upon it, either before or after death. In any case, a rapid modification in its characters is observed when a fresh section is exposed to the atmosphere, particularly if the latter is dry and ozonic.

Small, microscopical, mobile, protoplasmic masses, composed of an aggregation of small round cells, have been discovered in this tissue; they are technically termed *psorosperms*, and from the fact that he first described them, "*the Cattle Plague bodies of Beale.*"

These protoplasmic bodies are not peculiar to Rinderpest; they have been discovered in muscular tissue under a variety of circumstances.

The blood-vessels throughout the body are in the advanced stages, and especially in animals which have succumbed to the disease, charged with black semi-fluid blood, though it may form a coagulum of tarry consistence and colour. The integrity of the vessels—particularly the capillaries—is largely interfered with; hence (independently of the effects of stagnation and engorgement) the readiness with which they give way and allow of the extravasation of their contents. Capillary congestion is, universally, marked. In addition to its being of a dark colour and more or less fluid in consistence, the blood is altered in its histological characters: the fibrin, depending upon the stage of the affection and the intensity of the diseased processes, may be increased or decreased in quantity and altered (usually deteriorated) in quality; consequently, if the

blood is withdrawn during life, it clots with tolerable firmness in the early stages and in convalescence, but remains more or less fluid during the height of the disease; the red corpuscles are often shrivelled, crenated, or even partially disintegrated; the white corpuscles increased quantitatively and presenting modifications in their contents, in size, and in motile properties; a tendency to aggregate is also sometimes noticed in the red corpuscles. The soluble matter of the blood, according to Beale, is increased in quantity, and the serum is stained by colouring matter (hæmato-globulin).

All the tissues of the body—the muscular, the serous, the connective, and the nervous—may be the seat of effusions, exudations, and extravasations. The substance of the brain and spinal cord is sometimes softened and of a dark colour, and the vessels injected or even ruptured; at other times no appreciable departure from the normal state can be detected. If brain and spinal symptoms have been pronounced during life, important and extensive cerebro-spinal changes will be found after death.

Emphysema of the sublumbar connective tissue, and probably of the connective tissue elsewhere, will be observed in those cases in which pulmonary emphysema exists.

The small quantity of milk which remains in the ampulæ and lactiferous ducts is usually much thicker in consistence than normal milk, often of a dark or yellow colour, sometimes tinged with blood, at others viscid; the globules, as a rule, show a great tendency to aggregate, and are in some instances intermixed with granule corpuscles—in fact, the milk presents much the same characters as in Foot-and-Mouth Disease (Fig. 6, *a*, Plate XI.)

The muscular fibres of the heart are often pale in colour and flaccid; and, as in fevers generally, the sarcous elements may be somewhat granular and cloudy.

Its cavities—particularly the right—are frequently filled with masses of imperfectly-coagulated dark-coloured blood, and occasionally with pale-coloured *ante-mortem* clots. Extravasation of blood underneath the endocardium, especially on the left side, is by no means uncommon, and in some instances is very extensive.

The liver and spleen may be normal or engorged with blood, soft and friable with extravasation under the capsules, and even where these organs appear to be normal careful examination will frequently show that there is more or less organic change in their structure.

The kidneys, except being somewhat pale and flaccid, or congested, with ecchymoses in their structure or under the capsule, do not generally present important structural alterations.

The lymphatic glands—particularly the gastro-intestinal ganglia—are usually somewhat enlarged and softened, and their cell elements show a tendency to degenerative—granular or fatty—changes; these cell changes have also been observed by some inquirers in the liver and kidneys.

The mesenteric glands may be engorged with blood, soft and friable; they may be simply congested, or apparently healthy.

The structures more particularly involved in the morbid processes in Cattle Plague are the skin, the mucous membranes of the alimentary, respiratory, and urino-genital tracts ; and the intestinal glands.

The skin does not present other changes than those which may be observed during life—viz., irregular roseolous patches, papulæ, vesicles or pustules, with proliferation and softening of the epidermis, congestion of its capillaries, and thickening as the result of exudation. Of course, if blood has not been abstracted at the time of death, the vessels of the subcutaneous connective tissue will be found engorged with dark blood, drops of which will ooze from the cut capillaries, and form red streaks on the carcase.

The mucous membrane of the mouth will also present those changes which have been observed during life, the destructive processes, as already observed, not extending deeper than the mucosa itself; the papillæ of the tongue may be swollen, and of a livid hue, or pale and somewhat shrivelled.

The tonsils are sometimes enlarged and clinging tenaciously to their openings, as also to the orifices of the glands of the fauces, there may be masses of thick viscid mucus of a pale colour, or masses of lymph—croupous exudation—of a yellow hue. Congestion, ulceration, and sloughing of the faucial membrane, with extensive ecchymoses, is described by some authorities. Similar changes to those just noticed are also seen in the mucous membrane of the pharynx, and even where they are not observed it will be found more or less thickened and softened, and its epithelium altered in character—*i.e.*, granular or adipose, or both.

The œsophagus—owing to its denser structure and its less vascularity—presents no structural changes of any importance, at least any alterations observable do not extend deeper than the epithelium, which may be softened, and easily detached from the membrane.

The character of the contents of the rumen will depend upon the length of illness and the kind of material upon which the animal has been feeding. If the food has been succulent, decomposition and fermentation may be progressing owing to the arrest of the vital functions; if, on the contrary, it is agglutinous, it will be found rolled up into masses, particularly in the pouches. As to the stripping off of the epithelium, to which attention has been so much directed by some writers, it is purely a negative sign, and always takes place when the food lies in the stomach for any length of time, either before or after death. Diffused congestive patches are seen in the mucous membrane, and where such exist the papillæ are primarily congested and enlarged, secondarily shrivelled and atrophied. In some instances absolute necrosis of the membrane, which assumes a dirty brown colour, and sloughing (Fig. 25, Plate VI.) with ulceration, takes place.

In the reticulum there is nothing of great importance to be found—patches of congestion occasionally exist, and even erosion.

The lesions of the omasum are by no means pathognomonic of Rinderpest; they are annular patches of congestion of an irregular shape, with central sloughs, such as I have described in speaking of the pathological anatomy of Foot-and-Mouth Disease, and as shown in Fig. 26, Plate VII. The contents of this stomach, unless death has taken place in an unusually short period, are usually dry and caked, and the surfaces of the cakes are marked or pitted by the papillæ of its folds; the epithelium, as in the rumen, adheres to, and strips off with, the cakes of ingesta.

The changes observed in the abomasum are of greater importance and far more distinctive than those seen in the other gastric compartments. The mucous membrane of the folds of the stomach presents a variety of alterations, according to the stage of the disease in which it is examined. Superficial congestions, proliferation, and softening of the epithelium, with rapid degeneration and shedding producing erosions; are seen in the earlier, while deep-seated congestion, ulcers, and even sloughs are seen in the later stages. Many of the lesions in this situation bear a strong resemblance to those produced by corrosive poisons, the prominent borders of the folds presenting the greatest evidence of organic change. The colour of the mucous membrane in this portion of the abomasum varies from a slight red blush to a deep mahogany hue.

In the pyloric portion (antrum) more important changes are usually seen. Here, however, as in the folds, they are not constant. In the case of the cattle which were slaughtered on board the *Benachie*, numerous oval, dusky, or rose-coloured spots, very similar to those seen in Foot-and-Mouth Disease, about the size of a croton bean, and having depressed surfaces, existed in the earlier stages; and running between these spots, and linking them, as it were, together, were to be seen irregular white lines about the sixteenth of an inch in breadth, the structures comprising which were softened, friable, and opaque; in the more advanced stages solution of continuity was established, producing cracks (rhagas), through the mucous coat revealing the muscular fibres—slightly separated from each other—beneath. These cracks resembled, at first sight, those produced by an incision with a knife, but differed from them (on closer examination) by the structures composing their borders being of a whiter colour and opaque; by the edges being less abrupt, not inverted, and firmly connected with the submucous tissue. On looking into the fissures produced by the knife, the fresh fat, which usually lies between the muscular and mucous coat, is distinctly visible, and the muscular fibres are of their normal character.

The structures comprising the rose-coloured spots mentioned, from being simply hyperæmic, lose their integrity, undergo molecular degeneration, and are cast off, leaving superficial ulcers having a red margin, with numerous new vessels radiating from their borders, as seen in Fig. 27, Plate VII. The ulcers ultimately cicatrise.

The white lines and subsequent cracks map out circumscribed areas of the mucous membrane, which, in some instances, become deprived of their vitality, and are cast off as sloughs (Fig. 28, Plate VII.); in other instances the membrane undergoes no such

important change, but remains simply congested or ecchymosed. Collections of viscid mucus or of lymph are more often seen on the mucous membrane of the antrum than on that of the folds.

In the small intestines the lesions—allowing for differences in structure—are very similar to those seen in the abomasum. They are distributed with tolerable uniformity over the duodenum and ilium. Hæmorrhages (Fig. 24, *c*, Plate VI.) are usually present, while the solitary glands, becoming enlarged and prominent, produce nodular elevations on the surface of the mucous membrane (Fig. 24, *b*, Plate VI.), though hypertrophy of these glands is not at all uncommon in old cattle, particularly if they have suffered from an attack of muco-enteritis.

The changes which Peyer's patches undergo are not constant, variations being marked with the different stages of the disease, and in different epizootics.

It is, I think, pretty well agreed that in the earlier stages of the affection they are somewhat tumefied, and consequently slightly raised above the level of the surrounding membrane, but that in the more advanced stages they become atrophied (shrivelled), paler in colour, and depressed; such a condition is depicted in Fig. 24, *a*, Plate VI. In some instances aphthous deposits in the early, croupous in the later stages, are found on these patches, and they have even been described as undergoing molecular degeneration and mortification.

The deposits on the surface of the mucous membrane of the small intestines generally have been looked upon by some authorities as of a diphtheritic character; those which I have had the opportunity of examining were croupous. That the more superficial portions of the membrane undergo a degenerative process in many instances is unquestionable, and in this condition they assume a greyish or yellowish (Fig. 24, *d*, Plate VI.) hue, but this is quite independent of the yellow exudations alluded to.

The dark mahogany colour, described as existing in the abomasum in the advanced stages of the disease is even more pronounced in the ilium and duodenum, especially at the pyloric extremity of the latter; and, what is more, the blood pigment, from being retained in the tissues and subjected to the action of the intestinal fluids, imparts to the membrane, more or less permanently, a variety of hues.

Several authors—notably Klebs—have described a form of micrococcus as being found in large numbers in those parts of the intestinal and gastric mucous membrane in which the lesions are most pronounced, while Beale directs attention to the large quantities of germinal matter existing in the cells and tissues.

The large intestines present no lesions calling for particular comment; the cœcum and the rectum are generally the seat of the greatest changes, which do not differ materially, except in the absence (usually) of sloughs, from those observed in the small intestines. The contents of the colon in the earlier stages are usually pasty in consistence, dark in colour, and possess the peculiar odour already alluded to as being exhaled from the fœcal discharges during life; the same remarks equally apply to the contents

of the rectum, and, with the exception of their being as a rule fluid, to those of the cœcum also.

In the respiratory tract we find, in the first place, considerable tumefaction and vascular engorgement of the nasal mucous membrane, and in severe cases and advanced stages, hæmorrhages and ulcerations with extravasations and exudations into the facial sinuses; these conditions persist in the mucous membrane of the posterior nares, the fauces, the larynx, trachea, and bronchia, though ulceration extends with comparative rarity beyond the larynx. Croupous exudations, giving the membrane a yellowish tint, are tolerably constant.

The lung-structure, except in malignant cases, is not much altered; it may, however, present ecchymoses, and, if emphysema exists, the lobules will be more or less compressed and of a somewhat florid hue. In the Third Report of the Cattle Plague Commissioners, 1866, a representation of emphysematous lung is given of an extremely red colour. I have sometimes seen these organs blanched, usually, however, the appearances presented are much the same as those shown in Fig. 3, Plate I. When an animal has succumbed to the disease without abstraction of blood, the lungs are frequently the seat of hypostatic congestion—especially the lobes which have been lying undermost—and are then increased in weight and size and charged with blood of a dark colour.

TREATMENT.

Although medical treatment of animals affected with Cattle Plague is now prohibited, and wisely so too, I can scarcely pass over the matter without according to it a little notice.

Whatever system of treatment may be adopted, the percentage of deaths will always far outnumber that of recoveries; and if a history could be compiled of the various nostrums, specifics, and therapeutic agents—the filthy compounds of onions, garlic, asafœtida, tar, &c., the more secret and infallible (?) "cure alls," and the scientific remedies of the regular practitioner—which were employed for the cure of this disease during the outbreak of 1865-6, I fancy it would make up a volume of such dimensions as few amongst us would care to wade through.

In the case of the few animals I had the opportunity of treating before the stamping-out system was ordered, I adopted several methods of treatment, none of which appeared to me to give so much relief as the early administration of a laxative—sufficiently strong to overcome constipation—followed by tincture of benzoin, chloric æther, camphor and cinchona in weak brandy and water two or three times daily, and in the course of a day or two allowing a liberal supply of cold milk mixed with beaten-up raw eggs, and arrowroot, starch, or fine oatmeal gruel. In addition to these, I had the whole of the body enveloped in hot rugs, covered over by waterproof sheets. I need scarcely

say that the withholding of all solid food is a *sine quâ non* in the treatment of this and all allied affections, even though the patient may evince a ravenous desire for such food. It was my experience, and I believe that of most other practitioners who had opportunities of treating the disease, that whenever solid food such as hay, though in small quantities only, was allowed on the approach of convalescence a fatal relapse was the inevitable result.

Sedative emollient enemas—particularly cold milk—are extremely useful, as are also judicious doses of chlorate of potash, or one of the sulphites ; probably the salicylate of soda would be of great value during the progress of the disease, while liquor arsenicalis would assist in restoration to health.

The remote consequences of Cattle Plague are chronic gastro-intestinal ulceration, stricture of the small intestines from contraction of the cicatrices where ulceration has been deep and extensive ; nasal gleet ; chronic ulceration of the larynx ; thickening or constriction of the pharynx, producing difficulty in swallowing ; chronic pulmonary emphysema, and sloughing of the mucous membrane of the stomach and intestines, the disintegrated tissues being passed out with the fœcal discharges. Masses—sometimes cylindrical casts—of exudative matter are frequently expelled by the rectum ; and, owing to the destruction of the intestinal villi, emaciation and debility are often extreme and prolonged, leading, in some cases, to death by inanition.

PREVENTION AND SUPPRESSION.

Any remarks bearing upon the subject of Prevention must necessarily refer to the importation of foreign animals.

The danger of *direct invasion* comes, of course, from those countries in which Cattle Plague is enzoötic.

The danger of *indirect invasion* is from infected cattle being smuggled through countries whose ports are open to our commerce, and where strict inspection is not enforced; or through the medium of fomites.

Of the danger of *direct invasion* we had a good example (to which I have referred) in 1872. This danger, however, is now reduced to a minimum.

The first step taken towards realising this minimum was in the reduction of the number of ports at which animals from scheduled countries could be disembarked. The second and most important step was in the recent vigorous action of the Privy Council in prohibiting the importation of cattle from scheduled countries, immediately the disease was known to exist in them. It may be said, and justly so, too, that if importation of live animals is permitted at all from such countries as Germany, there will be still great danger of the disease being introduced, and the outbreak of 1877 may be quoted in support of the argument ; nevertheless, it is hardly possible that animals could be

brought from any distance without Rinderpest—supposing it to exist amongst them—declaring itself either during the voyage, or while they are kept alive in the sheds for the purpose of slaughter, and I am satisfied that the Privy Council Inspectors will now be too much on the alert to let the slightest suspicious circumstance escape their observation.

It has been suggested that if importation is allowed at all from scheduled countries, inspectors of our own appointing should be stationed at the ports of embarkation. Doubtless such a system would still further minimise the danger of invasion, especially if the British Consuls and inspectors were instructed to keep themselves thoroughly posted up as to the existence of Cattle Plague in neighbouring States; but, on the whole, if the slightest suspicion or doubt exists, the wiser course is to prohibit the importation, not only of live stock, but everything which is likely to become a medium of introducing the infection.

As to the danger of *indirect invasion* through unscheduled countries, such as Denmark, I think it is a very remote contingency indeed. Perhaps there is no country in the world so jealous and careful of the health of its live stock as Denmark, or in which such strict protective measures are in force. Since I have held the post of Inspector at Leith, I have not seen even a suspicion of the existence of contagious disease amongst Danish, Swedish, Iceland, or Norwegian animals, except the solitary case which I have referred to.

The Danish country people depend so much upon their live stock as to make it an absolute necessity for their Government to take every precaution that human foresight can devise to prevent the introduction of contagious disease into their country.

Into whatever country Cattle Plague is introduced, the most rigorous suppressive measures should be adopted; and from our experience of the last visitation it is plain that the execution of these measures should not be entrusted to any other power than the Privy Council. Many local authorities have, in the past, been extremely lax in their efforts to suppress not only this but other and less important zymotic affections, and in a matter of such moment the Privy Council has done well to ensure for itself absolute and unrestricted power to deal with Cattle Plague in the future as it sees best, and not to trust to less responsible and feebler bodies; but while saying this, I cannot help expressing the opinion that it would have been better had greater power been placed in the hands of port inspectors than that which is conferred upon them by the "Foreign Animals Order of 1878." Certainly, inspectors have absolute power so far as isolation is concerned; nevertheless much valuable time might be wasted, and risk of mediate contagion run, while the inspector is telegraphing to the Privy Council Office for instructions, especially if the responsible authorities should be out of reach for a few hours. The way in which delay may endanger a district was well exemplified—though happily the danger was averted—in the case of the *Benachie* cargo, and the still greater danger arising out of adherence to forms of law, was strikingly shown by

the difficulty which arose at Deptford in the destruction of the carcases of the *Castor* cargo, as related at pp. 9, 10 of the Blue Book for 1877.

A knowledge of the manner in which an enemy comports himself is of the greatest advantage to the combatant, and the experience gained in 1866 and 1877 has not only taught us what weapons to use in dealing with it, but that no temporising measures are of avail in suppressing Cattle Plague, and that it can only be annihilated by the absolute destruction of every living animal which is the subject of the disease or which may have been in contact with affected cattle; and not only so, but by destruction, when practicable, of everything which is contaminated or may be supposed to be contaminated with the virus of the disease; or by thorough disinfection when destruction is impracticable.

I imagine the *fiasco* of vaccination will never again be revived in Great Britain, and the adoption of curative measures would be as insane a proceeding as it is to attempt to treat a rabid dog or a glandered horse; in fact, infinitely more so, as attempts to cure rabies are more than justified by the benefits which would accrue to human sufferers if a cure could be discovered.

I need not here detail the measures which should be adopted for the suppression of Cattle Plague, as they will depend largely on the peculiarities of individual outbreaks; but I may be permitted to remark that if the same energetic measures which were had recourse to for the arrest of the last visitation were applied to Zy. p. p., it would soon cease to be one of the bovine scourges of Great Britain.

SCROFULA—TUBERCLE.

BEFORE entering into the detail of this section of my task, I deem it necessary to make somewhat extended introductory remarks, as the nature of the affection precludes the possibility of treating it in the same systematic manner as ordinary zymotic diseases.

In August, 1872, I had the pleasure of reading an essay on the subject of Tubercle, at a meeting of the West of Scotland Veterinary Medical Association. The subject-matter of that essay was the outcome of much thought and study and of an extensive practical experience; nevertheless, on perusing the remarks I then penned, I find that I entertained many opinions as to the nature of this disease, which, by the light of subsequent research and the investigations of pathologists, I must acknowledge were crude and erroneous; neither can I, even at the present day, affirm that the nature of Tubercle is an open book to me. The researches of pathologists are conducted on very different principles to those which were followed a few years ago, and their conclusions are based upon much more reliable foundations; nevertheless we are accustomed, every now and then, to read ideas embodying such startling contradictions, and to see theories expounded which totally upset old notions, as to make us sometimes wonder if the truth will ever be arrived at.

Much, however, has been done by patient investigation and hard work to elucidate the exact nature of the disease.

One of the most important changes which my ideas have undergone is in reference to the relation which Tubercle bears to Glanders in the horse.

At page 672 of the *Veterinarian*—in the issue for September, 1872, of which Journal the essay above referred to was published—I find myself stating that "Miliary Tubercle is found in the lungs of the horse as an accompaniment of Glanders;" and, further, expressing the opinion that "Glanders is nothing more than a form of Tubercle." Do I entertain this opinion now? Far from it. Glanders is a disease *sui generis*. Tubercle is equally so. Again, in the same essay, I find myself asserting that "no domestic animal is free from it [Tubercle] in some form or other." I am

not prepared to endorse that statement now, and although, so long ago as 1864, I recorded a case of Tubercle in the horse—*vide* January number, *Veterinarian*, 1864—I am free to confess that, since I have gained a clearer insight into its characters I have not seen a particle of any morbid product in this animal which I could recognise as Tubercle, and very rarely indeed have I seen it in the sheep or the dog; but while saying this, I am quite conversant with the fact that Tubercle can be propagated in the system of most animals by inoculative contagion.

Of the compartive ignorance which up to a very recent date prevailed, and even now prevails (judging from the simple cases we so frequently see recorded in the veterinary periodicals, and from the tone of many of the communications I have received on the subject) some idea may be gathered from the following remarks which I quote from my essay of 1864 :—

"Mr. President and Gentlemen,—The subject which I am about to bring before you this evening is one of immense importance, not only to veterinary surgeons but to the public generally; to the veterinary surgeon, because no subject offers more food for earnest thought, or more scope for scientific research; to the public, because it has a very close connection with the great social problem of providing our masses with a sufficiency of nourishing and healthy food. To stock breeders and agriculturists it is even of greater import, as by the spread of such a disease—and it is undoubtedly on the increase—their best hopes are rendered fallacious and their profits materially curtailed. To the causes of the spread of the disease I shall have to refer hereafter.

"In proof, Mr. President and Gentlemen, of my assertion that the disease of Tuberculosis has of late years greatly increased, I will call your attention to a remarkable passage in the *Veterinary Record* for January, 1847, p. 29, in which the following note by the editors (in commenting on a case of Tuberculosis, the particulars of which were furnished to them by Mr. W. Cox, of Ashbourne) will be found :—'The tumours attached to the viscera of the thorax of a cow are similar to some we have *occasionally* met with.' Again :

"'On this subject our English veterinary authors are silent, and we believe the above to be the only case of the kind recorded.'

"A quarter of a century has elapsed since these lines were penned, and one only of the three who were responsible for them now remains to bear witness to the vast strides that have been made, during that period of time, in our knowledge of Tubercle and the disease to which it gives rise.

"Other authorities, too, who at this time looked upon Tubercle only as a chance product, have gone to their long home; some of them, perhaps, equally as ignorant of its true nature as they were on the day of their birth."

It is only within the past few years that veterinary surgeons have devoted much attention to the matter, and I think I may justly assert that the essay to which I have referred was one of the first attempts made to bring it prominently before the profes-

sion and the stock owner; since that time much has been written on the subject by Mr. George Fleming, and, by translations of articles by continental veterinarians, he has done still more towards extending our knowledge and affording us a clearer insight into its peculiar characters.

The insidious nature of Tuberculosis has perhaps had much to do with the comparative slowness with which professional and public attention has been directed to it, but the strides which it has made and the hold which it has gained on our stock render it one of the most—if not *the* most—important questions affecting the future well-being of the bovine species.

Looking at an individual Tubercle we might be led to despise its comparative insignificance, and to ignore its deadly meaning; but when we see thousands upon thousands of these knots existing in the organism of a single animal, a truth is forced upon our minds which we cannot refuse to recognise—viz., that we have to deal with an insidious, implacable and deadly foe; and, independently of its ultimate fatality, I think I may with safety reiterate what I have before asserted, that no morbid substance known to the pathologist is so protean as Tubercle in the number of functional derangements to which it gives rise.

Zymotic Pleuro-Pneumonia, Foot-and-Mouth Disease, Cattle Plague, are each in their turns terrible scourges! Are they greater scourges than Tubercle? In each of these diseases we see—patent enough—fearful results both to life and to property; the veriest tyro can tell you that two, at least, cause the death of a great many animals and that the other causes great losses in flesh and milk, but he does not take into account the vast deterioration, the slow but certain decimation of many of our best herds, the destruction of human food, the danger, not—as it is now proved—chimerical or hypothetical, to human life and human comfort, and the insidious progress of that fell destroyer—Tubercle. The other three bovine scourges sweep their victims off in a manner which is seen by all, but the ravages of Tubercle are only realised by those whose duties are connected with public abattoirs, or who are called upon to act as arbiters on the nature of disease.

So little is the importance of the subject realised that our Legislature does not even give it a passing glance in the Contagious Diseases (Animals) Act, and no instructions are issued to our market inspectors as to the way in which they are to deal with tuberculous flesh.

I have already stated that this disease has made rapid strides within the past few years, and seeing that many predisposing causes which operate in the production of the disease now were in existence twenty or thirty years ago, it will naturally be asked, how is this increase to be explained?

I answer, By *multiplied consanguinity, by artificial feeding and manuring, by excessive forcing, by early and over-breeding.*

The very nature of the land has been altered within the last twenty years;

necessarily, its products must be altered, and, consequently also, the tissues of the animals feeding upon these products.

The evil effects of the present system of management are becoming more apparent every day; tissues are rendered prone to degenerative changes by over-forcing—*i.e.*, by feeding largely upon food rich only in material which produces fat instead of good firm flesh, and by over-taxing the digestive and assimilative functions; this degenerative tendency is increased by over-breeding, and perpetuated by in-and-in breeding. The system is fostered by agricultural societies, and it is encouraged by the fabulous and fanciful prices given for fashionable breeds. Rudely is the so-called successful breeder every now and again wakened from his golden dream by one of his best cows failing to breed, by a young bull showing symptoms of tubercular meningitis (often thought to be sturdy), by another animal coughing and gradually wasting away, or another falling with tuberculous joint disease.

As Scrofula and Tubercle are intimately connected with each other, it is necessary that they should be studied together.

The term Scrofula is derived from Greek words signifying "swine" or "sow," and "evil," hence the term "*Swine Evil*" or "*Swine Swelling,*" a synonym dating back to the time of Pliny, and having its origin in a prevalent notion that pigs were particularly prone to the disease. The term "*King's Evil*" is sometimes applied to this condition in human practice; while another synonym, *Struma,* is used, indifferently, with Scrofula.

Scrofula is applied to that constitutional condition in which the tissues are peculiarly susceptible to inflammation, and in which the products of that process when once established tend to undergo retrograde or degenerative changes, and to infiltrate surrounding tissues instead of becoming organised or absorbed as in the case of the products of inflammation in healthy constitutions.

The *scrofulous diathesis*—for it is a true diathetic condition—may be inherited or acquired, and its manifestations are usually evidenced by hypertrophy of the external groups of lymphatic glands, notably those about the pharynx, larynx, and fauces. Inflammation, when once established in these glands by injury or by sympathetic irritation, as in a common cold, runs a comparatively slow course, is very liable to recur, and its products are notably cellular in their character, the comparatively large size of the cell elements precluding—according to Rindfleisch—the possibility of their being removed by the process of absorption; the same well-known pathologist also insists that the pressure produced by the accumulation of these cell elements obstructs, to some extent, the small blood-vessels of the involved tissues, thus depriving the latter of their blood supply and accounting for the rapid manner in which scrofulous exudates undergo cheesy (*caseous*) degeneration.

The relation which Scrofula and Tubercle are believed to bear to each other may be summed up in a few words.

Tubercle is a visible local manifestation of a constitutional diathesis, "Scrofulosis." While this proposition is in the main correct, it must not be assumed that Tubercle does not originate independently of the constitutional condition known as Scrofulosis. From a variety of causes, or rather from a concatenation of circumstances—not yet distinctly understood—tubercular inflammation may be established and the usual local manifestation (Tubercle) be produced; and, in different ways, Tuberculosis can be originated in the systems of animals in which not a trace of Scrofula is to be discovered.

The term Tubercle had its origin in the resemblance which tubercle nodules present to "knots" or "kernels," or in the Latin *Tuberculum* (dim. of *tuber*). This idea evidently permeates even the minds of the common people, who in many parts of England designate the tuberculous growths so frequently developed on the serous membrane of the chest and abdomen, "*grapes;*" while in Scotland the almost synonymous term "*angleberry*" is used to indicate the same kind of product. In many parts of Great Britain the term "*kernel*" is used. In Norfolk *clyer* (a synonym applied to sitfasts on the back of the horse in Scotland) is a common designation.

Before proceeding to the consideration of the origin of Tubercle, its growth, method of dissemination, and results, it will be as well to glance briefly at its anatomical or histological characters, and as this is a special department of pathology, I have taken the liberty of introducing the description of this product given by Dr. Green in his "Pathology and Morbid Anatomy," art. *Tubercle*:—

"HISTOLOGY OF TUBERCLE.—The miliary lesions in acute Tuberculosis, although presenting certain differences according to their age, and to the nature of the tissue in which they originate, are tolerably uniform in their histological characters. Their most marked feature is the prominent place which large multinucleated masses of protoplasm—the so-called giant cells—occupies in their constitution. These large cells, which somewhat resemble the myeloid cells met with in sarcomatous tumours, &c., were long ago alluded to by Virchow, Wagner, and others, but it is only during recent years, mainly owing to the researches of Oscar Schüppel and Langhans, that they have come to occupy a prominent place in the histology of Tubercle.

"The most characteristic features of these multinucleated cells are their large size, the number of their nuclei, and the irregularity of their outline. Some of the larger ones measure as much as $\frac{1}{200}$ inch in diameter. They possess no limiting membrane, but are simple masses of protoplasm, containing numerous round or roundly-oval nuclei, each enclosing a bright nucleolus. As many as forty nuclei may occasionally be counted in a single cell. Some of them are much smaller, and contain only three or four nuclei. Four or five, or even more, of these multinucleated masses are sometimes found in a single tuberculous nodule. Many of these large cells possess long branched processes, in connection with which, and evidently originating from them, are smaller protoplasmic masses, also nucleated and branched. The meshes between

the branched cells are, according to Schüppel, filled with epithelial-like elements. These elements I have failed to observe, and the meshes I have either found empty, or containing a few lymphoid cells. It would thus appear that the original protoplasmic mass gives origin to a network of large branched cells.

"With regard to the histological elements from which the giant cells originate—this varies. In the lung, Dr. Klein states, that they are derived from the alveolar epithelium. They may also probably originate from the cells of connective tissue, and from the endothelium of the blood-vessels and lymphatics. Their formation takes place either by the fusion of two or more cells, or by the excessive development of one cell. In the latter case, the cell increases in size and its nuclei multiply, but here the process of development ceases—there is no subsequent division of the cell.

"Associated with, and surrounding the giant cell and its branches, is a small-celled adenoid-like structure. This small-celled structure, which often contributes largely to the formation of the tubercle, somewhat resembles that of an indurated lymphatic gland, which is commonly known as adenoid tissue. A similar structure, as has been already stated, is also met with in chronic inflammations of the liver, lungs, or other organs. It consists, in the main, of lymphoid cells, which are either separated from one another by fine bands of homogeneous transparent-looking material, or by a more or less distinctly fibrillated, and sometimes nucleated reticulum, within the meshes of which the cells are grouped. This reticulum is sometimes dense and well marked, whilst in other cases it is much less prominent. In addition to the small lymphoid cells there are often seen some rather larger cells containing one, and in some cases two nuclei.

"An elementary tubercle thus consists of a giant-cell reticulum surrounded by, and in direct histological continuity with, a varying-sized zone of small-celled tissue. The larger tuberculous nodules consist of several of these giant-cell systems.

"Although the above characters of Tubercle are to be observed in successful preparations and in certain stages of the tuberculous growth, many of them will often be found wanting. The nuclei and processes of the giant cells will not always be seen, and the cells often appear simply as yellowish, somewhat granular masses in which neither nuclei nor processes are visible. In this imperfect state they are to be found in tuberculous nodules from all organs in which Tubercle is met with.

"It remains to speak of the blood-vessels of Tubercle. The vessels of the tissue in which the nodule originates gradually become obliterated in the process of its growth, and there is no new formation of vessels, such as takes place in more highly-developed inflammatory tissue. The Tubercle is therefore, except in the earliest stages of its development, non-vascular.

"Although the structure which has been described is that most commonly met with, it must be borne in mind that all tuberculous lesions are not thus constituted. When treating of the changes in the several organs, it will be seen that the precise histological constitution of the nodules varies somewhat, according to the characters of

the tissue in which they originate. In the lung, for example, many of them consist largely of accumulations of epithelial cells within the pulmonary alveoli."

As giant cells are now ackowledged to enter so largely into the composition of Tubercle, it is perhaps advisable that the different ideas entertained of their connection with this product should be laid before the reader. The most recent *résumé* is to be found in the *Lancet* of the 29th of June, 1878, and runs as follows :—

"GIANT CELLS IN TUBERCLE.

"The nature of giant cells is a subject on which there is much difference of opinion amongst pathologists.

"Some have ascribed to them an especial importance in the formation of Tubercle, others regarding them as accidental productions; but few now deny their pretty constant occurrence. M. Cornil has recently brought before the Société de Biologie a communication designed to show that they are formed in the interior of obliterated vessels.

"He showed specimens from a tubercular infiltration of the pericardium, prepared by hardening with osmic acid and alcohol, and staining sections with picro-carminate of ammonia.

"From sections thus prepared he succeeded in isolating a large number of free giant cells, consisting of masses of granular protoplasm of various shapes, with numerous prolongations, some of which were bifurcated and contained many nuclei which were always ovoid or budding, thus showing an active process of growth. They usually occupied the more peripheral part of the cell and varied from two or three to twenty or thirty or more. M. Cornil believed that he was able to trace the process of growth of the cells in these and many other preparations of Tubercle on serous membranes, and he describes it as consisting of a special inflammation of a limited part of a vessel, with coagulation of fibrin, the accumulation of leucocytes in the clot, an active process of growth and multiplication of these and of the endothelial cells, softening of the walls of the vessel, which, by infiltration with cells, become indistinguishable from the surrounding tissue.

"In opposition to this view, M. Malassez brought forward evidence to show that the true giant cells of Tubercle are not due to obliteration of the vessels. He objected to the views of M. Cornil, that the number of giant cells was often enormous, that they presented prolongations which could not arise in a vessel, that they were often much larger than the vessel in the part, and that they contrasted with obliterated vessels in the fact that their protoplasm was actively growing, not mere altered coagulum, and that they lacked all trace of a muscular wall. He himself urged that many of them were cells which should form vessels, but failed to do so, corresponding to the 'angioplastic' or vaso-formative cells of new growth.

"This view has before been advocated by Brodowski, and adopted by Professor Charcot.

"But M. Malassez holds that other methods of formation may also be observed.

"There can, we think, be little doubt that such is the case; that many of the so-called giant cells are only obliterated vessels, but that true giant cells are also formed, and that these may be formed either by angioplastic cords or by accumulation of actively growing, but not yet differentiated, protoplasm, derived usually from endothelial cells."

Of the secondary changes through which Tubercle passes, Green says:—"It invariably undergoes more or less retrograde metamorphosis, although the extent of this varies considerably, and in some cases the nodules may become developed into an imperfect fibroid structure. The occurrence of retrograde metamorphosis is mainly owing to the obliteration of the blood-vessels which accompanies the growth of the lesions. The change commences in the centre of the nodule, this being the part first developed, and consequently that which is the furthest removed from vascular supply. The nodule breaks down into a granular fatty débris, so that its central portions soon become opaque and yellowish. In some cases the process of disintegration is rapid, whilst in others it is more gradual. It is usually most marked in the larger and more diffused lesions, and hence it is these lesions which are most commonly of a yellow colour and soft consistence ('yellow Tubercle'). In other cases the retrograde change is less marked, the reticulum of the nodule becomes denser and more fibroid, and although the imperfect fibroid tissue usually ultimately undergoes in its central parts, more or less fatty metamorphosis, the nodule may remain as a firm fibroid mass. This occurs more especially in the smaller lesions. The extent and rapidity of the retrograde change depends, I believe, partly upon the intensity of the infective process, and partly upon constitutional conditions. The existence of scrofula favours retrograde changes in the tuberculous lesions as it does in all inflammatory products, and it is in those who are markedly scrofulous that Tubercle undergoes the most rapid degeneration. These changes will also be influenced by the intensity of the infective process. The more intense the process the greater is the tendency to the degeneration and softening of the nodules; the less intense and more chronic, the more liable are the miliary lesions to become fibroid."

The exact nature of Tubercle and the methods by which its miliary lesions originate is even now a moot question. It has been variously regarded as simply a degenerated aplastic exudate, the result of inflammatory action occurring in a system already debilitated by disease or constitutionally liable; as, a miserable abortion; and as the result of some specific morbid principle or organism which by some means has gained access to the system or originated therein by perverted glandular or nutritive functions.

Experimentation has done much towards enlightening us as to the true nature and origin of Tubercle; but while acknowledging the great value of experiments generally

in elucidating pathological processes, I must, nevertheless, remark that the results of many of the experiments which have been initiated are totally opposed to the teachings of clinical experience, and all tend in the minds of many experimenters to one conclusion—viz., that Tubercle is the result of absorption and dissemination of the particles of a degenerated exudate; but if it is due simply to degeneration of an exudate, why do we not see it in the horse? The answer may be, because the horse is not predisposed to it. I ask, why is he not so predisposed? Can any material difference be shown to exist between his blood and tissues and those of the ox, to account for a simple inflammation in the one animal going into the development of a malignant product, and in the other to some harmless tissue?

Is Tubercle simply of a lymphatic character? if so, why do we so seldom see it in the spleen of the ox, the most lymphatic organ in the whole body?

As to the argument "that it is due to absorption of broken-up matter from a caseous centre," let us see upon what it is based—*Firstly*, Upon experiments with setons and certain products of degeneration.

The experimenters do not, if I understand them aright, absolutely assert that the introduction of a seton or other foreign matter under the skin *will* produce Tubercle, but a substance which cannot be distinguished, histologically, from Tubercle. If *Tubercle* can be so produced, how is it that calves—animals pre-eminently disposed—are not more frequently the subject of it in districts in which setons are inserted in the dewlap as a prophylactic measure; and not only inserted but allowed to remain *in situ* for an indefinite period? How is it that Tubercle does not follow setoning or rowelling in the horse and the ox when had recourse to as a curative measure? I have seen pyæmia follow setoning, but Tubercle, never.

Secondly, The argument is based on the fact that miliary Tubercle—of recent origin—is *often* (it is not asserted *always*) found side by side with an old caseous centre from which infection has taken place, as proved by the radiating character of the miliary depositions. I do not think that any pathologist would assert that tuberculisation arises from *any and every* caseous centre, no matter what its origin; but, that it is found coexistent with *a* caseous centre, leaving it to be assumed that the caseous centre is, or may have been, of tuberculous origin.

In practice we find that an old caseous centre existing in the body of a horse or a dog will under certain influence—as fever—break up, its encysting envelope become active, absorption of infective particles take place, and pyæmia (multiple abscesses), not Tubercle, result. We see the same thing in young animals in septic inflammation of the umbilical veins—not Tubercle, but pyæmia.

If Tubercle is simply the result of absorption of the particles of broken-up tissues and degenerations, why does it not always follow the breaking up of the lung after Zy. p. p.? I may be told that it does do so; I answer unhesitatingly, that, practically, it does not do so unless there is a tuberculous diathesis. I have made autopsies of dozens

of animals in whose thorax pounds of degenerating Zy. p. p. lung existed without finding a trace of Tubercle.

I have made autopsies, again, in which Tubercle did coexist with degenerating lung-tissue; but, which had primary existence? As often as not the Tubercle was easily proved to have had prior existence by the condition and characters of the two materials; moreover, how often do we see very old tuberculous deposits in animals which have been killed in the very earliest stages of Zy. p. p.?

If Tubercle is a simple pyæmic process, how is it that we do not see the characteristic tubercular ulcers developed in the intestines when these organs become the seat of ulceration from the absorption of purulent matter? How is it, that in the one case the ulcer has a definite and specific character, and seldom goes on to sphacelus or perforation, while in the other its character is indefinite, and it shows a great tendency to terminate in sloughing and perforation?

Finally, will pyæmic products, like Tubercle, graft themselves, if ingested, upon the mucous membrane of the mouth and alimentary tract, or, if inhaled in a fine state of division, upon the mucous membrane of the bronchial tubes? Is pyæmia, or a tendency to it, hereditary?

In some experiments which I made several years ago with the caseous products of ordinary inflammation and tuberculous products respectively, it did not appear to me that the points of resemblance in the resulting lesions were so great as to preclude the possibility of differentiating the two, even without the aid of a microscope; and certainly, the lesions of pyæmia, as seen in the lungs, or on the pleura or peritoneum of an ox, as the result, say, of Zy. p. p., are totally distinct from those which are produced by auto-inoculation from a tuberculous centre.

I may be pardoned if I venture to direct attention to the fact, that in the degeneration of Tubercle, especially when a cyst is formed, and communicates with a bronchial tube, other than purely tubercular elements are produced, as, *e.g.*, infective pus, which, becoming absorbed by the lymphatics, originates a veritable disseminating pyæmia.

In whatever way I consider the character of Tubercle, as Tubercle, my mind always reverts to the same conclusion, viz., that Tubercle is as much a specific disease, whether inherited or acquired, as glanders, syphilis, or any other affection of the kind with which we are acquainted.

Before discussing the nature and characteristics of Tubercle further, it will be as well to consider briefly one preliminary question which has been frequently raised, viz., "Is the Tuberculosis of animals identical with that of man?" I imagine there are few who have carefully studied the disease in animals who do not acknowledge that it is identical with that of man, and yet the statement is made that it is not so; and in support of the statement, it is urged that we have not ulcerative lung-disease, or hectic fever, such as is seen in man—in the phthisis pulmonalis of the ox.

I shall show hereafter that *we have* ulceration both of lung-tissue and of the

bronchial and larnygeal mucous membrane with hectic; not, perhaps, so often as in the human subject, but sufficiently often to establish—so far as this point is concerned—their identity.

All doubt, too, is now set at rest by the various experiments which have been instituted by veterinary surgeons abroad, as to the effect of inoculation and ingestion of the Tubercle of man in and by animals.

The late Professor Fordie, at the meeting of the Association in Glasgow, at which I read my paper in 1872, strongly supported the opinion that there was no relation between the Tubercle of man and cattle, and advanced, as evidence of this view, the fact, that he had seen many animals at the abattoirs in splendid condition, which yet, when killed, were found to be the subjects of extensive tubercular formations; and he particularly instanced a number of cows from a certain dairy in Glasgow; but, unfortunately for the argument, Mr. Wm. Anderson rose and stated, that every one of the cows particularly specified had been the subject of Zymotic Pleuro-pneumonia, and that the so-called tuberculous changes in the lungs were nothing more nor less than degenerations of lung-tissue produced by this disease.

PREDISPOSING CAUSES.

These are numerous and important, and will be considered *seriatim.*

1. *Species of Animal.*—The bovine tribe is pre-eminently disposed, equally so, indeed, with man; next in order comes the common rabbit, which animals, in some districts, are almost annihilated by the disease; the pig is very prone to Tubercle, as are also poultry, our feathered household pets, and, under certain circumstances, game birds. I have even seen it in the rook, though it is curious that the palmapedes are exempt from it. Tubercle is rarely seen in the sheep, cat, or dog, and, as I have already remarked, I have not seen it in the horse, though it is said by some authors to be of common occurrence in this animal. The late Dr. Davidson, ex-Professor of Anatomy in the Dick Veterinary College, showed me, several years ago, a well-marked case of pulmonary phthisis in a monkey, which animal, as also many others which are naturally wild, seems readily to contract a predisposition to it under the influence of domestication.

2. *Hereditary Tendency* may be divided into *direct* and *indirect;* the former when it is transmitted by a sire or dam to its immediate progeny, the latter when only transmitted to the second or third generation—constituting *atavism.*

No predisposing cause with which we are acquainted exercises such a potent influence in the production of Tubercle as this; from sire to son, from dam to offspring, from generation to generation—often in unbroken succession—the fatal tendency is transmitted; the more consanguinity is multiplied, the more the tendency is increased, and the greater the virulence of the resulting products.

It must not be assumed, however, that Tubercle, as Tubercle, is propagated *congenitally,* at least I am not aware of the existence of any proof of such a phenomenon, and, most certainly, I have never seen it—that the germs of Tubercle may exist in the blood or tissues of the fœtus may be allowed without sacrificing any principle; and further, that these germs may remain dormant for a considerable period after birth, *i.e.,* until certain influences favourable to their development or increase are brought to bear upon the individual which harbours them. Be this as it may, all we contend for at present is that the existence in particular animals of the *Scrofulous Diathesis* is as certainly proved as that the animals themselves are living entities, and that the development of the local manifestations of this diathesis is, so far as we know, *extra-uterine.*

3. *Breeding in-and-in* may be placed as a predisposing cause, equal in potency with hereditary tendency. In spite of the terrible and palpable examples of this truth met with by breeders, some of them still pursue, year by year, the suicidal policy of clinging to *one strain,* refusing persistently—as they say—to contaminate with anything which is inferior or likely in the long-run to deteriorate it.

4. *Breed.*—The breeds of animals which, in my experience, are most subject to Tubercle are Alderneys, Guernseys (the latter in a much less degree, however, than the former), and shorthorns amongst home cattle, and, amongst foreign cattle, the Danish. It must not, however, be assumed from this remark that all shorthorns are equally predisposed; it is only in particular districts and with particular strains that this holds good. Neither would I have it assumed that all pure and highly-cultivated strains are contaminated; but I do with confidence assert this—that quite half, if not more than half, of the well-known strains are tainted with the leprosy of Scrofula. With regard to the majority of our pure breeds, I can only speak positively of those with which I am practically acquainted. In Highland cattle I have never seen Tubercle, though it is very possible that those who have opportunities of seeing autopsies of old cows may have done so. In some districts Herefords are peculiarly exempt from the disease; while in others, as in some parts of North Wales, I have seen Scrofula frequently developed. The old smoky-faced Montgomeryshire cattle—few though they were during my residence amongst them—I seldom saw affected, and the same remark holds good with reference to the old Staffordshire longhorns. The Ayrshires in certain districts are somewhat prone to Tubercle, while in others they are free from it; but—as I shall show hereafter—under the influence of change of climate they become particularly predisposed. The polled Aberdeenshires seem to be particularly exempt—at least, I have never seen Tubercle in them; and I have it from Mr. M'Combie, "that he has never seen it in any cattle of the polled breed, however closely bred." Of the Devons and other breeds not referred to, I know nothing personally; but I am informed by Mr. Kettle, of Market-Drayton, that during a residence of some years in Devonshire he never saw a single case of Tubercle.

5. *Early, Late, and Over-Breeding—i.e.,* breeding from animals at an age when

their tissues are imperfectly formed or worn out, and the system incapable of elaborating healthy cell materials. It is certain that breeding may be carried on with profit amongst, comparatively, very young animals—Mr. M'Combie commencing at fifteen months old—and the same may be said, in many instances, with regard to old animals which have been carefully tended, and whose systems have not been debilitated by prolonged and frequent lactation. I have seen very old cows throw, year after year, very healthy strong calves, and the same excellent authority whom I have quoted above tells me that some of his go on breeding until past their twentieth year. Nevertheless, it is, on the whole, an extremely unwise system to carry either early or late breeding too far in any breed, and particularly so in the more delicate breeds.

6. *Physical Conformation.*—Those animals which are possessed of light barrels, narrow chests, and disproportionately long legs, are undoubtedly more predisposed to Tubercle than those in which conformation may be said to be perfect; but it must not be forgotten that these physical defects are often in themselves evidences merely of stunted growth from the existence of the tubercular diathesis—mere deficiency in this respect not being of itself sufficient to engender the tendency. With physical conformation we may class *colour*, some authorities holding that the lighter the colour the greater the tendency to Tubercle. Now, while it is, in the main, true that very light roans are liable to the disease, I have seen many strains of pure whites totally free from it; on the contrary, I have frequently seen reds succumb to it.

7. *Climate and Locality.*—It is a remarkable fact that climate exercises a material influence in the production, not only of Tubercle, but of allied diseases—and that not only in reference to native breeds, but often, and in far greater intensity, to exotics.

A striking illustration of this is afforded in the decimation of several herds of Ayrshires which have from time to time been introduced into Sweden—facts narrated to me by an eye-witness, and late pupil of my own, Mr. John Nonnen, of Degeberg. Of the nature of the influences which are at work, in particular climates or localities, in the production of Tubercle we have not at present any very definite or reliable evidence; and while some authorities assert that these influences are barometrical, others with equal confidence pin their faith to chemical peculiarities of soil, and others to the characters of herbage. It must, I think, be patent to all that an unusually relaxing or a very cold climate, in fact, any extreme in this direction, will largely influence the nutrition of the tissues and the activity of the glandular and other functions of the body: with equal force may the proposition be applied to excess or deficiency in the normal inorganic constituents of the soil, and to the nutritious or innutritious nature of the herbage. It is a well-known law that for the production of healthy tissues, and for the maintenance of perfect health, a certain proportion of saline matter and of flesh-forming and heat-producing materials are absolutely necessary, and that if the balance of these essentials is upset, very slight adverse influences determine important functional and structural changes.

In my own experience I have found that those animals which are depastured on low-lying, or elevated, damp and cold lands, in which the herbage is largely mixed with iron-grass, rushes, and moss; are the most predisposed to such special diatheses as Scrofula.

In some Continental countries it is believed that lime-salts exercise a material influence in the production of the tubercular diathesis, as it has been found in these districts that both animals and man are particularly liable to the affection; but it is only right to mention that while in some districts such a belief exists, an exactly opposite opinion is entertained in others.

8. *Debility*—Is a certain predisposing cause, whether produced by organic disease, deficiency of nourishment, light, and exercise; excessive milking, obesity, exposure, over-exertion, bad ventilation or drainage; exposure to an impure atmosphere, or excessive depletion by bleeding, and the administration of drastic, purgative, or other lowering drugs; prolonged and inordinate discharges of blood, pus, or mucus; or long-continued excitement, as in œstrum.

But acknowledging that every one of the influences just referred to predispose to the development of Tubercle, it must not be inferred that I think *any one, alone,* is sufficient to produce it. Such predisposing causes require to be combined in order to culminate in this, or any other special, diathesis.

DISSEMINATION AND PROPAGATION OF TUBERCLE.

The propagation of Tubercle by *inoculation* is now such a well-established fact that it is quite unnecessary for me to allude to it at any length.

Since Villemin published the results of his inoculative experiments, which were conducted with great care, and practised upon a great number and variety of animals, many pathologists—medical and veterinary—have entered this field of research, and the majority have unhesitatingly confirmed his conclusions, differing only in unimportant points. Villemin, as have others since his time, found that inoculation with tuberculous matter produced both local and general lesions, and that the new material formed at the seat of the operation was as capable of reproducing Tubercle as was the primary tuberculous matter; he also found that abortion, in pregnant animals, was a frequent result of inoculation, still further proving the constitutional disturbance set up by the local introduction of the virus.

One authority, Desmartis, of Bourdeaux, has even asserted that he has succeeded in the inoculation of plants with tubercular matter.

Ingestion.—In the paper which I read on the subject of Tubercle at the West of Scotland Veterinary Medical Association in 1872, I made two statements in reference to the propagation of Tubercle by ingestion which I here reproduce. 1stly, "If the contagious

character of Tubercle is established, another (though, perhaps, a remote one) source of contamination is opened up before us—viz., the eating, by persons or animals, of raw or improperly cooked meat; or, more strongly, the liver or other internal organs of animals which have suffered from this affection."

2ndly, "Another very remote cause—and perhaps you will say it is a very remote one—I must mention, is this. Animals suffering from intestinal and hepatic Tubercle, especially rabbits which are constantly running over pasture, must necessarily pass with the fæces a large quantity of tubercular matter in various stages of development and degeneration. May not this be picked up with the grass, and small portions of it become lodged in accidental wounds or abrasions of the mouth or throat (especially as we know that small abrasions and wounds do frequently exist in these situations), and thus, by direct inoculation, propagate the disease?"

When these (hypothetical) propositions were penned, I little thought that many facts would be subsequently brought to light which would more than justify the publication of such suppositions. Even as I uttered these words, Chauveau was publishing the results of some ingestive experiments in calves with tubercular products; but long before this (in 1866-7), according to Fleming, experiments with the milk and flesh of diseased animals had been carried on in the Hanover Veterinary School by Gerlach, and these experiments proved indubitably that Tubercle could be transmitted or propagated by ingestion. I have myself seen it transmitted in this way to the common fowl.

Since attention was prominently directed to the subject by Gerlach and Chauveau, other veterinary surgeons—notably Viseur of Arras, and Klebs—have confirmed the conclusions at which they had arrived. Professor Axe, of the London Veterinary College, has also, I believe, experimented largely in this direction with the same results. Only a few days ago I received a communication from Mr. William Bromley, M.R.C.V.S. of Lancaster, in which he says:—"A short time ago two pigs were killed in a farm in this neighbourhood which had been fed with milk from a cow afterwards found to be the subject of Tuberculosis—and from which she died—and upon *post-mortem* examination characteristic pulmonary and pleural tubercular lesions were discovered." In a subsequent letter Mr. Bromley further says:—"The mother of the two pigs to which I referred in my last has, within the past few days, been slaughtered. She was perfectly healthy, no trace of tubercular or other deposits being discoverable in any part of her body."

I cannot enter into a lengthy discussion on the results of ingestive experiments, it will be sufficient to quote the following from an article on the subject by Fleming in a recent number of the *Veterinary Journal*, and to refer those who wish to inquire farther into the matter to Fleming's "Veterinary Sanitary Science and Police," vol. ii., to the article above alluded to in the journal; and to another article by the same author in the *British and Foreign Medico-Chirurgical Review*, October, 1874.

The conclusions arrived at by Gerlach, and as quoted in the *Veterinary Journal*, are as follows :—

"1. There is a specific virulent material in tubercular matter,—the presence of a tubercle virus is evident. The disease is produced through the digestive canal, by tubercular matter.

"2. The fibrous tubercles from the serous membranes contain this virus.

"3. The virus is present in fresh, but not in decomposed matter—in the fibrous and cellular tubercle, in the crude as well as the broken-up cheesy masses; though it exists in its greatest intensity in the latter,—a small quantity producing the largest amount of tuberculisation.

"4. Infection of the alimentary tract commences in the mucous membrane of the mouth; if the tubercular matter has been left sufficiently long in contact with this membrane, there may be general infection of the entire lymphatic system. This membrane shows an aptitude for infection, from the mouth to the cæcum.

"5. The tubercle of other animals and man is more or less virulent.

"6. The fibrous tubercle of horses, destitute of cheesy matter, is as infectious as the tubercle of cattle : though real Tuberculosis is rare in this animal, and it is very difficult to obtain fibrous tubercle which is not present in Glanders; the miliary tubercle of that disease is not proper Tubercle. Glanders is not, therefore, Tuberculosis.

"7. The Tuberculosis of *birds*, and especially of the *common fowl*, is very virulent, and identical with the Tuberculosis of mammals,—a most important conclusion, from a sanitary point of view.

"8. The *flesh* of a tuberculous cow is also infectious, though not nearly so much so as Tubercle itself. It requires a much larger quantity of flesh to produce infection than of miliary or cheesy tubercle.

"9. Tuberculous matter, boiled from a quarter to half-an-hour, is still infectious ; though to a much less degree than when uncooked. The period of boiling required to render it inert depends upon the thickness and density of the mass : in this respect it resembles trichinosed flesh.

"We cannot refer to any satisfactory extent to the symptoms and lesions produced by feeding the experimental animals on tuberculous matter, nor on the different results obtained in different species. Suffice it, therefore, to say that they existed in two degrees—the largest number of animals being affected with catarrhs—follicular catarrhs—due to relaxation and tumefaction of the tissue of the mucous membrane and its glands—both the solitary and Peyer's patches, most of which were more or less swollen, and covered with a copious layer of thick mucus. Rabbits chiefly suffered from this milder form. Sheep were affected with the severer form of Tuberculosis, and in them were found crude and cheesy tubercles. In pigs the mucous membrane of the pharynx was involved when they were not fed with the tuberculous matter by means of an œsophageal tube.

"The lymphatic glands in the vicinity of the diseased mucous membrane were swollen, œdematous, and abnormally filled with lymph corpuscles; at a more advanced stage, there were miliary tubercles and small cheesy deposits; still later, partial or general caseation of the glands; and after a long period from the date of infection, there was thickening of the connective-tissue septa of the gland-lobes, with layers of miliary tubercles. The mesenteric and intestinal glands, as well as those of the throat, were infected and diseased. The mucous membrane of the mouth and pharynx, as already mentioned, became affected through contact with the matter on which the animals were fed.

"After the lymphatic glands were involved, other organs became affected: first the lungs; after them, though seldomer, the liver; still more rarely, the spleen and kidneys; and at times other parts—as the joints, *dura mater*, &c. The lungs generally exhibited the Tuberculosis in *optima forma;* in the subpleura, and deeper in the lung, were tubercles in various stages and of various sizes. When the disease was of brief duration, there were recent, grey, smooth, miliary and submiliary tubercles; when it had run a longer course, there were cheesy masses the size of a pea, and larger in some cases,—in rabbits particularly, with inflammation of parts of the lung; and where adhesion had taken place between the pleura pulmonalis and the pleura costalis, there were generally the largest cheesy masses—Tuberculous Pneumonia. The bronchial glands were always affected, either indirectly through the lungs or directly through the alimentary canal."

Viseur's experiments produced the following results:—

"In all the animals the respiratory organs were seriously affected (*gravement atteint*). The lungs were studded, superficially and deeply, with grey, hard, semi-transparent nodules. The deep-seated ones were about the size of a large pin-head; those more superficial were spherical and reduced to the size of the finest miliary granulations, difficult to break up, and giving the pleura a roughened aspect. The submaxillary, retro-pharyngeal, and pre-pectoral glands were very visibly tuberculosed. In one animal the lesions had reached an extraordinary degree of generalisation; the laryngeal mucous membrane offered different phases of the disease—granules and ulcerations closely set together. The digestive organs offered a still more remarkable condition. In the peritoneum the granulations were particularly grouped in the terminal portion of the ileum and the commencement of the colon: they were dense, resisting, and the size of millet or hemp-seed. In the small intestine Peyer's patches had more or less undergone the effects of tuberculous inflammation. Around the ileo-cæcal valve in one case (cat) were ulcers, some of them extending through the muscular coat. In another cat the lesions were startling (*effrayantes*), the ileum being the seat of a veritable confluent eruption, its walls were thickened, hard and rough to the touch, and its serous membrane had also become greatly thickened, and showed layers of superposed granules. The mesentery was shrunken, and its folds from the points where

absorption had been most active exhibited on the course of the lymphatic vessels numerous semi-transparent granules the size of small grains of sand disposed in a typical manner. The glandular apparatus was in such a state of tubercular hypertrophy, especially in the young animals, as to account at once for the marasmus that ensued. The pancreas of Aselli, and the other glands annexed to the ileum and colon, were more than double their natural volume, and at some points were filled with a whitish semi-transparent more or less cheesy matter, and yellowish or cretaceous matter in others. The liver and spleen, chiefly the latter, contained salient tuberculous nodules. The other abdominal organs appeared to be healthy.

"None of the cats had escaped infection, and the infecting agent had acted so powerfully that the multiplicity, generalisation, and gravity of the lesions were remarkably apparent. With the dogs the results were less uniform, though they had gluttonously eaten a large quantity of tuberculous matter."

Infection through the medium of the breath of animals suffering from Pulmonary Phthisis.—In 1872, and, indeed, prior to that year, though I had not then any very direct evidence on the point, I felt quite convinced that Tubercle could be propagated by inhalation of the breath of phthisical animals, as also of man. The outbreak to which I referred as occurring at Bolton, and which came under my notice during my residence in Manchester, could only, to my mind, be accounted for by pulmonary infection. From a variety of sources, during the past few years, the view that Tuberculosis is contagious by the medium of the expired air has been confirmed. In the *Veterinary Journal* for January, 1876, the editor calls attention to a confirmatory opinion expressed on this point in 1871 by Mr. Graham Mitchell, of Melbourne, and reiterated, with further proofs, by that gentleman in 1875.

In a communication to the editor of the *Veterinary Journal* (p. 373, November, 1875) on the subject of Tuberculosis, my esteemed friend, the late Mr. Dewar, of Midmar, thus expressed himself in reference to this question:—

"My attention was particularly directed to the subject of Tuberculosis in 1839, immediately after I left the Edinburgh Veterinary College. In my locality the disease prevailed to such an extent, and was so very fatal, that the owners would not sell what were considered diseased animals; and one after another became affected. It appeared as if all were equally susceptible—cows, queys, and working oxen—and all died sooner or later. Being but a tyro, I was grieved to the heart because I could never make a permanent cure of any; although several flattering cases presented themselves, yet they only lasted for a time, and succumbed in the end. I called in the district physician, and made a *post-mortem* examination of one of the animals in his presence; we were both of the same mind with respect to the disease—viz., tubercle of the lungs—he termed it 'phthisis pulmonalis;' and we both agreed that the byres (cowsheds) were too close. They were recently built, and ceiled and plastered like a drawing-room. The proprietor agreed to remove the 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, and tonics, and stimulants may have prolonged life ; but yet no cure was effected, and, as far as I can remember, that herd died out. I resorted to every possible 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 after there was as good a lot of cattle as any in the district. The occurrence of disease in that herd left an impression on my mind that never can be effaced, and made me always dread the evil consequences of Tuberculosis. Having practised in the same locality, which is an extensive breeding and rearing district, I have seen several herds decimated. Although I sometimes foretold the owner what he might expect, yet he did not believe me until so many were affected that the byres were infested with the fearful malady ; and your remarks with regard to nose contamination are so true, that I do not think it possible any can escape which are so exposed. I have also seen it as Grad has—the same stall infecting beast after beast, until a thorough cleansing and disinfection, with plenty of fresh air, had removed all infection from it. But the experience I have had of it makes me fear when once it has been witnessed in a few animals in a herd, and in different stalls ; for my observations would go to prove that in all probability some animals are born with it, and if congenital, the stock is difficult to purge of its seeds, if I may so say. If one of the parents is tuberculous, the progeny is more susceptible by three-fourths. My experience of it makes me dread it, and fear it even more than what you have written upon it. It is so insidious in its attacks, that when you think you are all right it may appear in a grandchild of what was considered a very healthy animal, and outbreak after outbreak will occur years after all has been seemingly healthy. We must take into consideration soil, climate, locality, &c. I see in last week's *Lancet* that a medical gentleman had been called in by the sanitary inspector, and condemned a cow affected with Tubercle, but two veterinary surgeons pronounced the carcase sufficiently healthy for human food. I believe it is customary in England, as well as here, to eat the flesh of animals affected with, or which may have died from the effects of, Tuberculosis ; but, although customary, in my opinion all animals killed and found affected with this disease should be burned. I hope you will forgive me for taking the liberty of writing you upon this subject, but I felt so pleased with your views of it that I deemed it to be my duty to thank you."

In the *British and Foreign Medico-Chirurgical Review*, October, 1874, Mr. Fleming quotes the observations of Grad, a veterinary surgeon at Wasselonne, Alsace, which are, I think, fairly conclusive as to the contagiousness of Tubercle, though Grad himself, while stating that the animals contracted the disease by being tied up in stalls recently occupied by phthisical cattle, seems to incline to the opinion that the disease was propagated rather through the medium of contaminated food than by vitiated air.

Zundel, an Alsatian veterinary surgeon, confirms Grad's opinions.

In my essay on the subject, to which I have before alluded, I remarked that many circumstances had come under my notice which tended to prove that consumption was contagious. Many members of the medical profession have for long entertained this opinion; and I think I may confidently say that nothing which has occurred for a very long period will tend more to strengthen if not to confirm it, than the experiments recently conducted by Dr. Tappeiner, of Meran, and alluded to by the editor of the *Lancet* (p. 741, 23rd November, 1878) in the following terms :—

"The remarkable instances now and then seen, in which persons without hereditary tendency to phthisis become phthisical after long-continued attendance on sufferers from the disease, have suggested to many physicians the idea that phthisis is contagious. If there is such a contagion, the mechanism has been supposed to be the inhalation with the breath of fine particles of tuberculous sputa, atomised into the air by the patient's cough. An attempt has been made by Dr. Tappeiner, of Meran, to ascertain whether, by a similar means, animals could be rendered tubercular, and the results of the experiments, which are published in the current number of Virchow's *Archiv*, are of great interest. The animals experimented on were made to breathe for several hours daily in a chamber in the air of which fine particles of phthisical sputum were suspended. The sputum having been mixed with water, the mixture was atomised by a steam atomiser. In all cases the sputa were from persons with cavities in their lungs. Dogs alone were employed in the experiments, since they very rarely suffer from spontaneous Tuberculosis. The result was that of eleven animals experimented on, with one doubtful exception, after a period varying from twenty-five to forty-five days, all, being killed, presented well-developed miliary tubercles in both lungs; and in most of the cases tubercles were present to a smaller extent in the kidneys, and in some cases also in the liver and spleen. Microscopical examination was in accord with the naked-eye appearances.

"The quantity of sputum necessary for the effect is certainly a very small one. In three experiments only one gramme of sputum was daily atomised in the air of the chamber, and the quantity of dry sputum must have been exceedingly small. Two ways are conceivable in which the infection is produced. The particles certainly may reach the alveoli, for powdered cinnabar administered in the same way was found to have stained the alveoli in twelve hours after an inhalation of only one hour's duration. But some particles may lodge in the mucous membrane of the throat and pharynx, and thence, being absorbed, may affect the lungs as organs specially predisposed. Hence some comparative experiments were made by feeding dogs with the same sputum as that employed in the inhalation experiments. Fifteen grammes were mixed daily with the food of each dog. In two dogs fed at Munich miliary tubercles were found in the lungs after six weeks' feeding; in six others fed at Meran all the organs were normal—a difference the explanation of which is not very clear. In the cases in

which the disease was produced by feeding, the intestinal tract was affected, whereas it was free in those cases in which the inhalation was employed. It is remarkable that, with two exceptions, the animals, up to the time at which they were killed and found diseased, were well and lively, and indicated their disease neither by emaciation nor other external symptoms. This suggests that sometimes in man a miliary tuberculosis of the lungs may remain latent, and cause no symptoms until a catarrh, with foci of inflammation, sets up phthisis.

"A preliminary account of these experiments of Tappeiner led Dr. Max Schottelius to make some similar experiments, not only with the sputum of phthisical individuals, but also with that of persons suffering from simple bronchitis, and with pulverised cheese, brain, and cinnabar. The result was that miliary tubercles were found in the lungs in all cases, and in equal quantity with both phthisical and bronchitic sputum. Cheese produced a smaller quantity; pulverised brain still less; and the cinnabar least effect of all, merely a few whitish tubercles with pigmented centres, with an interstitial deposit of the substance, which had caused no inflammatory reaction. Tappeiner has also experimented with calves' brain in two cases, but with purely negative results. No changes in the lung followed such as resulted from the inhalation of tuberculous sputum.

"These experiments are of much interest, but they need repetition on a larger scale in order that the discrepancies may be removed, before much weight can be attached to them as evidences of a specific influence of the phthisical sputum. They unquestionably show, however, that the inhalation of foreign organic matter will cause tubercles in animals naturally indisposed to their development. The appearance of granulations in other organs than the lungs in some of Tappeiner's experiments is a fact of great importance. Whether tuberculous matter produces tubercle when given in this manner more readily than other substances or not, it appears certain that different forms of organic matter produce effects in different degree. It appears also that the inhalation of these substances is more effective than their administration by the alimentary canal. These are facts of great importance in regard to the question of the contagiousness of phthisis."

GENERAL INDICATIONS OF TUBERCULOSIS DURING LIFE.

Diseases of organs having their origin in tuberculous infection may be distinguished from those of idiopathic origin and of a sthenic character, by previous history, coincidences, and other collateral circumstances; thus, an animal may be suffering from nymphomania; coincidentally (internally or externally), laryngeal obstruction exists, or it may be tubercular disease of a joint. On inquiry we find that other members of the herd have been the subjects of Tuberculosis, and, though the animal may be fat and

in good muscular condition, a tolerably certain diagnosis can be arrived at. Again, when an animal has shown undoubted signs of the disease, and the diathesis exists, it will be stunted in its growth, easily tired on exertion, lanky in body, and more or less hide-bound; emaciation may not be noticed at first, and indeed, if the supply of nourishment is in excess of the demands of the tubercular growths, and they do not interfere with the functions of important organs, the host will approximate the excess of nutrient material and become fat; but in the majority of these cases the tuberculisation is confined to the exterior of organs, and the products do not undergo softening, neither do they produce secondary results by pressure on important organs or vessels of supply.

Animals may also become very fat when the tubercle, even in the lungs has become *obsolete* or *quiescent*, more particularly is this the case in pulmonary phthisis when the function of one lung is completely annihilated by invasion of tubercular elements and secondary inflammatory products; such a condition of things existed in the ox from whose lung the drawing for Fig. 2, Plate X. was obtained.

The explanation of the latter phenomenon is simple, as the destruction of a lung materially diminishes the process of oxidation and thus favours the accumulation of fat.

Specific disease, due to Tuberculosis, may run its course quickly or slowly, depending upon a variety of circumstances—as age, keep, improper care, or whether the blood is, or is not, in a condition favourable to the rapid multiplication of the tubercle. In some instances, its progress is, for a considerable period, slow, and, on the occurrence of some exciting cause, it assumes an acute character; thus, if Miliary Tubercle exists on the pleura or peritoneum, and an attack of inflammation (either idiopathic or traumatic) is induced in the membrane, the tubercle grows very rapidly owing to the increased vascularity of the parts and the larger amount of blood (nourishment) presented to it upon which to multiply; in the same way, after each period of œstrum, and after parturition in females, the disease makes rapid strides. During pregnancy, increase of the tubercular growth is held in abeyance the energies of the nutritive processes of the body being diverted to the nourishment and growth of the fœtus; after parturition, the system, from a combination of causes, is, for a time, debilitated and rapid extension of the tubercle is favoured. At the period of œstrum all the functions are enhanced, excited, or perverted, and the circulation more rapid—followed by depression and debility.

In the diagnosis of tubercular disease during life much difficulty is often experienced, more especially when the history of the case is not forthcoming. As already remarked, if there are coincidences and collateral circumstances to guide us the diagnosis is rendered more easy; otherwise, acute tubercular disease of an organ may give rise to no symptoms of a positive character; particularly would this be so in the case of an organ having no external outlet, and, even where one exists, it would be of far less aid to us in acute than it would in chronic disease. In acute Hepar Tuberculosis, for instance, there might be no symptom which would not be equally present in acute

hepatitis from any cause; so in acute pulmonary, renal, or intestinal Tubercle; nor (either in acute or chronic) should we have any symptoms to guide us where the disease is confined to the internal lymphatic glands. In chronic Tuberculosis of organs having an external outlet there is always just the possibility of our being able to detect by patient ocular and microscopical examination, tubercular elements in the secretions or excretions.

Tubercle kills—1stly, By destroying the structures, and consequently the functions, of organs in which it is deposited, as the liver, lungs, kidneys, &c. 2ndly, By pressure on some important organ or vessel, producing effusion; this result is most dangerous in organs surrounded by unyielding structures, as the brain and spinal cord. 3rdly, By the degenerated tubercular products becoming absorbed and producing purulent infection. 4thly, By small particles of tuberculous matter becoming drawn into the general circulation and plugging up or obliterating vessels of supply, producing, as a consequence, degeneration and atrophy of organs; but, according to M. Thaon, obliteration of vessels may be due to coagulation of blood in the vessels, or to vegetating endoarderitis, though in some instances the organ may be saved from destruction by the formation of new vessels in the neighbourhood. Obliteration of the cavity of a vessel may further be produced by the pressure of large tubercular deposits in juxtaposition to its external coats. 5thly, By appropriating the albumen of the blood for its own nourishment and depriving the system of it, thereby resembling a parasite; this is more often the case when the tubercle is situated outside organs, as in the pleura and the peritoneum. 6thly, By hæmorrhage (apoplexy); if into the brain substance, or at its base, producing pressure and organic disease, as softening; if into the bronchial tubes, asphyxia, with their attendant symptoms.

It frequently happens that with the tuberculous growths we have independent disease of one or more organs existing, quite sufficient in extent to account for death; thus, we may have hepatitis followed by consolidation without any tubercle in the liver, or organic disease of the kidneys—producing death—with the presence of tubercle in the lungs or other organs. This is a matter of importance in a medico-legal point of view.

POST-MORTEM CHARACTERISTICS.

Tubercle may be confounded *post-mortem* with a great variety of morbid products. In order to distinguish it from these it is necessary to bear in mind the anatomical characters of Miliary Tubercle in its primary and secondary conditions. It forms small hard nodules, is intimately connected with the tissues in which it is deposited, in fact the tissues interpenetrate it, and when removed by decomposition or by insects—as weevils—one or more small characteristic perforations are observed in its structure. The small nodules when deprived of adventitious matter, which is best done by the

weevil, vary in size from a pin's point to a radish seed, somewhat irregular, but with a tendency to a spherical shape and of a greyish colour.

The nodules are frequently surrounded by a large quantity of firm new connective tissue, the result of exudation and organisation of lymph, which may, and frequently does, undergo degeneration simultaneously with degeneration of the nodules, or becomes indurated and in some cases even cartilaginous.

The hard miliary tubercle in the interior of organs induces hyperplasy of the connective tissue (Fig. 5, *b*, Plate X.) which causes aggregation and insulation of the nodules.

The degenerative processes are principally softening, which commences centrally (Fig. 31 *c* and 30 *d*, Plate VIII.), and cretification, though the latter may succeed the former if it is not very rapid in its progress; usually, as it softens, the amount of surrounding tissue increases and condenses, constituting a veritable capsule (the old so-called encysted tubercle), which in rapid softening becomes extremely hyperæmic.

In some cases it will be found that as the tuberculous mass softens, bands of lymph (granulations) interpenetrate it from the internal surface of the surrounding capsule. In all forms of secondary change, the new interpenetrating and insulating tissue, being lowly organised, is very apt, as seen in Fig. 2, *d d*, Plate X., to become invaded by Tubercle from adjacent parts.

The depth of colour in Tubercle varies very materially with its age, and the changes —primary or secondary—which it undergoes; thus, as already indicated, the primary miliary growths in the earliest stage of subacute or chronic Tuberculosis of serous membranes are usually of a pale grey colour, while in acute infective Tuberculosis they are of a bright scarlet hue; in either case they subsequently present a translucent fibrous appearance which gives place in due time to varying degrees of grey and yellow, these being regulated by the advent and extent of degenerative processes. The different degrees of colour are well depicted in Figs. 30, 31, Plate VIII.; Figs. 1, 2, 4, 5, Plate X.; and Fig. 33, Plate IX. respectively.

The miliary tubercles do not coalesce unless, in common with the surrounding tissues, they undergo degenerative changes; they remain discrete, but very frequently, both in the interior of organs and on serous membranes, become aggregated together to form large masses, the weight of which, with the new tissues connecting them, may be very great: thus, from the diaphragm of a three-years'-old heifer I removed on one occasion a mass which weighed 28 lbs., composed solely of grey nodules and new material. In organs—as, *e.g.*, the lungs—the new growths, with the tubercular products, sometimes lead to an enormous increase in size and weight. The lung from which the section represented in Fig. 2, Plate X. was removed weighed upwards of 60 lbs.; a liver removed from a cow by the late Mr. Dewar, of Midmar, being nearly of the same weight.

It must be distinctly borne in mind that in none of its stages can Tubercle, as

Tubercle, be said to possess vascularity, very rarely indeed are blood-vessels seen to penetrate its structure, and even when present, are very superficial and quickly disappear; they never form in the interior of a tuberculous nodule.

In the horse, Tubercle may be confounded with three products—

1stly, *Psammomata* in the brain; which have been described as tuberculous, but which differ materially, both anatomically and clinically, from this disease. Psammomata are most likely to be confounded with Tubercle in the early stages of their growth, especially when situated in the choroid plexus of the fourth ventricle. Coalescence, or rather aggregation, taking place in a manner somewhat similar to that of tubercle, tends to heighten the naked-eye resemblance between them.

2ndly, *Lymphadenoma* bears, in its history, method of dissemination from pre-existing centres, and clinical characters, the closest analogy to Tubercle of any morbid product with which I am acquainted; in the particulars mentioned it is the Tuberculosis of the horse. It differs, however, from Tubercle in the fact that it is occasionally seen in all animals, though resembling it in most frequently choosing for its victims certain species (as the canine and equine), in which it may commence in the lymphatic glands, and extend thence by a process of auto-inoculation to almost every tissue in the body, exactly in the same way as Tubercle does in acute tubercular infection, and when so disseminated the secondary nodules are often very small in size.

The important distinctive characteristics of Lymphadenoma are, that it is nearly always found in the spleen; that it is not encapsuled by dense connective tissue, but continuous with the structure of the organ in which it is developed; is of a white or greyish-white colour, sometimes soft and brain-like in consistence, at others firm; in the latter case large capillary vessels are seen radiating towards the centre of the nodules. It does not show much tendency to retrogressive changes, and acute (infective) secondary processes—in mucous membranes or otherwise—are usually accompanied by considerable effusion of serum and exudation of lymph into the tissues; consequently, the capsules of organs become considerably thickened and vascular. It is frequently accompanied by melanosis, and, like Tubercle, it is, in one sense, malignant. The characteristics of Lymphadenoma are well seen in the section of spleen depicted in Fig. 36, Plate IX., though the sketch is somewhat diagrammatic, as the original drawing was lent to a friend and mislaid. The nature of the connection of the new growths in Lymphadenoma, with the structures in which they are developed, is best understood by removing from the spleen the whole of its pulp by washing.

3rdly, *The Miliary Nodules and the Bronchial Ulcers of Pulmonary Glanders*, the distinction between which will be considered in speaking of Pulmonary Tubercle.

4thly, *In Degenerated Multilocular Hydatid Cysts* the cyst-wall remains intact, the laminæ of the hydatids themselves having become degenerated, shrivelled, and of a yellow colour (Fig. 3, Plate X.), but still preserving their stratified character. The degenerated hydatid structures can be easily removed by a little pressure, from the cyst walls leaving

behind numerous distinct loculi. The surface of the organ in which the cysts are contained—*i.e.*, if they are superficially situated—is nodulated.

TREATMENT OF TUBERCULOUS DISEASE.

In a discussion on this subject—which arose out of a paper read by me—at a meeting of the Lancashire Veterinary Medical Association in July last, I was taken to task for not giving the members of the Association any information as to the *cure* of Tubercle. My answer was, "That I did not perceive how I was to be blamed for not devising measures for the combating of a disease which had taxed the energies and defied the efforts of many successive generations of physicians and pathologists;" and, moreover, "that as no practical purpose could be served by curing a tuberculous animal —seeing that it was useless for breeding, dangerous for dairy purposes, valueless and dangerous as a companion, and its flesh nocuous for human food—I failed to perceive why time should be wasted in such an inquiry, but rather that all our energies should be directed towards the prevention and eradication of the disease."

The remarks above quoted I here homologate, and consequently shall not allude further to the subject.

PREVENTION OF TUBERCLE.

A consideration of the causes furnishes the key-note to that of prevention, and all that can be said in the matter can be summarised in a few sentences.

1stly, All flesh and offal of affected animals, especially in the advanced stages of the disease, should be destroyed by fire or otherwise.

2ndly, All suspected animals should be carefully isolated until pathognomonic signs have become developed.

3rdly, All actually affected animals should be slaughtered.

4thly, All contaminated food, litter, &c., should be disinfected or burnt.

5thly, All infected houses should be disinfected.

6thly, No animal whose history is tainted, even in the slightest degree, or in whose system there exists the least suspicion of Tubercle, should be used for breeding purposes.

7thly, Great care should be exercised at the period of birth to avoid any influences which will weaken the tissues in adultism.

8thly, Breeding animals should be carefully shielded—as far as is practicable—against debilitating influences of any kind.

9thly, The system of feeding and general management of our high-class stocks should be regulated on a more rational and conservative basis than that on which it at present rests.

I am quite aware that it may be said, "These propositions are all very true perhaps, but how are they to to be applied in practice?" I know that there will be some difficulties in the way of carrying them out in some cases, but this is a matter in which no half measures should be permitted.

Those who pooh pooh these propositions, and who deliberately elect to continue in the paths which they have so long trodden, will one day find their efforts terminate in grievous loss and disappointment.

This subject is one, more for the consideration of individuals than of the State, nevertheless, if the former refuse to recognise the necessity for taking vigorous and prompt defensive measures against the common foe, the latter may yet require to see what can be effected by legislation.

CONDITION OF THE TISSUES OF THE BODY AND THE BLOOD IN, AND THE SANITARY ASPECT OF, TUBERCULOSIS.

In acute Tuberculosis, and especially if it is of a very infective type, the magenta hue, with albuminous effusion; or the iridescent glint already referred to in speaking of the other diseases, may be observed in the muscular tissues. If the lungs have been the seat of extensive and active disease, the muscles may be very dark in colour, soapy to the feel, and flabby. If the tubercular cachexia has become developed the muscles are atrophied, pale (anæmic), and flaccid, while in all cases where there is effusion they are watery—dropsical.

In those instances in which the process is circumscribed and not very acute, or where the Tubercle is quiescent or obsolete, the tissues of the body and the blood may be, and usually are, perfectly normal in character.

Flesh and Milk.—If the oxidation of the blood has been interfered with, it will—as under all similar conditions—be dark in colour and imperfectly coagulated.

In all cases where there is emaciation the blood is diminished in quantity, pale in colour, watery in consistence, and scarcely stains the skin of the hand as it flows over it; and although it may coagulate quickly it only does so imperfectly.

In the examination of a tuberculous carcase for the purpose of inspection, much difficulty is often experienced in arriving at a just and definite decision, especially when all the internal organs have been done away with. The examiner should, however, carefully examine all the serous membranes—particularly under the diaphragm—and all the lymphatic glands, and by so doing he will frequently obtain valuable corroborative evidence.

As to the question of condemning the flesh of tuberculous animals for consumption as human food, much difference of opinion exists. As I have already stated, the muscular tissues may, to all appearance, be quite healthy, indeed I have never yet seen

Tubercle developed in muscular tissue except in the neighbourhood of tuberculous joints or lymphatic glands, and occasionally in the tracheal and laryngeal muscles; in the larynx depicted in Fig. 1, Plate X., there was more infiltration than I have ever seen in muscle. If the internal organs can be obtained and indications of active processes exist—as softening, ulceration, or inflammation—the flesh should be unhesitatingly condemned. Under any circumstances, the internal organs should be destroyed, and it is a matter for grave consideration whether the flesh of an animal suffering from even a slight degree of Tuberculosis should not be condemned. Certainly, exposure to a high degree of heat will render the flesh innocuous, but can proper cooking always be ensured?

As to the use of milk from animals in which Tubercle is suspected to exist, no two opinions can be held; its deleterious effect, even when exposed to a tolerable degree of heat, has been abundantly proved: nevertheless, with the object of preventing a little loss, I have known gentlemen sell cows for dairy purposes in whose systems they have been told Tubercle existed; and cow-keepers do not always hesitate to add the products of a graped or angleberried cow to the bulk of their daily yield of milk. This matter should be taken cognisance of by local authorities in the working of the recent Privy Council Order referring to dairies and milk shops. It would be far better to give compensation and have even a suspected animal destroyed than allow her to remain in a herd or byre with the probability of spreading the disease to her neighbours and poisoning the consumers of the milk.

Jurisprudence.—It should be borne in mind that if an animal is sold as "healthy," "all right," or "correct," and tuberculous symptoms show themselves subsequently, the vendor is liable to the vendee for all contingent loss if it can be distinctly demonstrated that the tuberculous lesions were in existence at the time of sale.

TUBERCLE ON SEROUS MEMBRANES.

There is perhaps no structure in the body in connection with which tubercular lesions are developed to so great an extent as the serous membranes. The external serous layer of the pericardium—seldom the internal—the pleura, the peritoneum, and the meninges of the brain and spinal cord are all in their turn the seat of tuberculisation. The process in these structures may be *primary, secondary,* or *infective.* In the former, the early inflammation is usually less intense than in the latter, except its localisation has been determined by traumatic influences, in which case it may be as intense as that accompanying the secondary process.

Infective tuberculisation is usually the result of dissemination of virulent particles by the lymphatics from a neighbouring caseous centre—as, *e.g.*, the pleura from a bronchial or mediastinal lymphatic gland; the pericardium from a bronchial

or cardiac; or the peritoneum from a mesenteric, or other abdominal lymphatic gland.

The early changes in the process are diffuse and intense capillary congestion, followed rapidly by the formation of innumerable, villous-like, vascular processes; very minute and giving the membrane an appearance closely resembling the pile on red velvet.

In the course of time the extreme vascularity of these little processes passes off, they assume a definite shape and become converted into small hard globular nodules, of the colour of connective tissue; gradually, however, they become grey and somewhat translucent on section, and constitute the so-called grey or fibrous tubercle. These grey miliary nodules may remain discrete, and scattered over the surface of the membrane like millet seeds; they may become connected together by delicate bands of new connective fibrous tissue (Fig. 33, Plate IX.), forming the so-called *grapes* of England, the *angleberries* of Scotland; or they may become aggregated together, as already indicated, and form immense masses which may degenerate in particles or *en masse;* they may remain fibrous.

When tuberculisation is very rapid, distinct polypoid processes, as seen in Fig. 7, Plate X., may be formed on the membranes. These bodies are often at the outset very vascular, in fact, hæmorrhagic (Fig. 7, *b*, Plate X.); somewhat conical at the free extremity and subsequently becoming on their external surface of the normal colour of the serous membrane. Section through them reveals, in the early stages, a fibroid tissue; in the later, numerous nodules of tubercle in various conditions. In some instances these polypoid bodies grow to a very large size, and not infrequently become united together by adhesive inflammation.

Tuberculisation of serous membranes gives rise to *effusions* into the cavities which they line, to *adhesions* of contiguous surfaces of organs, and of the visceral and parietal layers of the membrane itself. The results of effusion are the same from this as from any other cause—viz., *hydrops pericardii* if into the pericardium; *hydrocephalus* if into the lateral ventricles or subarachnoidean spaces of the brain; *hydrorachitis* in the spinal sheath; *hydro-thorax* in the chest; *ascites* in the abdomen, and *hydrocele* if into the tunica vaginales of the testicle.

The symptoms indicative of these dropsies are—in the pericardium, increase in the area of cardiac dulness; masking or irregularity of the heart-sounds, and, if the sac is not too full, a metallic tinkling, not at all unlike the movements of a clock pendulum in a basin of water, synchronous with the cardiac pulsation: in the thorax, bulging of the chest-wall, difficult (laboured) breathing, dulness on percussion and auscultation up to the line of the water-mark, and occasionally, a gurgling or splashing sound synchronous with the respiratory movements: in the abdomen, to a pendulous condition of the walls of that cavity, dulness on percussion—except at the upper part where the organs are not subjected to the pressure of the fluid and where resonance replaces dulness—fluctuation on palpation, thoracic breathing, and, if the effusion is excessive,

to uncertain gait: in the tunica vaginalis, to a fluctuating painless tumour; and in the cranial and spinal cavities to symptoms pathognomonic of pressure on the brain and spinal cord, and which will be noticed at greater length in speaking of tuberculisation of those organs.

The use of an exploring needle, except in the two last situations, will always determine the presence or absence of effusion. I have occasionally seen dropsy of the pericardium, chest, and abdomen in the same animal; the amount of effused fluid being almost incredible. Effusion, according to its extent, is associated with more or less emaciation, anæmia, and local external dropsies—dropsy of the dewlap being particularly pronounced if there is hydrops pericardii.

Effusion may, and frequently does, result from pressure of the tuberculous growths on the veins of a part or organ, and the serous membranes themselves, with the subserous tissues, sometimes become thickened to the extent of a quarter or half an inch.

Exudations, by forming connective bands or layers, circumscribe the action of the organs with which they are connected, and, as they contract, may produce partial obliteration of the calibre of an intestine or other tube.

The large masses which form on the pleuræ and peritoneum frequently give rise to displacement of adjacent organs—as the heart, lungs, liver, &c.—and, by compression, interfere materially with their function.

The presence of miliary tubercle on the pleuræ is marked by dulness, on percussion and auscultation, in patches, or over a large surface; and, occasionally, by bulging of the ribs in the later, and pain on pressure and friction-sounds on auscultation in the early stages.

There may be entire absence of cough and little or no interference with respiration if the lungs are unaffected.

The indications of tuberculisation of the peritoneum are even less marked than in the case of the pleuræ. In the early stages there may be tenderness on pressure and friction-sounds on auscultation; while Mr. Gray, M.R.C.V.S., Bakewell, has recently recorded a case in which he found, in a bull, hard nodules, with some tenderness on pressure over the abdominal region, during life; the introduction of an exploring needle into such a nodule would soon determine its character—examining the contents of the needle microscopically and otherwise—and, in any case, the hand should be introduced into the rectum and the abdominal parietes manipulated.

TUBERCLE IN GLANDS.

Tubercular infiltration of glandular structures is confined to the lymphatic and mesenteric glands. I have only occasionally seen it in the mammary but never in the salivary glands or the pancreas.

The disease in these organs may be primary or secondary. In the case of the LYMPHATIC GLANDS they become separate focuses of infection and give rise to the development of Tubercle in adjacent or widely-distant organs. The lymphatic glands are usually invaded in groups, as those of the larynx and pharynx; the lungs, heart, and trachea; the abdominal, inguinal, or prepectoral ganglia. They frequently attain a very large size, from 1½ to 2 lbs. weight, and the intervening lymphatic vessels are sometimes dilated to the size of a crow's quill, masses of granular tubercle being visible through their coats, which are much distended, and causing the vessels to appear like strings of yellow beads.

Further than abolishing their physiological functions and rendering them separate sources of infection, involvement of the lymphatic glands, unless extensive, does not produce serious mischief. The only exception to this, is in those cases where the enlarged glands contact with easily compressible and important tubes or blood-vessels.

Invasion of these glands, according to some authorities, commences peripherally; I have, however, frequently seen it otherwise, the centre of the gland only being invaded. The structure of the gland ultimately becomes destroyed by pressure and by degenerative processes, as softening (*caseation*) and calcareous degeneration (*calcification*); the gland in the former case, or rather the capsule with its contents, frequently attaining an enormous size. One lymphatic gland which was removed from a cow by Mr. Kerr, of Ballymena, weighed 9½ lbs.

Of the external groups of glands, disease of the laryngeal and pharyngeal ganglia is of the most importance to the veterinary surgeon; as large clusters of these tuberculous or scrofulous organs are frequently congregated round the larynx and pharynx—externally and internally—interfering with the functions of deglutition and respiration. They are also frequently attached to the mucous membrane of the epiglottis, and project into the fauces, or, if the pedicle is long, into the posterior nares or the posterior part of the cavity of the mouth. The nature of their attachment and their size will determine the intensity of the symptoms they induce; thus, if attached by a pedicle they alter their situation with the assumption of the recumbent or erect posture, interfering most with respiration in the former position: if the base is sessile there can be no alteration in their situation, and interference with respiration is constant in any posture. A loud whistling or a snoring sound on inspiration at once directs attention to the condition of the larynx, and, even though the external glands may not be enlarged, the disease may, by the physical symptoms, be easily localised and, with the aid of collateral or coincident evidence, readily diagnosed—if no tumours exist externally the larynx and pharynx should be manipulated through the mouth. The characteristic appearance of these tumours inside the larynx is well shown in Fig. 2, Plate XI. If superficially placed and not too closely adherent to important organs, they should be extirpated by the knife; if projecting into the mouth, by the ecraseur; otherwise, the local application of iodine with the internal administration of anti-scrofulitics is all

that should be done, except the obstruction gives rise to threatened suffocation, when tracheotomy may be had recourse to. It is in connection with these glands that external tuberculous ulcers are formed; they are not often seen in animals.

Cutaneous tubercular ulcers are characterised, previous to external eruption, by the subdermic structures becoming glued to the enveloping dermis and remaining closely adherent throughout the process, and even after the ulcer is healed; the skin at the same time being extensively undermined and thinned. The edges of the ulcer after eruption are thin, tapering, and irregular, and show but little tendency to approximation.

In the treatment of these ulcers the general constitutional conditions must be attended to, and specific medicines—such as iodine, iron, and mercury—administered; the ulcer being treated locally by total excision, or by scarification of its edges and the use of carbolic pads. The glandular enlargement may be reduced by the application of iodine.

The groups of internal lymphatic glands most frequently involved are the mediastinal, bronchial, and cardiac, in the thorax; the hepatic and mesenteric in the abdomen. In some cases I have seen immense masses of enlarged lymphatic glands grouped around the roots of the lungs.

A very material difference is observed in the characteristics of the lymphatic glands in young scrofulous animals as compared with the glands in which tubercle is actually deposited. In the former, the gland is greatly hypertrophied, increased in density, firm in consistence, lighter in colour, sometimes grating against the knife on section (owing to calcareous degeneration of small portions); at other times softened—caseous. In the latter, as already indicated, the organ may attain an enormous size and may undergo softening or calcification, but, prior to these changes taking place, the masses of tubercle are very distinct. In some cases they present appearances closely allied to those depicted in Fig. 2, Plate X.—allowing, of course, for the difference in the structures.

Tubercle in the Mesenteric Glands gives rise to rapid emaciation—hence the term *tabes*, "wasting"—by preventing the absorption of chyle by the lacteals and its elaboration by the glands and by chronic diarrhœa—in the fæces of the dog oil-globules may be detected microscopically. The interior of the lacteals may be filled with miliary granules as in the lymphatics.

In the Mammary Gland.—The Tubercle is usually of the fibrous form. The nodules are very numerous, developed in the acini; but, even when the gland has been extensively invaded, I have not been able to trace any tendency to cretification. The acini are hypertrophied and the interglandular tissue increased, giving the gland, on section, a coarse rarified appearance. Some nodules may be developed on the mucous membrane of the ampulæ or the ducts, but I have neither seen softening or ulceration. The gland in the areas of invasion is of a reddish hue—from increased vascularity.

Involvement of this organ, especially if ulcers are developed on the mucous membrane, is a matter of great danger, inasmuch as the irritation of the tubercle gives rise for a time to increased functional activity of the gland, the secreted milk becoming contaminated with the tuberculous products and propagating the disease to any animal (or man) which may be unfortunate enough to partake of it.

TUBERCLE IN LIVER.

Hepar Tuberculosis occurs less frequently in the ox than Pulmonary Tuberculosis; but, in the rabbit the liver is more often affected than any other organ.

It may arise primarily or secondarily; in the former case running a comparatively slow course, and terminating in the development of miliary nodules, often grouped together like multilocular hydatid cysts; in the latter being more or less acute, and marked by extensive deposits and rapid softening.

Either in acute or chronic Tuberculosis of the liver the symptoms may be quite negative, in fact, they cannot, unless we have collateral evidence or coincidences to guide us, be distinguished from those produced by any other form of organic disease.

If the malady is acute there will be a considerable amount of constitutional disturbance and interference with the normal functions; fever, pain on pressure over the region of the liver, jaundice (*icterus*), hot, dry mouth; constipation of the bowels, scanty high-coloured urine, and, in milch cows, yellowness of the small quantity of milk which may be secreted.

It may terminate in the course of a few days or weeks in death, or may merge into the *chronic* form, in which case icterus will be persistent and marked: emaciation, more or less rapid, ensues; the pulse becomes weak and below the normal standard; the coat staring, and the skin tight to the ribs (*hidebound*): loss of hair (*depilation*), desquamation of the cuticle, and persistent itching (*pruritis*) will also be present.

If the liver becomes enlarged, there will be bulging in the hepatic region, and, if the portal circulation is interfered with, dropsy of the abdomen; the bowels are always irregular, either relaxed or constipated, and very frequently the fæces are clay-coloured.

There may or may not be a cough, and on microscopical examination an increase in the white cells of the blood (*leucocytosis*) may be detected.

Judging from the puckerings which I have sometimes seen on the surface of tuberculous livers, I am inclined to think that in some instances the softened tubercle finds its way through the biliary ducts into the intestines and thus gives relief for a time; it is obvious that in these cases it has not been thrown into the peritoneal cavity, as death, from peritonitis, would almost inevitably have ensued.

On *post-mortem* examination the liver may present a variety of pathological changes; thus, Glisson's capsule may be opaque, thickened and elevated in patches by

the development of tuberculous nodules underneath it, or adhesion to adjacent organs may exist. The liver structure itself is sometimes congested or inflamed; at others, the seat of fatty degeneration in patches. In the latter case it is pale in colour, imparts a greasy feel to the skin of the hand, is very soft, friable, increased in bulk, but diminished in specific gravity, and stains paper when allowed to lie upon it for a short time. The liver cells may be simply infiltrated with fatty matter, or may have undergone fatty degeneration, causing them to lose their natural characters and to become increased in size—fat globules and molecules replacing the cell contents. The tubercular masses vary materially in size, and are not encysted in the true sense of the word, though, if softening has been rapid, a distinct boundary wall of new connective tissue is formed which is sometimes very vascular. In some instances, owing to chronic congestion, the liver becomes enormously enlarged and hardened, and of a dark maroon colour, this, giving place, in the course of time, to a whitish colour as the result of fibroid substitution; which is again followed by wasting—constituting cirrhosis. In some livers all the changes noted may coexist, but its structure may be invaded and altered to a considerable extent without its function of secreting bile becoming totally annihilated. Mr. Dewar, of Midmar, removed a liver from an ox weighing 59 lbs. I have myself seen this organ of an equal size and yet the animal fat.

I have never found tubercle deposited in the coats of the gall bladder, and but rarely in those of the biliary ducts, though we frequently find it in juxtaposition to the latter, and I have, as depicted in Fig. 30, *c*, Plate VIII., also seen it deposited in close contiguity to the portal vein, in fact, on its external surface. If ascites is associated with tuberculous liver the fluid is of a yellow colour.

The section of liver, Fig. 30, Plate VIII., illustrates some of the conditions seen in tuberculous disease of this organ.

Hepar Tuberculosis is frequently complicated by hydatid cysts in the structure of the organ, and flukes (*distomata*) in the biliary ducts.

TUBERCLE IN THE ALIMENTARY TRACT.

I have never seen Tuberculosis of the mouth, palate, pharynx, œsophagus, or stomachs. The formation of tuberculous growths have, however, been noticed in the posterior part of the mouth—*i.e.*, the soft palate, and in the mucous membrane of the pharynx.

In the intestines, the process is—except in the case of ingestion of tuberculous material—usually secondary to some tuberculous lesion elsewhere, the infective matter being conveyed through the lymphatic vessels, or, in the case of pulmonary tubercle, coughed up into the fauces and swallowed.

It is more frequently seen in the small than in the large intestines—occasionally

the cœcum—and is deposited in the lymphatic structures, occurring in circumscribed patches varying in size from a sixpenny-piece to a florin. If the lesions are extensive, they interfere with digestion and assimilation, and rapid emaciation results ; if circumscribed, irregularity of the bowels is induced : in the former case, the fæces of cattle will be fluid and of a fœtid odour ; in either case, they may be tinged with blood, and, by patient microscopical examination, tubercular elements may be detected in them. The appetite is morbid or fickle, and the natural functions of the body more or less interfered with according to the extent of the tubercular disease. In the dog and pig, vomition will, in all probability, be superadded, and pain evinced on pressure.

Examination of the intestines after death shows the coats more or less thickened and, internally, ulcerated ; the ulcer is somewhat radiating in its character, its edges and base thickened, and the former, in some instances, intensely hyperæmic. Prior to irruption of the ulcer, in chronic cases, the mucous membrane is elevated by the tuberculous nodule, which is readily distinguished by its yellow colour ; after irruption, small masses of tubercle are seen adhering to the submucous tissue, and if the ulcer heals—always a slow process—cicatrisation gives rise to irregular puckerings of the membrane, which closely resemble those produced by parasites ; the two forms of ulcers and cicatrices occasionally coexisting.

Perforation of the intestine is extremely rare ; if it did take place, death would be produced by peritonitis. In some instances, the inflammation—extending to the serous membrane—gives rise to circumscribed adhesions between contiguous portions of intestine. In acute infective Tuberculosis the ulcers are sometimes extremely numerous and grouped together in longitudinal patches ; they are of a circular form and soft : there is, however, owing to the rapidity of the process, an absence of the usual tubercular elements in the base of the ulcer.

TUBERCLE IN THE KIDNEYS.—Tubercle may invade their structure, it may be deposited underneath, or upon, their capsules or in the connective tissue of the mucous membrane. The common symptoms, are those produced by other diseased conditions of these organs ; the positive, are the presence, microscopically, of blood, pus, lymph casts, tubercular elements and *débris* in the urine ; with albumen-urea, pain on pressure over the lumbar region, and the detection, by manipulation through the rectum, of the enlarged kidneys themselves.

Post-mortem examination reveals, in some instances, tubercular abscess in the pelves, which, with the ureters, may be dilated, and the mucous membrane ulcerated.

If the lesions are located in the structure of the organ, it will be found excessively vascular, and, on section, parti-coloured—yellow and red. Renal Tuberculosis is occasionally associated with hydatids, and when it is extensive the tubercular cachexia becomes established.

TUBERCLE IN THE GENERATIVE ORGANS.

In the Male, tubercle is deposited—

(*a.*) In the structure of the testicle, producing *tubercular orchitis*, with the usual symptoms of inflammation of these organs—viz., heat, pain, swelling, and tenderness; with abduction of the limbs, lameness or stiffness, and more or less fever according to the activity or chronicity of the process. If it is slow, another symptom may be superadded—viz., a constant desire for copulation (*satyriasis*); though in the course of time the function of the gland becomes abolished.

(*b.*) In the tunica vaginalis, producing dropsy (hydrocele).

(*c.*) In the structures of the cord, producing scirrhous.

(*d.*) In the vasa deferentia, obliterating their calibre, and preventing the transmission of semen.

(*e.*) In the prostatic bodies of the dog, producing pressure on the urethra, retention of urine, and chronic inflammation of the coats of the bladder—leading to thickening, sloughing, and ulceration.

In the Female, the neck of the uterus, the os uteri, and the ovaries are the usual seats of Tuberculosis.

In the two former situations, it gives rise to straining and the voidance of thin, ichorous, pus and mucus, in which may be detected, microscopically, tubercular products. Constriction or hypertrophy of the os uteri, forming an obstruction to future parturition, may result.

In the latter situation sexual desire and persistent œstrum (*nymphomania*) is induced, with obliteration of the Fallopian tubes—the latter producing sterility. The deposition of tubercle in the ovaries frequently explains the reason why tuberculous animals so often suffer from excessive œstrum.

In whatever part of the uterus the tubercle may be located, abortion, in pregnant animals, may result.

The enlarged ovaries may be easily detected in the cow by exploration per rectum; and the condition of the os and cervix uteri per vaginum.

The mucous membrane of the vagina is rarely invaded; in one remarkable case of acute and extensive infective Tuberculosis which recently came under my notice the walls of the uterus and vagina were each the seat of tubercular depositions and ulceration, the former giving rise to prominent longitudinal elevations in the mucous membrane, and, in one of the ovaries, a large cyst was formed which contained a great quantity of puriform matter: the fimbriated edges of the Fallopian tube were enormously hypertrophied.

TUBERCLE IN THE BRAIN AND SPINAL CORD.

Tubercular deposits in connection with these organs are nearly always of secondary origin, and are, primarily, located in their coverings (*meninges*) either in the vascular membrane (*pia mater*), or the serous (*arachnoid*) ; but, either as the result of infiltration of tubercular elements from one of these membranes, or from the commencement of the process in the sheaths of the small vessels, the substance of both the brain and the spinal cord may become the seat of Tuberculosis; the tubercular masses in this situation being larger than in the membranes, grey in colour, and somewhat translucent.

According to Green, the process originates in the peri-vascular lymphatic sheaths which surround the small blood-vessels.

The granules in the brain are always small, more frequently than otherwise discrete, and most numerous at its base, and in the choroid plexuses of the lateral and fourth ventricles, in which parts the granulations sometimes agglomerate and form large tumours, which, by pressure, give rise to a considerable amount of absorption of contiguous structures. The tubercular deposits in this region originate, by the irritation they produce, a variety of lesions, the most prominent of these being inflammation, constituting *tubercular meningitis,* and giving rise to the formation of large masses of new connective-tissue structure, and, consequently, to adhesions; and to effusion of serum into the ventricles (*acute hydro-cephalus*), or in the interspaces of the serous membrane (*subarachnoidean spaces*) at the base of the brain. Effusion may be produced by mechanical pressure upon the vessels simply, and in the same way congestion and extravasations (*apoplexies*) result; the latter also being occasionally produced by ulceration of the coats of the arteries.

The miliary lesions in the meninges of the spinal cord are not confined to any particular part; they frequently bestud the whole of its circumference, and give rise to the same pathological conditions as in the cerebral meninges, the most prominent result being paralysis.

Tubercular meningitis is seldom seen in old animals; in fact, as in the human subject, it is mainly confined to the earlier periods of life—*i.e.*, in calves, up to six or nine months. I have seen it in a bull of about four years old, and one case, to which I allude hereafter, occurred in the practice of Mr. Corbett, of Hexham, in an adult animal. The pathognomonic symptoms are somewhat obscure as they are closely allied to those produced by other subacute or chronic morbid processes which go on in the cerebral substance or the meninges, more especially to those produced by hydatids ; and in the majority of instances nothing of importance attracts the attention of the owner of an animal until effusions or extravasations result, when, by the aid of collateral circumstances—as the animal's appearance and condition and previous history, or concur-

rently the presence of external scrofulous tumours—the veterinary surgeon is enabled to arrive at a tolerably certain diagnosis. The relation of Mr. Corbett's case will give a good idea of the probable symptoms and results. The animal, a heifer three and a-half years old, was seen by Mr. Corbett in June, 1874. She had been noticed, as the farmer said, "peculiar" for about four months—there being hanging of the head with slight inclination to the right side. When Mr. Corbett saw the heifer she was lying in the pasture chewing her cud, and upon rising stretched herself; on being made to move, however, she staggered and inclined to the right, occasionally standing still and attempting to balance herself; closer examination revealed blindness in the right eye, and slight mucous discharge from both nostrils (that from the right being most copious): the pulse and respiration were normal. Mr. Corbett's diagnosis, and also that of his father, was "hydatid in the brain," and on communicating with me and detailing the symptoms, I concurred in the diagnosis. Trephining was had recourse to without satisfactory results, and the animal was destroyed. The autopsy, which must have been very carefully carried out by Mr. Corbett, revealed the following lesions which satisfactorily explained the symptom during life and rendered obvious the reason why the trephining failed to give relief.

The miliary deposits in the pia mater were extensive, especially in the velum interpositum, crura cerebri, and hippocampi; in the two latter positions the resulting tubercular lesions were most prominently marked—diffused tubercle existed also in the diploe of the cranial bones of the right side, *i.e.*, in the occipital and temporal, their structure being softened and expanded. The right nates contained two small pea-shaped masses of caseous tubercle, left nates healthy; a small mass escaped, like the kernel of a nut from its shell, from the left testes, the right testes being normal. The pia mater was highly injected in the regions above noticed. Mr. Corbett forwarded to me portions of brain structure which were much softened and filled with miliary granules.

In some cases paralysis is the most prominent symptom, the mind being free, the pupils of their natural size, and all the functions performed properly, but the animal may be unable to elevate its head in the slightest degree. This was seen in Mr. Borthwick's case, as detailed by me in the *Veterinarian* for September, 1872, p. 686. The temperature, respiration, and pulse are not much altered, except in acute cases. In the case of the four-years'-old bull—a valuable shorthorn, which I saw in consultation with Messrs. Tait and Son, of Elgin—the brain symptoms were complicated by a considerable amount of gastric disturbance, but, by taking other evidence into consideration, we had no difficulty in arriving at a correct diagnosis.

Treatment is useless, but, should any be determined upon, the internal administration of calomel, with the application of blisters to the poll, is the only course that can be pursued which is likely to prove of benefit.

TUBERCLE IN THE HEART.

Tubercle is sometimes found in the structure of the cardiac walls; I have never seen it in this situation myself, but a case is recorded (*Veterinarian*, September, 1873) in a two-years'-old heifer by Mr. James, of Thornburn, in which also the more unique lesion of a tubercular abscess in the wall of the left ventricle existed.

The symptoms indicative of cardiac tumour are not positive; there may be intermittent, irregular, weak, or double (*dicrotic*) pulse; but, unless collateral evidence of a positive nature exists, a correct diagnosis can scarcely be made.

TUBERCLE IN BONES AND JOINTS.

Tuberculosis disease of these structures is, as compared with its frequency in the human subject, rare; I have only seen it in cattle and swine.

It may be primary or secondary, usually the latter, and may commence in the periosteum, the cancellated structure, or in the synovial membrane of the joints.

It is most frequently seen in the soft irregular bones, as the squamous temporal and occipital, in the cranium; and in the bodies and superior spines of the dorsal vertebræ; originating in the diploe or cancellated structure, and, owing to the amount of inflammation and exudation, the bone becomes thickened and softened, and the compact structure absorbed: the tubercular nodules are embedded in the new material (Fig. 34, Plate IX.), and are easily distinguished.

In the long bones, the process commences in the cancellated structure of the extremities, or, perhaps, in some cases, in the lining membrane (*endosteum*) of the medullary canal. The tuberculous products, with the exudative matter, may become surrounded by a callous cyst of new connective tissue; in the great majority of instances, however, it softens, and establishes a tubercular abscess, which may, if situated at the extremity of a bone, cause absorption of the articular layer and the articular cartilage, and evacuate, by the medium of a cloaca (Fig. 5, Plate XI.), into a joint. Very rarely, in animals, do the contents of a tubercular abscess gain exit through the compact structure; if it does occur, a sinus is formed in the superjacent soft structures which defies, in some instances, the power of surgical science to heal. After the evacuation of a tubercular abscess, the medullary tissue is probably never restored, but is replaced by a gelatinoid material.

Necrosis or exfoliation of the compact structure may—as in the human subject—take place; it is, however, rare; it has been described as a cause of bustian fouls

in the coronets and pasterns of cattle: such a case has not as yet come under my notice.

Tuberculosis of the periosteum I have not observed as an independent process; usually, it is secondary to disease of the bone or to tubercular arthritis.

When the structures of joints are the seat of the disease, the lesions (except the pathognomonic one of miliary nodules) are, on the whole, very similar to those of specific arthritis of young animals and, in part, to those of rheumatism—the activity of the processes determining, to a great extent, the relative similarities.

Tuberculosis of Joints may originate—as a primary process—in the synovial membrane, or,—as a secondary process—from the evacuation of an abscess through the extremity of a contiguous bone. In either case it is usually acute, the constitutional disturbance great, and proportioned to the activity and extent of the disease; local pain, heat, swelling, and tenderness are marked: stiffness and lameness, in the case of the extremities, is almost invariably present.

The duration may be quite indefinite when it assumes a subacute or chronic character, but, when extensive and rapid, disorganisation ensues, and death quickly results, either from exhaustion, hectic fever, or pyæmia.

The pathological alterations observable in *post-mortem* examination vary with the activity or chronicity of the process. In either case, the surrounding muscular and connective tissue will be found largely infiltrated with lymph, or thickened and indurated by the exudation and organisation of fibrin. If the process has been active, the component structures of the joint will be found highly inflamed, the articular cartilage softened, degenerated, and separated from the bone; the synovial membrane thickened; and the cancellated structure of the extremities of the bones vascular, softened, rarified, and sometimes ulcerated, with extravasation of blood into the structures or into the cavity of the joint.

If the process is slow, the function of the synovial membrane becomes gradually annihilated; no synovia is secreted, the articular cartilage is removed by attrition, the articular layer of bone exposed, and, ultimately, either worn away—exposing the cancellated structure—or saved from destruction by the deposition of a porcelain-like material (*porcellaneous deposit*) identical with that which is so often seen in rheumatic joints. New bony matter may be also formed on the articular surfaces or round the extremities of the long bones, in the latter position, forming a clasp to the joint and producing permanent stiffness (*anchylosis*): in the majority of instances the neighbouring periosteum is found bestudded with miliary nodules, and, where the joint is clothed with muscles, tubercular infiltration of their structure or connective tissue may have taken place.

TUBERCLE IN THE RESPIRATORY ORGANS.

I have already referred to the fact (*ante*, p. 171) that scrofulous tumours are sometimes developed in the interior of the LARYNX. Tuberculosis of this organ is only found in connection with the mucous membrane, and, more frequently than otherwise, associated with a similar condition of the bronchial or tracheal membrane, or with pulmonary tubercle. As a rule, the process is secondary to tuberculosis of the lungs and commences in the submucous tissue, the tubercular nodules, as they increase in bulk, elevating the membrane, and gradually, by pressure, producing absorption; the tubercle, undergoing softening, becomes evacuated into the cavity of the organ and is expectorated or coughed up into the fauces and swallowed. After irruption a distinct ulcer is formed, having vascular edges (*hyperæmia*), of an oval or circular shape, and, usually, discrete.

In some cases, the ulcers become confluent; in all, after the tuberculous material has been completely cast off, cicatrisation goes on, new vessels being formed, which pass, in a radiating manner, into the surrounding membrane. These peculiarities are well shown in Fig. 1, Plate X., and in the tracheal and bronchial membrane (Fig. 35, Plate IX.)

In the TRACHEA the tubercle is also, at the outset, deposited in the submucous tissue, though in rare instances—as was the case in the trachea, Fig. 1, Plate X.—it commences in the structure of the transverse muscle.

In the BRONCHIAL TUBES the process originates also in the submucous tissue, though in many cases I have not the slightest hesitation in saying that the nodules situated in contiguous lung-structure, produce, by pressure, absorption of the bronchial walls, and, undergoing softening, become evacuated into the tubes; this is best seen by drying a section of lung, with a bronchial tube attached, in which the process is going on.

Diffuse bronchitis does not, as a rule, accompany the process; it is, however, always seen, and of a very intense character, when tubercular elements gain access to the tubes from a neighbouring vomica.

TUBERCLE IN THE LUNGS is, next to tubercle in serous membranes, the most common form of the disease, and the most widely known. It may be primary or secondary—usually the former; it is designated Pulmonary Consumption, or Pulmonary Tubercular Phthisis. To a certain extent, and in some states of the parenchyma of the organs, these designations are correct, in others they are misleading. The word "phthisis" simply signifies wasting, or consumption, of a part, and is applicable to all diseases in which rapid consumption of the solid textures of the body is a prominent feature. Pulmonary Consumption at the present day, too, has a wider significance

than is simply represented by the presence of tuberculous lung lesions; for by the use of this term any pathological condition of the lung marked primarily by consolidation, and secondarily, by rapid disintegration, or wasting, is designated Pulmonary Phthisis.

As Tubercular Phthisis is only one form of Tuberculosis, I need not enter into detail with respect to its production, the localisation of the disease in the substance of the lungs being, in the main, an accidental circumstance governed by no fixed law. Inasmuch, however, as it interferes with the functions of organs of vital importance, upon which, in fact, the due oxidation of the blood depends, it becomes the most fatal form of the affection with which we have to deal. The same conformation of the body is found in phthisical animals as in all tuberculous subjects—viz., narrow chest, small barrel, flat ribs, and, in many instances, a watery, dull appearance of the eye, with a (generally) dull temperament; and it is frequently remarked of such animals, even before symptoms of disease are manifested, that they are more readily acted upon by such external influences as cold, wet weather, exposure, bad keep, and fatigue.

It is this form of Tuberculosis which is transmitted, more than any other, from animal to animal, as not only is the expired air charged with infective particles, but large quantities of infective tubercular elements find their way through the expectorations into the grass and food of healthy subjects.

Symptoms.—The symptoms of tubercular phthisis are, in the early stages, very deceptive; in fact, in the majority of instances, the malady has made considerable progress before its existence is even suspected.

At the commencement of the disease it will be probably noticed that after a very wet or cold day, or a frosty night, the animal is seen isolating itself from the rest of the herd, its back being arched and its coat erect; if made to move it does so stiffly and unwillingly, giving vent at the same time to a slight irritable cough or hoose, and it may be an occasional grunt: pressure over the ribs or back at this stage may or may not give rise to an expression of pain. The appetite is indifferent, and the bowels irregular. Much thought is not often devoted to the reading of these symptoms, and the owner, looking upon them as the result of a slight cold, has the animal placed under shelter and a dose of warm ale and ginger administered to it, with the allowance of a few mashes to eat, and under these favourable conditions the ailing beast appears to rally and is turned out with its companions until the cause which gave rise to the apparently temporary illness is again brought into operation and reproduces it. It is this recurrent character of the early stages of tubercular phthisis which serves, to some extent, to distinguish it from most other lung affections.

If the attention of a veterinary surgeon is called to the animal at this stage he will in all probability discover slight disturbance of the respiration and circulation with irregularity of the temperature of the body; on auscultation of the chest a rasping sound may be detected, and here and there, on percussion, a non-resonant patch.

As the disease advances the symptoms become more characteristic; the appetite is fickle, rumination and bowels irregular, purgation easily induced; the muzzle may be bedewed with moisture even up to the last stage, and if the animal is a milch cow, milk, though in gradually diminishing quantity and thinner in quality, may be secreted. The eye is sometimes very deceptive, remaining bright up to a late period in the development of the affection, but on careful observation it will be seen that a certain amount of anxiety pervades the countenance; the pulse and respiration, except in being increased in frequency, may remain normal; the skin becomes harsher to the feel, often contracts an icteric hue, and is more tightly adherent to the ribs: emaciation commences and goes on very rapidly.

The lungs give greater evidence of disease by increased frequency in the cough which is caused by reflex irritation or by localisation of disease in the laryngeal or bronchial mucous membrane. If there is bronchial or laryngeal ulceration, or a communication between a tube and a tuberculous cavity, the cough may be accompanied by expectoration of mucus, or mucus, pus, and tubercle *débris*. On auscultation, a variety of sounds, pleuritic and otherwise, will be heard; there will be more marked fatigue on exertion, and indigestion and tympany may be troublesome.

If the symptoms assume an acute character at this stage, it may readily be mistaken for Zy. P. p.

In the last stages, all the before-mentioned symptoms are exaggerated, the diarrhœa is constant, the fæces frequently having a peculiar leaden appearance and often being discharged without an effort on the part of the animal, while bubbles of gas form on the surface of the excreta. If the animal happens to cough during the act of defœcation the fæces are violently projected to a considerable distance, hence the term "*shooter*" is often applied in the Midland counties to an animal in this condition; the diarrhœa may be due to secondary disease of the intestines. The cough is very frequent and annoying and sometimes possesses the characteristics of the Pleuro-pneumonia cough. The eyes become sunken, the mucous membranes pale and anæmic, the body extremely emaciated, the hair falls off, the breath is fœtid, and local dropsies make their appearance. The first evidence of dropsy is in the dewlap or in the subcutaneous cellular tissue underneath the lower jaw; in the latter situation, from its dependent character, the cognomen of "*wattles*" is frequently applied to it.

The physical lung symptoms now depend upon the changes which have taken place. There will be consolidation in patches, intersected by pleuritic friction. If an abscess has formed and burst into a bronchial tube a *cavernous râle* will be heard; and if the bronchial tubes are partially obliterated, or a direct passage is formed through an abscess, a whistling sound. On percussion, patches of consolidation will be detected by dulness, with, here and there, hyper-resonance, as the result of the emptying of an abscess, or, of emphysema of the more healthy portions of the lung.

The extreme symptoms of this last stage are, the expectoration of mucus inter-

mingled with blood, small particles of broken-up lung-tissue, and degenerated tubercular elements; occasionally a large quantity of blood is ejected, constituting *hæmoptysis*. The cause of hæmorrhage is due, according to some authorities, to the formation and bursting of minute aneurisms of the branches of the pulmonary arteries. In cattle it more frequently results from atheromatous changes in, or fatty degeneration of, the coats of the arteries, as the result of irritation produced by the deposition of tubercle within their structures, leading to ulceration. It is frequently due to ulceration of the bronchial membrane. If pus is absorbed into the blood, septicæmic symptoms will be developed, and, on the advent of ulceration, hectic fever is superadded.

If the tubercular formations in the chest are of great size, bulging of the ribs will be apparent; and if on the left side, the heart will be displaced; if one large, or numerous small abscesses have burst and discharged their contents into the bronchial tubes, collapse of the walls of the chest will be observed; more especially is this the case if adhesion exists between the external surface of the abscess and the internal surface of the walls of the thorax. If there is hydro-thorax we have the usual indications of that condition.

The rapidity of the progress of tubercular phthisis, and in a great measure the urgency of its symptoms, will depend as much upon the degenerative changes going on in the tubercular formations as upon the primary lesions, as frequently we find, where there is a tendency to insulation, or the tubercle becomes obsolete, that a long time may elapse before urgent symptoms are observed, and all the natural functions (though somewhat irregular) may be performed for months or even years.

Death results from exhaustion, from the destruction of more lung-tissue than is compatible with the functions of life; or from suffocation produced by the rupture of a vessel and hæmorrhage, or the bursting of an abscess; and, not infrequently, from accompanying heart disease.

Diagnosis.—Perhaps there is no form of Tuberculosis which is more likely to be confounded with other affections than this, and the reason is not far to seek. It is simply, *that so many pathological conditions, totally different in themselves, give rise to the same physical lung symptoms and general constitutional indications.*

The diseases with which we have the most difficulty in diagnostic differentiation are—Chronic Lobular Pneumonia, P. p. zy., and complications of Verminous Bronchitis (hoose) or hydatid disease, with either Tuberculosis, Lobular Pneumonia, or P. p. zy.

In *Chronic Lobular Pneumonia*, whether due to the inhalation of foreign matter, to the irritation of parasites, or any other cause, we have gradual emaciation, elevation of the temperature, increase in the frequency of the pulse, and acceleration of the respiration, more or less marked: the normal functions are always interfered with; cough irritable and frequent, accompanied sometimes by expectoration of viscid muco-

purulent matter; and the physical signs may be in every sense identical with those of Tubercle, while (as in phthisis) a fresh impetus is often given to the progress of the disease by exposure to depressing or adverse influences, such as cold, or cold and wet; parturition, œstrum, or intercurrent disease of a debilitating nature.

Broncho-pneumonia phthisis, equally with tubercular phthisis, is always aggravated and rendered more difficult to diagnose by the accidental passage of medicines or particles of food into the bronchial tubes, and, in young animals, by strongyles gaining access to them.

To *Zymotic Pleuro-pneumonia* I have already referred; nevertheless, I do not think it will be out of place to insist still further upon the great probability of the two affections being confounded with each other, especially if Zy. p. p. exists in a herd. The brief relation of one striking case will illustrate the difficulties sometimes encountered by the practitioner in diagnosing disease. Recently, in the course of my duties as Inspector, I observed—in a byre from which a Zy p. p. cow had been removed about four weeks previously—a shorthorn cow, which presented the following symptoms:—pulse 78 and weak, respiration 30, temperature 106⅘° F., harsh, dry skin with some icterus, and depilation; protrusion of the nose, cessation of lactation, anorexia, collapse of the flanks, and, on auscultation, puerile respiration left, absence of sound, with considerable tenderness on palpation at the postero-inferior part of right, chest; bowels irregular. I had seen this animal seven days before, and did not observe anything abnormal about her, in fact she presented the usual appearance of good health, and the attendant declared to me that she had only been noticed to fail in her appetite two days previous to my last visit.

While I had, in my own mind, little doubt of its being a case of Zy. p. p., I was, nevertheless, not sufficiently satisfied to justify me in ordering her removal to the abattoir for slaughter, and I left the owner the option of either sending her to the slaughter-house or keeping her in the byre for two days, when I would make another and more careful examination; he chose the former course, and on making a *post-mortem* examination immediately the cow was killed, I found one of the most beautifully-marked cases of Acute Miliary Tuberculosis I have ever seen, with an enlarged softened bronchial gland, and a patch of consolidation, with tuberculous softening, in the right lung at its extreme postero-inferior part; and some irritation of the bronchial mucous membrane.

Now, the question naturally presents itself, how are we to differentiate such cases? I see no other course open than the free use of the exploring needle, neither can I see any objection to its far more extended application in the diagnosis of all lung affections in which there is consolidation.

These difficulties do not so often cross the path of practitioners in districts where no Zy. p. p. exists, and where reliable collateral information as to the history of a case can be obtained; but when an opinion has to be given under opposite conditions the difficulty is sometimes very great.

Pathological Anatomy.—The *post-mortem* characteristics will depend upon the stage of the disease, the extent of the lesions, and the changes which the neoplasms may have undergone.

The pleura may or may not be involved; if it is, the appearances I have already described, in speaking of serous membranes, will be observed; probably accompanied by water (*hydro-thorax*), with flakes of soft, newly-formed lymph in the chest, and organised adhesions between the pleuræ. If the tubercular deposits are confined to the interior of the lung, the external surface may be parti-coloured (red, yellow, and pink), and somewhat nodulated and uneven; frequently, however, the pleura is pale in colour and smooth, though the nodules can be distinctly felt on manipulation in the deeper portions of the lung.

The involved portions present essential (tuberculous) and accessory (non-tuberculous) lesions; are more or less condensed (not often consolidated as ordinarily understood), somewhat heavy, non-resilient, do not crepitate; and, if calcareous, grate against the knife when cut. In the advanced stages all lung-structure is annihilated—no semblance of it remaining—and if it is carefully separated from pervious bronchial tubes and blood-vessels, and loose connective tissue, will sink when placed in water; but if a small portion of pervious, uninvaded, lung, or several tubes and blood-vessels, remain attached, it will float, partially or absolutely, according to the amount of buoyant (pervious) structure.

In the lung, as in other organs, the tuberculous lesions may be primary or secondary, or both, *i.e.*, we may find, on section, primary Miliary Tubercle developed in the inter-alveolar connective tissue—the nodules in their earliest formation, and in acute Tuberculosis being grey, somewhat translucent, firm, and small; and composed mainly of small-celled lymphoid tissue, with an intervening delicate fibrillated net-work. In chronic cases the nodules are usually large—varying from a millet seed to a pea—of a yellow colour, intimately adherent, though capable of being detached by a moderate amount of pressure, by delicate processes to the surrounding structures; they are non-vascular and show a tendency to central degeneration. At the outset they are (although somewhat grouped) discrete, and separated from each other by connective tissue which is usually hyperplastic (Fig. 5, Plate X.) As the disease advances, however, and they become aggregated together, the whole mass frequently undergoes softening or calcification simultaneously—the degenerative change commencing in the centre.

In very acute—as also in secondary—Tuberculosis the growths are much larger, located primarily in the alveoli, yellow in colour, composed largely of catarrhal products, with multinucleated cells, and show a greater tendency to retrogressive changes. The usual appearances presented by these larger masses are well depicted in Fig. 31, Plate VIII.

As the softening process goes on the tuberculous deposits become surrounded by a tolerably thick insulating wall or envelope of condensed and new connective tissue;

and, as in encysted pleuro-pneumonia lung, the inner surface of this boundary wall frequently becomes very active—granulations forming and interpenetrating the tuberculous mass. In some instances these softened masses coalesce, and, if the boundary wall preserves its integrity, form large tubercular abscesses, the contents of which, owing to the absorption of the fluids, may again become dry and caseous; but if the cyst wall becomes the seat of ulceration (a comparatively common occurrence), a communication is formed with the nearest bronchial tube, and the contents evacuated into it, subsequently being expelled by expectoration or coughing. In these cases the boundary wall is frequently very hyperæmic, with numerous small yellow nodules adhering to its internal surface, and evidence of recent hæmorrhage (Fig. 38, Plate IX.) I have in my possession a specimen of concurrent hydatid disease and tubercle, in which a communication exists—by ulceration—between the hydatid cyst and the tuberculous cavity.

After the contents of a tuberculous cavity are evacuated, its sides collapse, become adherent, and produce a distinct puckering (cicatrix) on the pleural surface, which (Fig. 37, Plate IX.) can be readily distinguished from other cicatrices by the presence of infiltrated tuberculous matter in the surrounding tissues.

The condition of the adjacent lung-structure will depend upon the activity of the process and the amount of compression exerted by the tuberculous masses and the hyperplastic tissues. If the compression is not great and the process slow, the lobules may be very little altered in structure, but if the opposite conditions obtain, they are usually more or less solidified—*i.e.*, imperfectly hepatised or carneified (Fig. 2, *b*, Plate X.), and frequently replaced by new fibroid tissue, which, in its turn, as shown in Fig. 2, *d*, Plate X., may become invaded by Tubercle from a contiguous centre. Collapse of the lower borders of the lungs is common, as is also emphysema of the lungs.

In very severe cases and advanced stages all semblance of lung-structure, as already indicated, is lost, a section revealing large tracts of new connective (frequently indurated, and sometimes semi-cartilaginous) tissue, masses of calcareous, intermingled with others of caseous, material; and, in rare instances, large quantities of fat, with occasional pigmentation of the new tissue (Fig. 2, Plate X.) The lung from which this section was obtained weighed upwards of 60 lbs.; it was removed from the thorax of a four-years'-old ox, the carcase being in prime condition; the other lung, however, was but very slightly invaded, and the tuberculous masses in the diseased organ were all obsolete.

In some instances we find caseous centres of various sizes, with Miliary Tubercle developed, within a limited area, around their circumference, as the result of *auto-inoculation* by the lymphatics.

If the arteries become involved in the process, minute ulcerations can be detected in the internal coats.

In almost every instance of pulmonary phthisis the bronchial, mediastinal, or other thoracic glands are found more or less affected, sometimes softened, with secondary Miliary Tuberculosis of one of the other lobes of the lungs.

It is desirable, before dismissing this subject, to glance at the different pathological conditions of the lungs, which, either directly or remotely, resemble those presented in tubercular phthisis.

I have already considered the characteristic differences between it and degenerated Zy. p.p. lung; and the remarks made at pp. 24, 25 (*ante*), in reference to the various forms of lobular pneumonia met with in cattle, will equally apply here. Of this form of disease, the nearest approach I have ever seen to the characteristic lesions of tubercular phthisis occurred in the case from which Fig. 32, Plate VIII. was taken.

In *Pyæmia Pulmonum* caseous nodules and miliary abscesses sometimes exist to such an extent as to produce a certain likeness to the lesions of Tubercle, but, as shown in Fig. 11, Plate X., the degenerated masses are always of a grey colour, composed of catarrhal products, pus, &c., and the intervening lung-tissue is more friable, intensely hyperæmic, and, not infrequently, gangrenous.

In Glanders in the Horse.—The lung lesions have often been compared with those of pulmonary phthisis. The glander nodules, when discrete and widely disseminated, are more regular in size and outline, less firm in consistence, always grey in colour, except in the earliest stage of their formation; are not so intimately connected with the lung-tissue, and, when numerous, are usually grouped together in a racemose manner. The vomicæ of glanders have a totally different character; they are more virulent and, as in pyæmia, surrounded by a boundary wall of intensely hyperæmic tissue, their contents partaking more of the nature of pus, mixed with lung *débris*, and usually possessing a fœtid odour.

The patches of consolidation in glanders are more entensive than in Tubercle, are brawny in character, and present many analogies to the gummata of syphilis, while in Tuberculosis I have never observed the extensive thrombism, embolism, and infarction so frequently seen in glanders.

The ulcers of the bronchial and nasal mucous membrane in glanders are preceded by distinct spherical nodules of a grey colour (at first hyperæmic), the softened matter of which resembles pus, does not adhere, after irruption, to the floor of the ulcer and is never yellow in colour. The succeeding ulcers have, at the outset, inverted edges; are distinctly circular in shape, but show a great tendency to coalesce and assume an irregular outline; they are also of a more markedly specific and virulent character, and their edges are more indurated.

In sheep the pathological changes produced by the strongylus filaria resemble those of pulmonary phthisis more closely than any other condition seen in the lungs of domestic animals, and the resemblance is to some extent heightened by the great increase of late—at least in Scotland—in the number of cases.

In very few instances do I see in the public abbattoirs of Edinburgh and Leith the lungs of a sheep which can be said to be entirely free from these verminous lesions.

In their earliest formation these nodules are preceded by circumscribed vascular spots about the size and shape of a linseed, and, where they are situated in the sub-pleural tissue, produce a distinct depression on the pleural surface. In their degenerated condition they are of a yellow colour, uniform in size and shape (about as large as a small hemp seed), somewhat translucent, hard, and, when under the pleura, cause nodular elevations of its surface.

As in Tubercle, however, these nodules frequently become aggregated together in masses, are interspersed with patches of pulmonary consolidation, and undergo slow softening—producing, in some instances, indolent abscesses.

Again, as in Miliary Tubercle and glanders, they give, both externally and on section, a distinctive nodular feel to the invaded lung; they are not, however, associated with ulceration, and are never found in other organs.

The remarks made as to the differential characters of multilocular hydatid cysts and Zy. p. p. lesions at p. 35 will equally apply to the lesions of tubercular phthisis.

APPENDIX.

THE INSPECTION OF LIVE ANIMALS.

I NEED scarcely observe that, in order to qualify for the office of an inspector, an intimate knowledge of the general habits of animals; the characters, not only of infectious, but of sporadic diseases; and of the alterations produced by exposure, neglect, starvation, travelling, and parturition, is absolutely necessary.

In the case of animals conveyed by water, it is always desirable that the inspector should go on board before the cargo is disturbed for the purpose of landing, as he will, by close observation, be able to detect many circumstances—perhaps slight in themselves, but at the same time pregnant with meaning and guidance—which will assist him in the detection of zymotic diseases.

During this preliminary examination a note—mental or otherwise—should be taken of anything which appears suspicious; and the animal to which it refers should be marked in such a way as to enable the inspector to again recognise it at a subsequent stage of the examination. It must not be forgotten, however, that, where animals have been exposed for many hours to the deleterious effects of foul air—in the holds or between-decks of vessels—or to inclement weather on deck, have been travelled long distances prior to embarkation, have been deprived of food or water for a long period, or, in the case of pregnant animals, have given birth to their offspring *en voyage*, many symptoms will be induced which can be traced to the influences above mentioned, quite independently of the existence of zymotic disease, and which will, in all probability, pass off in the course of a few hours.

During the time the cargo is being landed, the gait, carriage, and behaviour of each animal should be observed.

After the usual period of detention has expired, the inspector, before commencing an individual examination, should walk though the sheds and take a quiet but searching look at each animal, with the object of noticing any abnormal condition of the

respiration, posture, skin, and excreta; depression, anxiety, shivering, uneasiness, or unthriftiness; and, as to whether the animal has eaten anything; or, in the case of a ruminant, if it is chewing the cud; and, if a milch animal, if the udder is active. If an animal is recumbent, it should be made to rise, and any unwillingness, stiffness, or inability to do so noted; also whether it stretches itself. Where the animals are not tied up, any tendency to isolation should attract attention, and lead to more careful examination.

After these preliminary observations each animal should be forced to move about so as to enable the onlooker to detect stiffness, lameness, or uncertainty of movements, and if anything is observed its cause should be at once investigated. As Foot-and-Mouth Disease and Foot-Rot are the only affections which give rise to lameness, I may observe that an examination of the usual seats of vesicles in the former, and of the parts usually involved in the latter, should be made. For all ordinary purposes in the examination of the feet of sheep—where the animals appear otherwise healthy—it is sufficient to grip the hoof and coronet and notice the temperature, as it is always far higher in Eczema Epizootica than in Foot-Rot. Loss of hoof, especially in the case of pigs, or the finding of a cast-off hoof in the yard or sheds or on board a vessel, will always be a matter of grave suspicion. In cattle and sheep the existence of injuries, as foot-sore, wounds, &c., must be allowed due weight in diagnosing Foot-and-Mouth Disease.

After attention has been paid to the feet, the mouth should be next carefully examined—the absence or presence of salivation and smacking of the lips having been previously determined by the eye and ear—and in doing so it is necessary to notice any unusual heat, dryness, or odour, or any difficulty in opening the mouth. The usual seats of vesicles or eruptions should be also noticed, and, while I do not advocate such a proceeding, I may here observe that an experienced examiner can judge of the condition of the mouth, by the senses of touch and smell, as certainly as by that of sight.

While the examination of the mouth is being carried out, a glance should be directed to the eyes and nostrils, with the object of detecting the existence of any abnormal discharges or eruptions; and to the muzzle, in order to detect dryness. Although the mouth can be examined—in the majority of animals—without the aid of an assistant, it becomes a task of great labour when there are many to inspect; consequently, it is advisable to obtain the assistance of an active, experienced, and intelligent man, and to instruct him as to how it is desired he shall seize and hold the head.

Most cattle-men, when asked to assist in mouthing, make a dash at the nose and seize the nostrils between the finger and thumb of one hand, while with the other they take hold of the horn—except in hornless animals—on the same side; this method of procedure, though necessary in obstreperous animals, is uncalled for, and often—owing to the infliction of wounds in the membrane of the nose with the nails—cruel; it frequently causes an animal to struggle violently.

The same ends can be accomplished and a better grip can be obtained by simply taking a quiet hold of the tip of the horn with one hand and seizing the lower jaw with the other, when the nose, in the great majority of cases, is easily turned upwards and the mouth opened without any difficulty ; if any struggling takes place the hold of the horn should be released and the upper jaw seized with the liberated hand, the back being planted firmly against the shoulder of the beast. There are few animals which cannot be effectually held by an ordinary man in this way, and, if necessary, many can be put on their broadside by a dexterous twist of the head and a sharp push at the shoulder. Very frequently, it will be found, that if the animal is approached quietly, the hand placed without violence on the horn and the lower jaw seized, the nose will be voluntarily turned towards the manipulator and the mouth opened widely ; whereas, if attempts are made to seize the nose, especially if the same process has been gone through several times, resentment is often shown and the jaws firmly closed. If anything unusual has been observed, the next care of the inspector is to make a careful examination of the pulse and respiration, and to take the temperature ; if the latter does not exceed 102° F. in cattle, there is not, as a rule, anything of importance wrong, but if it reaches 103° it must be looked upon as suspicious.

The condition of the skin (wool in sheep) will have been observed, and if any interference with the fleece—such as loss of wool, discoloration, or separation—exists, its cause should be at once sought.

The only disease of a contagious nature which will be detected by the state of the wool, is *scab ;* and if it exists, quantities, more or less large, of inspissated pus, altered sebaceous secretion, and cast-off epidermis will be found at the roots of the wool, with eruptions of various kinds, sores, and moisture. If any appearances of scab are presented and they cannot be satisfactorily accounted for by simple agencies—such as attacks of flies or worrying by dogs—the roots of the wool should be examined by the aid of a lens or microscope with the object of detecting acari.

The skin, too, should be examined for eruptions or discolorations.

Of the former, we have the eruptions of Ec. ep., Variola Vaccina, and Cattle Plague in cattle ; Small-pox in sheep.

The characteristics of the eruptions of the three diseases in cattle above mentioned have been already considered.

In sheep-pox, the invasive stage is marked by diffuse rose-coloured patches on various parts of the body, particularly the inside of the thighs and arms, and the under surface of the abdomen.

These roseolus patches must not be confounded with the red (*rubeolus*) discoloration of the skin of the scrotum, thighs, and abdomen of the ram in the rutting season ; in the course of a few days *papulæ—i.e.*, small, red, round, hard bodies, which blanch on pressure—are developed on the roseola, the latter gradually disappearing with the advent of the former.

In other few days the papulæ are, in their turn, replaced by *vesicles* which form on the site of the papulæ, are tolerably large, somewhat flattened on the surface, and contain, at first, clear limpid fluid which subsequently becomes opaque from admixture with pus. Vesicles gradually merge into *pustules—i.e.*, collections of pus corresponding in size to the vesicles, and accompanied or succeeded by *ulceration*, which again is followed by the throwing off of the scab (*desiccation*) which has been formed by the inspissation of pus, epidermis, and *débris*. Constitutional symptoms—as great fever, prostration, broncho-nasal catarrh, muco-purulent discharge from the eyes, ulceration of the cornea, diarrhœa, and loosening of the wool in its follicles—will also be present.

Discoloration of the skin of the pig is frequently caused by bruising. The specific discolorations and eruptions of hog-cholera consist in the formation of patches (circumscribed or diffused), of congestion, sometimes extravasation, producing a modena red, purple, black, or yellowish-brown colour according to the stage of the disease; and, when the malady is severe and far advanced, the formation of an imperfect vesico-pustular eruption, desquamation of the cuticle, and in extreme cases sloughing of the skin.

The discoloration is seen on the underneath surface of the body, frequently in the skin of the ears; and, finally, on the body itself. This disease is also accompanied by marked constitutional symptoms, as great fever, shivering, isolation, prostration, anorexia, constipation followed by diarrhœa, the discharges becoming fœtid and black in colour—sometimes being tinged with blood, and subsequently contracting a dirty yellowish-brown hue.

In the inspection of animals at grass and in byres the same general rules as those heretofore detailed must be followed. In grazing cattle and sheep, isolation, coughing, stiffness, dulness, and arching of the back, with, in the case of the former, an unthrifty appearance of the coat or depression of the head, should at once attract attention.

INSPECTION OF MEAT.

In considering this part of my work I shall direct attention, only, to the most salient and most important points in connection with it; and, in doing so, I shall deal with the subject in such a manner as to lead up to several vital questions—which it will be as well to enumerate at the outset. They are—

1stly, What meat is marketable, what unmarketable?

2ndly, What is innocuous, what nocuous?

3rdly, Are the rules usually followed in the inspection of meat sufficient for the purposes intended to be gained; and, if not, how can they be improved?

4thly, Is the inspection of meat, as a rule, in proper hands?

In order that these propositions may be intelligently considered we must first notice the characteristics of healthy and unhealthy meat, and the effects of various influences, physical or otherwise, upon it. But, before proceeding further, it is necessary —seeing that in considering the subject of meat inspection we usually take the flesh of the ox as a standard—to glance at the distinctive characters of horse flesh.

The flesh (muscle) of the horse is much darker in colour and coarser in texture than that of the ox; in flavour, it is, on the whole, superior, but its odour is by no means so pleasant.

The fat is always yellow in colour, soft, and possessed of a rather unpleasant, sickly flavour.

Cases have been known in which meat contractors have attempted to substitute horse for ox beef, and while no direct harm could be inflicted by such substitution—providing the animal, from whose carcase the beef had been obtained, was in good health—seeing that in point of nutritive value, and often in flavour and texture, the flesh of the horse is superior to that of the ox, nevertheless, in a monetary point of view and in a spirit of fair trading, it is necessary that some simple test should be forthcoming to enable those engaged in the work of inspection to detect the difference between the two. The test, or positive proof, is to be obtained in the bones. The bones of the horse are, relatively, much larger than those of the ox; contain more semi-fluid fatty matter; their processes (both epiphyses and apophyses) are better developed and more numerous, and the floating bones differ materially in size in the

two animals; thus, the ulna or elbow-bone of the ox is much longer than that of the horse, it articulates in fact with one of the bones of the knee; *vice versa* the corresponding bone of the hind leg—the fibula—as also the splint bones of both fore and hind legs, are usually entirely absent in the ox, and if present are only rudimentary. The ribs of the ox are fewer in number than those of the horse (13 as against 18), but much broader and flatter.

The bone of the tongue in the horse is composed of five segments or parts, while in the ox there are nine, and the body is angular in shape, the spur process being very short and conical. The tongue, too, in the ox is pointed at its extremity, and its surface much rougher than that of the horse; lastly, in the heart of the ox a distinct bone (*os cordis*), which is found with extreme rarity in that of the horse, exists.

In a medico-legal, as well as in a trading, point of view, it is also necessary to distinguish between the carcase of a male (whether castrated or entire) and a female animal. In the bull the textures of the sternum (brisket) are much coarser, harder, and darker in colour than in the ox or the cow; the contour of the arm muscles is more perfect, and they are better developed; but in the ox they become comparatively fine, if castration has been performed for any length of time. In the bull and ox the penis is usually left attached to the left side of the flank, its greater size in the former animal forming a marked contrast with its smaller size in the latter. *

In the bull and ox there is always a mass of fat in either flank; its shape, in oxen, being regulated by the method of emasculation which has been adopted. If the end of the scrotum has been cut off, the mass of fat will be much more elongated and conical than when a lateral incision has been made into either scrotal sac. Those breeders who desire to see a plump purse always remove the end of the scrotum in castration.

The masses of fat in the flanks of the cow, when the udder has been removed, are always irregular and much smaller than in the flanks of the bull or ox.

These remarks apply equally to the two sexes in the sheep.

In the ox we expect to find that the muscular tissue is of a florid hue; in certain positions free from intermixture with fat, in others its connective tissue so infiltrated with that substance as to give a distinctly mottled or marbled appearance on section; it is of a firm or slightly elastic consistence, is tolerably dry after being exposed for a short time to the atmosphere, and *rigor mortis* (death-stiffening) is marked; it has a pleasant, sweet flavour, and exhales, when exposed to the action of heat, a savoury odour. *The fat* varies in colour, but is usually firm, has a suety taste and a greasy feel, is combustible, and does not readily putrify. *The connective tissue* is glistening and moist; and what blood may be left in the meat, when roasted, forms rich brown flakes. On section through a piece of good meat, when properly cooked, a rich

* It is only right, however, to mention, that some butchers remove the penis of the bull, on account of its large size, leaving only the (pale) retractor muscle.

sanguinolent fluid (which gelatinises when left at rest) flows out; lastly, healthy meat is easily preserved, and resists putrefaction for a tolerable length of time.

In examining meat for the purpose of deciding upon its nocuity or otherwise we have to note, firstly—

THE COLOUR.—In this, as in the consideration of all the other characteristics, we must determine whether the alterations we may detect are *general* or *local*—*i.e.*, whether due to a cause which equally affects the whole body, and thus renders the flesh all bad alike; or which confines its influence to certain well-marked boundaries, and only destroys the part in which it is actually located. The colour of animal flesh varies from the pale hue of the flesh of the unborn calf, the lamb, and other young animals, to the dark red of the horse, the old boar, working ox, or ram.

The flesh of many foreign animals is very pallid and the fat white; the latter characteristic is also sometimes seen in home-fed animals, and is often due to the character of the food; but pallor of muscle is seen in all effusive diseases, in Hydræmia, in so-called Grass Staggers (indigestion of cattle), sometimes in exhaustion, and very notably in death from choking—*i.e.*, impacting of a foreign body in the œsophagus, in which case the muscles have also a slow-boiled or macerated appearance.

A yellow colour or tinge in the fat and tissues is in many animals natural, and is not infrequently due to the food containing a large proportion of marzarine and oleine (predominance of stearine gives a white appearance); in other instances it can be traced to certain breeds, and the whole of the progeny of one particular sire may present the peculiarity: otherwise, systemic icterus—even of a moderate degree—is the result of functional or organic disease of the liver, while circumscribed yellowness is due to contact, for several hours, with the gall bladder, or to the effusion of gall from rupture of that organ.

In judging of the importance to be attached to yellowness, we should observe, if possible, whether the colour is confined to one, or is common to several animals of the same lot, and an authoritative opinion should never be given from an examination by artificial light.

A magenta hue is characteristic of general albuminous effusion as the result of the action of a zymotic poison or ferment on the blood, and indicates the existence, in the acute stage, of some zymotic or specific affection, as Zy. p. p., Ec. ep., Rinderpest, or Tuberculosis. It is most observable in making a fresh section and exposing it for a short time to the action of the atmosphere, and is accompanied by the glaze of albuminous effusion.

A scarlet hue is but seldom seen, and is indicative of carbonic oxide and, sometimes, arsenical poisoning.

A dark-modena red results from imperfect aeration of the blood, and is seen in carbonic-acid poisoning, in disease of the lungs where the area involved is so large as to prevent the access of air; or where mechanical obstruction exists in the air-passages. It is also seen in poisoning by narcotic agents, and particularly by those which, in

addition to their action upon the brain, arrest the process of oxidation in the tissues—as alcohols, chloroform, æthers, turpentine. Modifications of this colour are also seen in inflammatory affections, in high fevers, in apoplexy, in drowning, in suffocation, and in animals which have been imperfectly bled; the latter condition is easily distinguished by the marked infiltration of blood into the most dependent parts, by the back of the carcase being streaked with streams of blood, which has escaped from the cut ends of veins, and by the veins and capillaries, especially of the internal organs, being surcharged with blood. *Circumscribed darkness* in colour may be due to hypostatic congestion from the animal having lain on its side or back for some time after slaughter; it may be also the result of *post-mortem* staining of hyperæmia, inflammation, or extravasation of blood. *Diffused redness* (dyeing) is seen where meat has been subjected to the action of extreme cold and moisture, as in freezing; in blood-poisoning and in decomposition, and is caused by solution and diffusion of the hæmoglobin.

A mahogany hue is a blending of red and yellow, and is due to imperfect decarbonisation of the blood, combined with *icterus* from absorption of bile pigment; it is characteristic of extensive disease of the lungs and liver, and is most frequently seen in advanced P. p. zy. and phthisis. The flesh in this case is usually dry, though the flanks may be moist.

Iridescence on section is seen under a variety of circumstances, as in blood diseases, prolonged fevers, inflammatory affections, and difficult parturition (Fig. 8, Plate X.)

A green hue is indicative of decomposition or gangrene, and is seen in the abdominal parietes when the viscera have been allowed to remain in the abdomen for some time after death; in this situation, it may also be due to diffusion of medicinal agents through the coats of the stomach and intestines.

Black pigmentation is usually local, and is the result either of previous inflammation, extravasation of blood, or melanosis.

In judging of colour, age, sex, class of animal, mode of feeding and of treatment prior to slaughter, the kind of weather, and the quantity of water used in dressing the carcase, must be taken into consideration. Many medicinal agents—as Epsom salts, treacle, æthers, turpentine, camphor, &c.—produce a material difference in colour.

Odour—Varies in healthy meat from the vile stink of the old boar or goat to the scarcely perceptible odour of the young calf or lamb—though that of the former is modified by castration a few months prior to slaughter, and by partial starvation for a few days.

Abnormal odours are due—1stly, To animals having been fed on vegetables possessing volatile odorous principles, as turnips, alliaceous plants, and odoriferous plants in general.

The odour of turnips is most striking when an animal has been choked by a piece of the root.

2ndly, To animals having been dosed, prior to slaughter, with volatile medicinal

agents, as prussic acid, turpentine, tar, æthers, camphor, chloroform, methyl, alcohol, sulphur, phosphorous or essential oils.

3rdly, To putrefaction or sphacelus, or to contact with decomposing fluids, as pus, urine, &c.

4thly, To exposure, immediately after slaughter, to an atmosphere charged with the odour of tobacco, tar, carbolic acid, or paint; in this case the odour is superficial.

5thly, To uræmia, infiltration of urine from the bladder, or medicine from the intestines or stomach; in the first case, the odour being general; in the two last, localised.

6thly, To prepared meats undergoing acid fermentation, having been mixed with aromatic substances, or cured with pyroligneous acid or wood smoke.

Odours, as a rule, are most readily distinguished when flesh is recently slaughtered, newly incised and breathed upon or artificially heated—with or without moisture; slight odours often disappear as the carcase dries.

MOISTURE.—The amount of natural moisture is altered by the condition of the atmosphere—*i.e.*, whether murky and damp, or dry and desiccative; by thorough or careless abstraction of blood, by the condition of the animal prior to slaughter—*i.e.*, as to whether it has been subjected to exertion or not; and by the quantity of water used in the dressing. The moisture of young, especially the unborn, exceeds that of old, and of good-conditioned that of lean animals; while the neck and flanks always contain the greatest amount of moisture; the former from the gravitation of serum from the blood and of water from the carcase.

Abnormal moisture is due—1stly, To *albuminous effusion*, which is general, and results from the action of enzymes or saline matter in the blood; it is temporary, and in some diseases gives place to serous effusion; it varnishes the skin of the hand and produces an *albuminous glaze* on exposed sections of muscle.

2ndly, *To effusions of lymph*, which may be either localised or diffused, and resulting from blood depravation or deterioration, owing to inflammatory action, to specific disease—as rheumatism, synovitis, &c.—and to the irritation of blisters or of skin diseases; it ultimately causes fibroid substitution of the muscle, rendering it pale in colour, less vascular, and very hard.

3rdly, *To effusions of serum*—sanguineous or simple—and resulting from blood depravation or deterioration from the action of ferments, salines, or other medicines, debilitating diseases, hydræmia, disease of important organs, cachexia, thawing after freezing, &c. Effusion of serum may be general or local, the latter frequently being an accompaniment of inflammation, erysipelas, disease of the skin, as scab; and disease of the vessels; while either local or general effusion may be the result of the irritation produced by parasites and by the lodgment of the bladder-worm (hydatids) in the intermuscular connective tissues.

4thly, *To effusion of blood*, which may also be local or general; the former being produced, as a rule, by violence prior to death; by the ravages of migratory parasites,

as the strongyles; by the violent throes of parturition; or by local disease of the coats of one or more blood-vessels; general effusion being indicative of blood depravation or deterioration with or without disease of the vessels. Local extravasations (except those into the chest from the knife having penetrated too deeply, or those produced by parasites) are usually superficial and are unaccompanied by any detectable alteration in the blood; while general extravasations are always accompanied by physical or chemical blood changes, are more deeply seated—even in the interior of the bones under the periosteum, in the structure of organs, and in the cranial and spinal cavities—and when subjected to the action of fire evolve an unpleasant odour and do not form the brown savoury flakes seen in roasted healthy blood.

The blood, in either case, may be of much the same colour and may undergo the same changes on exposure to the atmosphere, although in anthrax it stains everything with which it comes in contact and is frequently of a crimson or purple hue.

5thly, *To effusion of urine,* which must necessarily be localised, and is due to obstruction at the end of the sheath of the male, or in the urethral canal, or to wounds in the urethra. It is easily detected by the urinous odour which it imparts to the infiltrated tissues.

Healthy Firmness is dependent, to a great extent, upon the same influences as those which regulate moisture. *Abnormal Flaccidity* is the result of debility, effusions, exhaustion, electricity, nervous influences, fevers, inflammatory affections, vegetable poisoning, decomposition, and blood alterations. Flaccid flesh has frequently an adhesive, sometimes soapy, feel and pits on pressure (doughy); it may be of any colour or odour, fat or lean.

Texture.—*Abnormal texture* varies from the catgut flesh of the patriarchal and hard-worked ox (sodger's beef) to the tender and easily-masticated muscle of the unborn calf. Muscle is frequently very friable, especially where much struggling has taken place prior to death, where pain has been severe, and in cases of choking. The flesh of the sirloin is always of finer texture than that of any other part of the body.

Flavour.—*Abnormal flavour* is regulated by the same influences and results from the same set of causes as do abnormal odours.

Emphysema—*i.e.*, the presence of air or gas in the cellular tissue—may be general or local—the former resulting from blood putrefaction, infiltration of gas from the alimentary canal, or rupture or injury to the lungs, or from blowing by the butcher: the latter from wounds or sphacela. The gas in emphysema—from sphacelus and blood putrefaction, or from the intestines—has an unpleasant odour and is accompanied by blood and serous effusions.

Having considered the characteristics of healthy and unhealthy meat, we will now set ourselves to examine the questions which I enunciated at the outset.

Firstly, As to marketable and unmarketable meat.

About this question, as about all others connected with this subject, a vast difference

in opinion exists; and it cannot be answered without including in its discussion the third question also. In the very great majority of instances inspectors of meat look only at the bare carcase, and that, too, after it has been dressed and hung up for a period of twenty-four or forty-eight hours; and, not infrequently, even after it has been quartered. If the flesh is tolerably firm, dry, devoid of unpleasant odour or flavour; is not much altered in its normal colour and the carcase sets, it is passed as marketable and fit for food. If the reverse of these conditions obtains, it is rejected, though in some districts food is condemned which would be passed in others. Thus, one inspector would reject a carcase which was dark in colour, even though otherwise all right, while another would pass it without hesitation. We shall see the same divergences in opinion when we consider the second question; and I think that much might be gained if the Legislature—by the aid of competent authorities—would authoritatively settle the question as to what should be considered fit for food and what unfit. It should decide, too—independently of particular diseased conditions—primarily, between two principles for the guidance of inspectors. These are—(*a*) Is the inspector to take into consideration collateral circumstances? (*b*) Is he simply to be guided by the condition and appearance of the carcase after it has been divested of all organs and appendages, and prepared in the usual way for sale?

If he has to follow the first principle he will have to guide him—the length of time the animal has been ill, the treatment to which it has been subjected, its sex, age, and condition; the presence or absence of morbid processes in the tissues or organs; and the microscopical characters of the tissues, and the fluids (blood, milk, and serum) of the body.

If he follows the second principle he can only call to his aid the special senses of touch, taste, sight, and smell—all of which may be deceptive.

Secondly, What is innocuous, what nocuous? In attempting to answer this question a very wide latitude must also be given for difference in opinion and, I must confess it, for the state of ignorance which exists as to the effects of different kinds of diseased flesh upon the human frame. We must not forget, too, that what is innocuous directly or immediately is in many instances injurious indirectly. Thus, while we pass flesh which shows no trace of disease, but is simply lean and deficient in juices, we must remember that, though it may do no direct harm to the consumer, it does an immense amount of indirect harm, as it satisfies the appetite for a time, but does not nourish the body, consequently the consumer had better purchase oatmeal and bread than throw his money away upon that which does not nourish his frame.

I suppose it will be admitted pretty well on all hands that when an animal has been slaughtered when suffering from any of the undermentioned affections, its flesh is absolutely unfit for food, as being innutritious, or it is nocuous.

The affections to which I allude are—mortification of organs or parts, pyæmia, septicæmia, retention of decomposing placenta or of a fœtus in utero, hog cholera,

anthrax (splenic fever particularly), small-pox, trichinosis, glanders, farcy, hydatid disease of the muscles, dropsy, advanced rabies, well-marked cachectic conditions, whether these be cancerous, tuberculous, or otherwise; and, also, when the flesh is undergoing putrefaction.

It is with regard to the flesh of animals suffering from Zy. p. p. and Tubercle that some authoritative settlement is urgently needed, and also *parturient flesh*, which (for purposes of deception) is sent into the market with all the discoloured or infiltrated tissues around the pelvic outlet carefully removed, and with the udder either nearly or entirely dissected out; or, in the case of the sow, with the teats pulled so as to make them resemble the teats of non-parturient animals. In the case of the cow, the flaccid condition of the sacro-ischiatic ligament is always a good guide. We have no positive proof that parturient flesh is nocuous, but its use as food is, at least, revolting and repulsive to the mind, and so much is this the case that in some districts even low-class butchers refuse to purchase it.

Extravasation of blood in flesh, if due to injury, does not necessarily render it unfit for food, as the injured parts can always be removed with the knife, but if they are so located and distributed as to afford positive proof of blood depravation they should enable us unhesitatingly to condemn such flesh, even though microscopical examination of the blood does not reveal bacteria or any material alteration in the red corpuscles.

In constitutional disease of the bones—as in softening or rickets—doubts as to nocuity may reasonably be entertained, providing the flesh is healthy in its character, although in those cases where it may be fairly assumed that the altered conditions have originated in blood depravation condemnation of the flesh should be unhesitating.

The flesh of animals which have met with sudden death and which, though otherwise healthy, have not been bled, may be consumed with as little fear as the carcases of those beasts of the field which are so frequently shot, but the loss in quantity of whose blood may be reckoned by ounces; and I think the same may be said of animals which have been suffering from slight ailments, but to which medicines (other than strychnine and allied substances) have been administered. It would be difficult, too, to prove, that injurious effects follow the ingestion of the flesh of the unborn, or but recently born, calf or lamb—however much the mind may revolt from such a procedure.

The last question I have to discuss—*i.e.*, Is the inspection of meat, as a rule, in proper hands?—can be answered in a few words. In the great majority of instances inspectors of meat are chosen from butchers, farmers, or those employed officially, in some other capacity, about slaughter-houses, any doubt that may exist as to the sanitary or insanitary condition of meat being usually referred to the medical officer for solution. Now, while I am free to confess that many inspectors (who know nothing of disease) attain, in the course of time, to a vast amount of practical knowledge and

sound tact, there are very many who are absolutely ignorant of the rules necessary to guide them in their work, or of the evil effects of unhealthy flesh. Medical men, too, some of whom, be it said to their honour, do not dabble in matters which they do not comprehend, are ill qualified to judge of the fitness or unfitness of animal flesh for human consumption, as they are, in the very great majority of instances, totally unacquainted with the characters of animal diseases and the effects of any particular malady upon the tissues of the body. Veterinary surgeons, as they are trained, or should be trained, at the present day, are, in my opinion, the proper consultees as to the condition of flesh from animals which have suffered from any pronounced form of disease.

(*POSTSCRIPT.*)

ZYMOTIC PLEURO-PNEUMONIA IN AMERICAN CATTLE.

As the above question has assumed a prominent public position since the proof-sheets which treat of Zy. p. p. in this work were printed off, the Author feels it a duty to make a few remarks upon the subject in the form of a Postscript, more especially as his name has been mentioned in connection therewith. Without entering into details as to the general bearings of the question, the Author would briefly observe that he had the opportunity, in company with Professor M'Call, of seeing the lungs of a number of the cattle which formed the cargo of the s.s. *Ontario*—recently landed at Liverpool—and that, although no decided symptoms of Zy. p. p. were evinced by any of the living animals, unmistakable evidence of it existed in the lungs of two oxen which were taken indiscriminately from the lot he was examining and slaughtered. The multiplicity of diseased centres, the distinct nodular elevations of the pleura caused by these centres, their circumscribed character, the distinctly mottled appearance on section, with the characteristic peri-bronchial and interlobular hyperplasy; the bronchial and vascular plugging, the lobular consolidation, the surrounding lobular and interlobular effusion, were well marked; while there was a total absence of the catarrhal products and softening, and of the intense vascularity of the bronchial membrane, seen in broncho-pneumonia. The pulmonary pleura over the diseased centres was adherent to the costal pleura, and the contiguous muscular tissue was infiltrated with serum and lymph. Granted that the necroscopical appearances were not exactly identical with those we are accustomed to see in dairy cows, or in fact in any class of cattle where the disease runs a rapid course, the departures from the ordinary characteristics were not so great as to leave even the slightest doubt in the mind of the Author as to the true nature of the disease, and these departures were no more than are produced by local circumstances, as breed, sex, and age of animals; mode of feeding, condition, strength,

of constitution, intensity of the disease, rapidity of its progress, and the primary localisation of its origin—*i.e.*, whether in the bronchial membrane, the pulmonary structure, or the pleura.

Since examining the above-mentioned lungs, the Author has seen several which presented appearances and conditions very closely allied to them—the differences being rather in individual than in general characters, such as may be seen in the localisation of the lesions in all zymotic, or even sporadic, affections.

Of the wisdom of the step taken by the Privy Council Authorities in ordering the slaughter of American cattle at the port of debarkation—even though the matter were one of doubt, simply—there can be no question; but that it is at all one of doubt is negatived by the fact well known—not only to the American but to the Canadian authorities—that Zy. p. p. exists extensively in the United States, and that an open trade in neat cattle has been for a long time kept up between America and other countries (even Great Britain) in which the disease has for years had a permanent home. The extract, hereto adhibited, taken from the Report of the Canadian Minister of Agriculture—kindly furnished to the Author by Professor M'Eachran, of Montreal—will speak for itself.

Whether the Privy Council Authorities may ultimately see fit to admit cattle from healthy States (providing an uncontaminated through-route is ensured) or not, their recent action in the matter will, I think, be homologated by most people.

REPORT OF SPECIAL INVESTIGATION INTO EXISTENCE OF CATTLE DISEASE IN THE UNITED STATES.

MONTREAL, 31*st January*, 1879.

Hon. J. H. POPE,
Minister of Agriculture, Ottawa.

SIR,—In compliance with your instructions I proceeded to Washington, D.C., where I arrived on Monday, 20th inst., and immediately proceeded to make inquiries concerning the reports of contagious diseases in cattle.

Having heard that the Sanitary Board of the District had made some investigations in the matter, I visited the medical health officer, Dr. Townshend, who informed me that he had caused some investigations to be made, which went to show that the disease Pleuro-pneumonia had been prevalent near Washington, D.C., during the past summer, but at the present time he was not aware of its existence. He mentioned the "Cabble Farm" as one of the places where it had been.

On visiting Capt. Cabble, he confirmed Dr. Townshend's statement, and introduced me to Mr. Wall, his tenant, who is engaged in the milk business. This gentleman very clearly described the disease as a contagious lung fever, brought to his farm by a cow bought in Washington market. He lost a number during the past summer. He con-

sidered it highly contagious. In his opinion over 200 cows died in the neighbourhood of Washington during the past summer. He could not show me any cases.

I next visited J. T. Bushman, M.R.C.V.S., who had not seen much of the disease personally, but had no doubt he could direct me to where it was existing.

We first proceeded to West Washington and visited the dairy kept by Mr. Kelly, who had five which died in September and October.

Mr. Davis, West Washington, had also five cases. They contracted the disease from running in the common with Kelly's cows. No cases were found there.

Our next visit was to Hyattsville, Prince George County, Maryland. B. F. Guy, Esq., lost ten out of eighteen; the others were sold, as well as the stables and part of the land, as the shortest way of getting rid of it. Mr. Guy gave us a very intelligent account of the disease. It was introduced by a cow from Washington cattle-market; he had no doubt of its contagious character; he found the symptoms and *post-mortem* appearances exactly as described by Professor Gamgee in his report of 1871. His experience was that it was no use treating it. We could not find any cases there during our visit.

Our next visit was to Alexandria in Virginia. Here we found that during the past summer and early winter the disease was very prevalent, but it was very difficult to get any one to allow us to see the animals living. However, after a time we succeeded in convincing them that we were not "health officers," and we found cases in abundance. We called upon the following milkmen:—Mr. Carral, Mr. Flood, Mr. West, and Mr. Darling, who had all lost severely by the disease. Miles Murphy, a grocer, had lost two only a few days before. Mr. John Bayless, grocer, had lost severely, and had two well-marked cases. Mr. Bailey, Barley's Cross Roads, had also a number affected. Mr. Hughes, Alexandria House, had lost one a few days before our visit. Mr. Graves, grocer, had lost two. Mr. Hunter had lost eight out of nine. Hughes informed us that one Miller, two miles out of town, was ruined by the loss of eighty cows.

The disease was generally believed to have been imported there from Baltimore.

When asked if many had died lately, the answer invariably was, "Yes." Hardly a day during summer, and even very lately, but they were carted away. Several hundreds at least had died within three months.

After a good deal of trouble we succeeded in obtaining a cow in an advanced stage, which, for a consideration, the owner was willing to let us kill.

The cattle in this place were owned by a coloured family named Franklyn. There were three cows ill in different stages of the disease. The subject of the *post-mortem* was a six year old medium-sized cow. Before killing her the pulse was 100, respirations 40, temperature 105°. She was observed to be ill for ten days (no doubt she had been longer). She stood with head protruded, nostrils dilated, breathing quick, short and jerking, elbows turned out, each expiration accompanied by a loud mournful grunt. The secretion of milk was completely arrested. The bowels were relaxed, fæces black and offensive. The pupils dilated, and she moved with difficulty, being dull and stupid. On being killed by concussion of the brain a careful examination was made, more especially of the cavity of the chest. The sternum being sawn through, the heads of

the ribs broken from the vertebræ, the side of the thorax was removed, thus exposing the organs *in situ*.

A small quantity of discoloured serum was found in the lower part of the cavity. The right lung presented about the middle of its costal surface a spot of lung in process of hepatisation; its pleural surface being adherent at this point only, the remaining part of this lung was comparatively healthy. The left lung was completely hepatised, and adherent throughout its entire pleural surfaces. The adhesions to the costal pleura, more especially, were thick and strong. The pulmonary pleura was very thick and spongy, and easily removed from the parenchyma. The weight of the lung was enormously increased, and it lifted out like a solid mass. When cut into there was no crepitation, the substance presented the characteristic marbled appearance (so correctly given in the illustration of Professor John Gamgee's work), caused by the deep red colour of the hepatised pulmonary lobules surrounded by the bands of interlobular-cellular tissue thickened and infiltrated by an extensive exudation of congealed lymph. The whole lung was swollen solid and œdematous.

The bronchial tubes contained a quantity of serum, frothy, and of a pink colour. The air-cells were almost entirely obliterated.

Owing to darkness overtaking us the examination was confined to the chest, which was sufficient, with all the collateral circumstances, to convince us both beyond doubt that the disease was the "Contagious Pleuro-pneumonia."

Before leaving Washington I had the honour of an interview with Honourable The Commissioner of Agriculture, Hon. Wm. G. LeDuc, during which I explained my object in visiting the district, being to ascertain the truth of certain reports which had appeared in the newspapers of contagious cattle disease, said to be Rinderpest. That the gentleman at the head of the Department is fully alive to the existence of Pleuro-pneumonia, its dangerous nature, and the enormous responsibility of the Government in relation to the matter, is amply illustrated by the following extract from his Annual Report to the President for November last:—

"One of the most dreaded contagious diseases known amongst cattle is that of Pleuro-pneumonia, or Lung Fever. It was brought to this country as early as the year 1843, and has since prevailed to a greater or less extent in several of the Eastern and a few of the Southern States. It made its appearance about a century ago in Central Europe, and has since spread to most European countries. With the exception of Rinderpest, it is the most dreaded and destructive disease known among cattle. Unlike Texas Cattle Fever, which is controlled in our more northern latitudes by the appearance of frost, this disease knows no limitation by winter or summer, cold or heat, rain or drought, high or low latitude.

"It is the most insidious of all plagues, for the poison may be retained in the system for a period of one or two months, or even for a longer period, in a latent form, and the infected animal in the meantime may be transported from one end of the continent to the other in apparent good health, yet all the while carrying and scattering the seeds of this dreadful pestilence.

"Since the appearance of this affection on our shores, it has prevailed at different times in the States of Massachusetts, Connecticut, New York, New Jersey, Pennsylvania, Maryland, Delaware, Virginia, and in the District of Columbia.

"It has recently shown itself at two points in Virginia (Alexandria and Lynchburg), where it was recently prevailing in a virulent form.

"At present the disease seems to be circumscribed by narrow limits, and could be extirpated with but little cost in comparison with the sum that would be required should the plague be communicated to the countless herds west of the Alleghany Mountains. This disease is of such a destructive nature as to have called forth for its immediate extirpation the assistance of every European Government in which it has appeared, many of them having found it necessary to expend millions of dollars in its suppression.

"The interests involved in this case are of so vast a character, and of such overshadowing importance both to the farming and commercial interests of the country, as to require the active intervention of the Federal Government for their protection; and for this reason the considerate attention of Congress is respectfully asked to this important matter."

As additional testimony, I beg to enclose the letter of Joseph Bushman, M.R.C.V.S., of the Quartermaster's Department, U.S. Army :—

"Sir,—In compliance with your wish for a statement of my knowledge of the existence of contagious disease amongst cattle in this vicinity, I would say, I have lived here since January, 1874; do not remember seeing or hearing of any cases in that year. During 1875, 1876, 1877, I saw occasional cases of Pleuro-pneumonia in some of the small dairies on the outskirts of this city. Was not called on professionally by owners, but was afforded opportunities to see several cases by Robert Smith, a veterinary practitioner in the eastern part of this city. Saw several well-marked cases, and the lungs of two which were slaughtered. Heard of many other cases. In July, 1878, there was a considerable outbreak of this disease, both in the District and adjoining parts of Maryland and Virginia. The disease prevailed during July, August, and September. Probably 75 to 100 died in the District of Columbia, although the disease was of a subacute type and many recovered.

"During these months I had frequent opportunities of seeing sick animals in all stages of the disease, and their lungs after death, and being familiar with the disease in England and Ireland, *have no doubt* as to its being 'Contagious Pleuro-pneumonia.' I was not called to treat any. Suppose that my being connected with the United States Government was the reason, as the owners, milkmen, wished to conceal the fact of having sick animals. I investigated for my own information, as I had called on the former Board of Health and present Health Officer and offered my advice and assistance, which were never called for. I did not publish the matter. In conclusion, I would say that no Rinderpest has existed; the newspaper reports to that effect had no foundation, except in the wrong use of that name for Pleuro-pneumonia by parties ignorant of the differences who were called in to treat the animals.—I am, dear Sir, yours very truly,

"Jos. Bushman, M.R.C.V.S.,

"*Veterinary Surgeon, 2 M. Department, U.S.A.*"

My next visit was to Philadelphia, where I received most valuable assistance from Mr. J. W. Gadsden, M.R.C.V.S. While rumours were plentiful, we failed to find any direct evidence of the existence of contagious disease of any kind among the cattle around or in Philadelphia.

At my request Mr. Gadsden wrote letters to a number of veterinary surgeons and others in the State of Pennslyvania, and received replies that there was none.

Accompanied by Mr. Gadsden I visited New York, and communicated with the Principal and Professor of the American Veterinary College, none of whom had any experience of the disease, and doubted the correctness of the rumours of its existence. At my request they telegraphed to Mr. M'Lean, of Brooklyn, who replied that he could not immediately show us any cases, but buried animals two or three days before. Being determined to see for myself, accompanied by Messrs. J. W. Gadsden, M.R.C.V.S., A. Lockhart, R.M.C.S., and Dr. Liautard, of the American Veterinary College, we visited Brooklyn, and, after a little inquiry, were directed to the cattle-sheds near to Gaff & Fleeschman's Distillery, Williamsburg, between King and Queen's Counties, Long Island; here we found between 800 and 900 dairy cows, owned by different parties, who pay 77½ cents per week for use of the shed and supply of swill from the distillery. Of all the pest-houses possible to imagine this one is the worst. In low-roofed sheds cattle are packed as close as they can stand in double rows, with a passage of about three feet between the rows. Swill nearly boiling hot is run into troughs in front of them, into which hay is placed and remains till it is cool enough for them to eat and drink. They have no kind of bedding. The swill, acting on the bowels and kidneys, increases the excretions, and what with the steam, effluvia from the excrements, want of ventilation and general sanitary defects of the place, it would be strange if disease was not prevalent.

In addition to the above disgraceful condition, the business carried on by the parties owning the cattle is that best calculated to ensure the continuance of the disease.

The disease Pleuro-pneumonia was introduced to Brooklyn in 1843, and again in 1850, by imported cattle, and it has prevailed in Long Island ever since. The disease is permanently established in the large distillery sheds and dairies in the neighbourhood, and few, if any, are expected to, or do, escape it. The period of incubation varies from nine days to three months, and strange as at first sight it may appear, the disease runs its course much slower when cattle are confined in a warm stable, where they have no room to move, and are not exposed to the variations of temperature which those in fields experience. Not only so, but for a time they milk freely and lay on fat rapidly; consequently, before the disease has approached the stage in which they die, they are handed over to the butcher, are killed and dressed, and sent to Washington Market, New York, where it is sold as prime beef.* They are immediately replaced by fresh cows, and thus it continues year after year. Many, however, die from the disease in the sheds. I can truly repeat the words and sentiments of Professor Gamgee (Report 1871): "No one can

* I was assured by a butcher, who dealt largely in this beef, that it commanded the highest price in the market.

hesitate in declaring that the cow-sheds of Brooklyn and other cities are a disgrace to a civilised people."

Owing to the dread of interference by sanitary police, it is very difficult to gain access to infected dairies; consequently, I had not an opportunity of prosecuting my search in Long Island as extensively as I could have wished. However, as remarked by Professor Liautard, who, up till now, was sceptical of its existence, out of the 800 or 900 animals which we saw we could not pick out 50 which could be called free from the disease. I am much indebted to Mr. L. M'Lean, graduate of the Edinburgh Veterinary College, for assistance in tracing out the disease in Long Island. Mr. M'Lean has had considerable practical experience as a Government Inspector in Scotland, and also in the United States. The following letter from him will bear me out in my statements concerning Brooklyn:—

"561 Carlton Avenue, Brooklyn,
"New York, 28*th January*, 1879.

"Dear Sir,—If you had any doubts of the existence of Contagious (or Epizootic) Pleuro-pneumonia being in this part of the country on your arrival here, the many evidences of it both in the living and dead subject brought under your observation, I was satisfied to observe, left no doubt in your mind as to its existence. For myself, I am sure there is no other place of the same extent as Long Island where so many cattle are to be found affected by Epizootic Pleuro-pneumonia, and I am astonished that the powers could be so apathetic as to allow such a virulent and contagious disease to get such a hold without using means for its extinction or suppression. If it is allowed to spread over this continent it will put an end to the exportation of live stock, and prove disastrous to cattle raisers in general. In my practice I find that owners of cattle are very anxious that some combined action should be taken to stamp out a disease that is so ruinous to them all.

"I trust your visit will incite the authorities at Washington to take the matter in hand.—Yours very truly,

"L. M'Lean, V.S."

In addition to the above evidences of the existence of Pleuro-pneumonia, I have brought with me, for the Museum of the Veterinary College at Montreal, a beautiful specimen of the diseased lung, which shows the characteristic marbled appearance, the solid, dark-red colour of the lobules and the yellowish-white infiltration of the interlobular, cellular tissue so faithfully represented by Gamgee.

In concluding this report, I have purposely omitted any lengthened description of the disease, merely confining myself to statements of facts with regard to its existence, and beg to call your attention to the statement made by Fleming (Contagious Diseases of Animals):—"It has been calculated that in Edinburgh alone the annual loss from 'Lung Plague,' is between £200,000 and £300,000.* For the six years ending with 1860, it has been estimated that there perished considerably more than a million of cattle in the

* This calculation is evidently exaggerated.—T. W.

United Kingdom, the value of which must have amounted to at least twelve millions of pounds. The tables of an English Cattle Insurance Company prove that from 1863 to 1866 the death-rate from this scourge was from fifty to sixty-three per cent. annually. If we can form any judgment from these figures, it will not be too much to assert that the 'Lung Plague' costs England, at the very least, £2,000,000 ($10,000,000) a-year."

Looking at it from the direct loss thus entailed by death, the necessity for active steps being immediately taken to prevent its importation to Canada from England or the United States, is very clear.

By referring to the (Contagious Diseases Animals) Act, 1878, Part IV., section 16, clause B, referring to animals from foreign ports, in which disease is discovered at the port of landing, it says :—"If the disease is Pleuro-pneumonia, the Inspector is to cause the diseased animals, and all cattle brought in the same vessel therewith, to be dealt with as follows :—(1.) The diseased cattle are to be slaughtered at the place of landing. (2.) The healthy cattle are to be slaughtered at the place of landing, or if landed at a port at which there is a foreign animal's wharf, the Inspector may permit them to be removed into that wharf for slaughter, but not elsewhere."

It will thus be seen that to our farmers and stockmen a double danger is imminent unless active protective measures are immediately adopted.

So far, I am happy to report, no contagious Pleuro-pneumonia is known to exist in Canada, but if the cattle from infected States are allowed to enter the Dominion, either for breeding, feeding, or shipping, we run a very great risk of importing the disease.

I believe, so far, no lung-disease exists in any of the Western States, consequently prohibition of cattle entering from them does not appear necessary, provided the United States Government do guarantee that no cattle from infected States will be allowed to be carried to the Western States, and that a thorough system of inspection and quarantine be maintained throughout the Union, under competent members of the veterinary profession.

In addition to which, I would recommend that cattle be admitted only at certain ports, and that at each a competent Inspector be stationed, whose duty would be to examine every animal carefully before being admitted into the country, even to pass through to a shipping port.—I have the honour to be, Sir, your obedient Servant,

D. M'EACHRAN, F.R.C.V.S.,
Montreal Veterinary College.

—*Extract Report of the Minister of Agriculture for the Dominion of Canada for* 1878.

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LORIMER AND GILLIES, PRINTERS, 31 ST. ANDREW SQUARE, EDINBURGH.

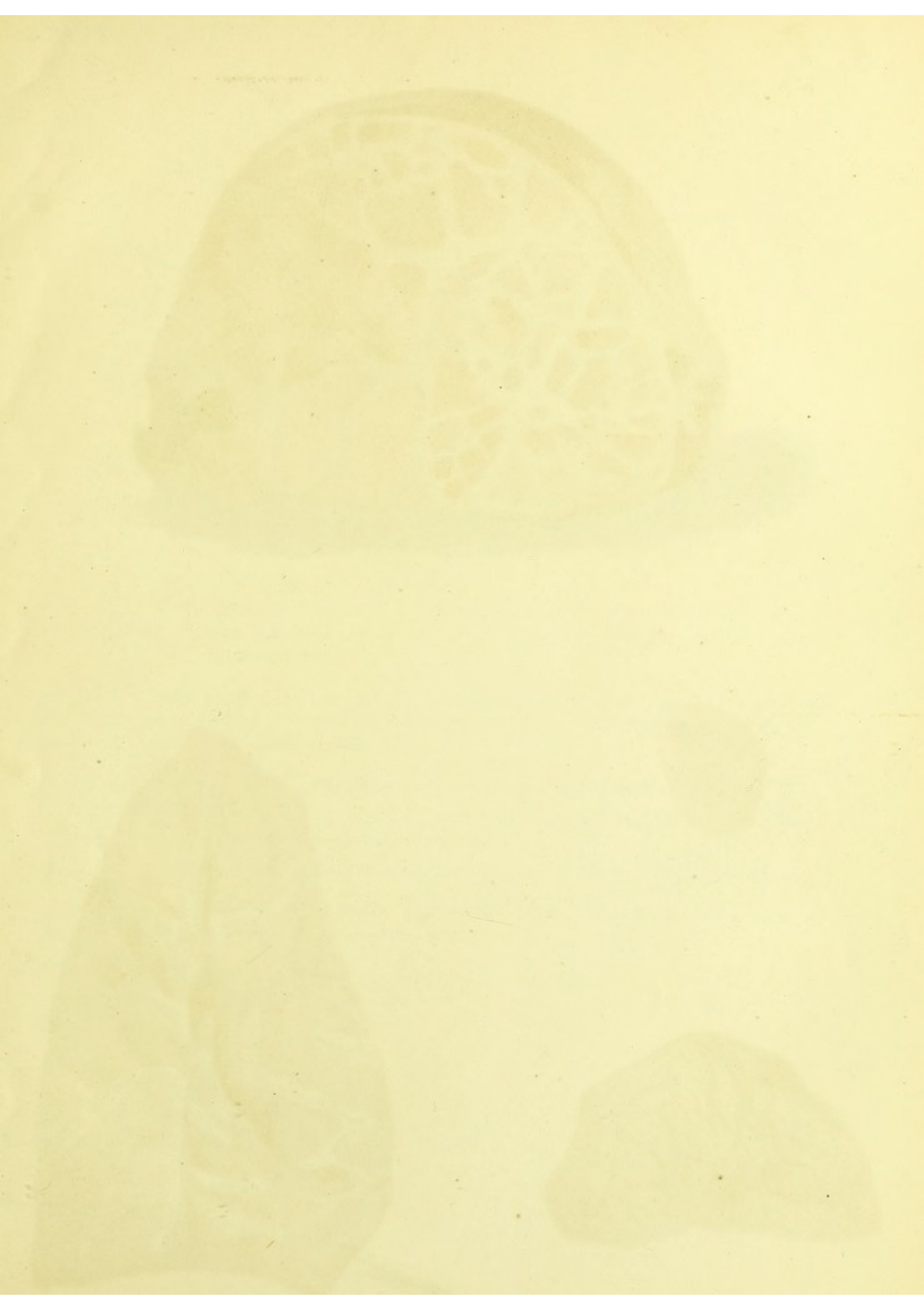

PLATE. I.

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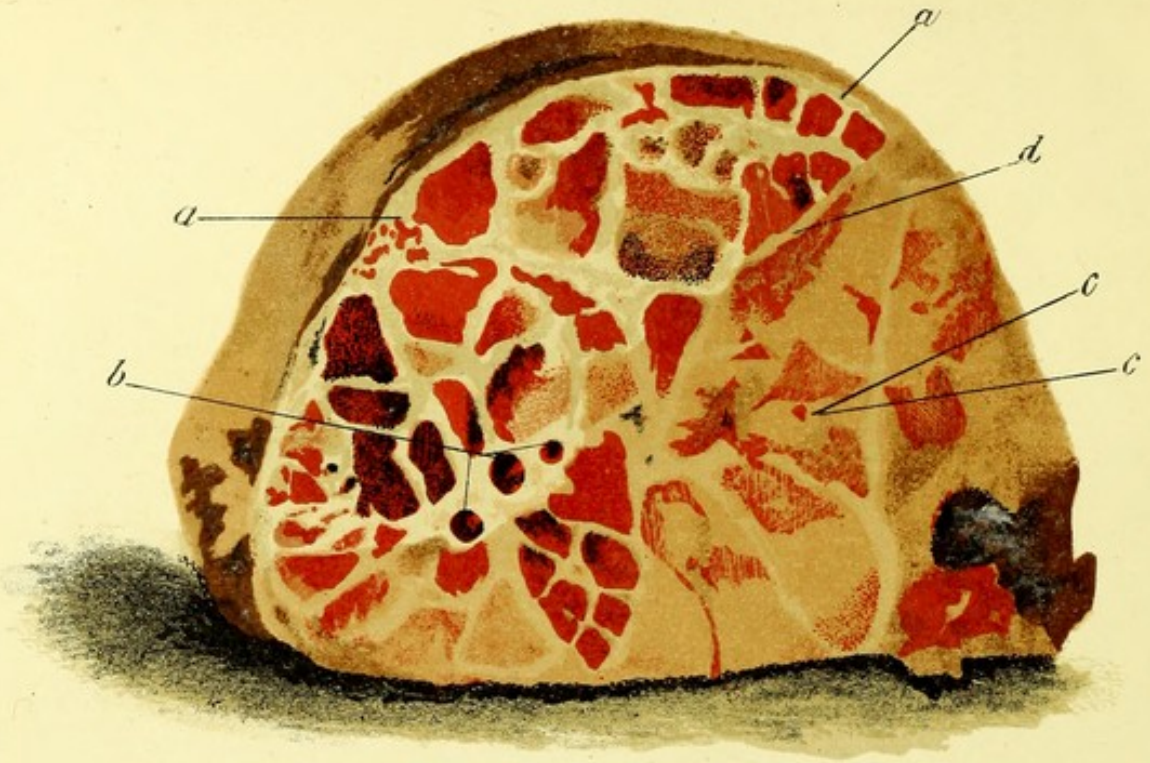

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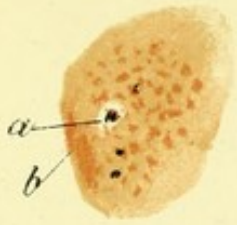

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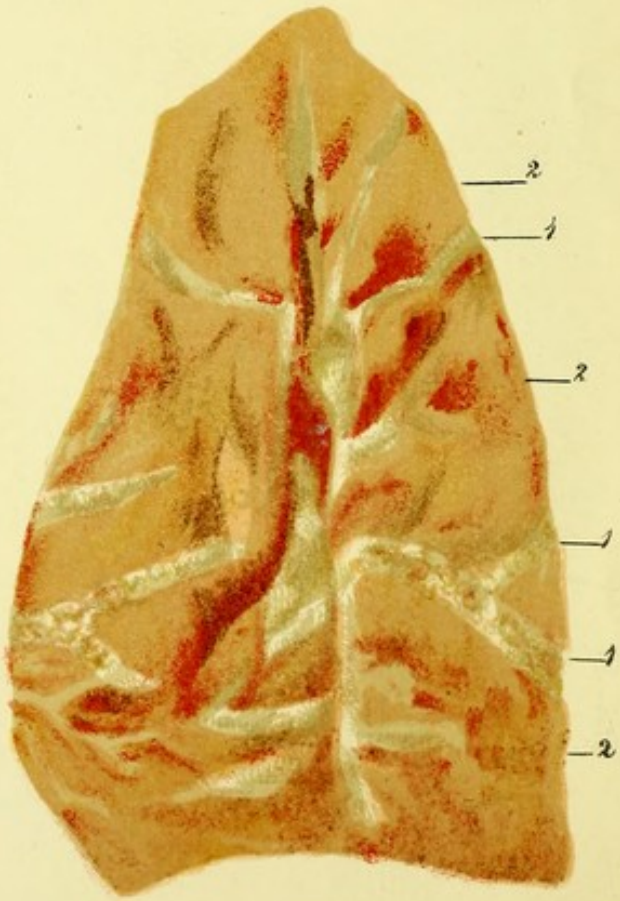

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PLATE I.

FIG. 1. *Transverse section of a Lung of Cow in an advanced stage of P. p. zy.*

a. a. Yellow lines formed by the exudation and consolidation of lymph into the interlobular cellular tissue (interlobular hyperplasy).

b. Blood-vessels and bronchial tube plugged with coagula of blood or lymph.

c. c. Brick-like hepatisation (mortification).

d. The encysting lymph-band of the sphacelated lung. The dark and red patches show respectively the black and red hepatisation, with the varied intervening depths of colour.

FIG. 2. *A small section of Lung* (P. p. zy.); showing the stellate vascular spots as the result of hyperæmia of the vascular tufts surrounding the air-cells, with (*a*) the cut end of a blood-vessel, and (*b*) diffused redness.

FIG. 3. *Portion of small lobe of emphysematous Lung of a Cow*—external convex surface.

1. 1. 1. Emphysematous interlobular connective tissue; at the lower part the cellular condition of Emphysema is well seen.

2. 2. Parenchyma—of a much denser consistence, and of a more florid hue than natural.

FIG. 4. *Interlobular Pulmonary Effusion. First stage of P. p. zy.* Parenchyma a little darker in colour and firmer in appearance than normal.

PLATE II.

FIG. 5. *Trachea and large Bronchii laid open;* showing the sometimes intensely scarlet, blackened, and gangrenous condition of the mucous membrane, with (*a*) circumscribed and (*b*) diffused ecchymoses.

c. Small yellow ulcers and a large one, with new capillary vessels at *d.*

e. Openings into smaller bronchii.

FIG. 6. *Costal Pleura of Cow* (P. p. zy.); showing granulations springing from its surface after the removal of the adherent layer of lymph, with fibres of organised lymph (produced by violent detachment of lymph from pleura) projecting from the surface of the pleura.

FIG. 7. *Portion of Pericardium of Cow;* showing the dilated and injected condition of the capillaries as a result of P. p. zy.

This sketch also illustrates the condition of the pleura in some of the stages of inflammation.

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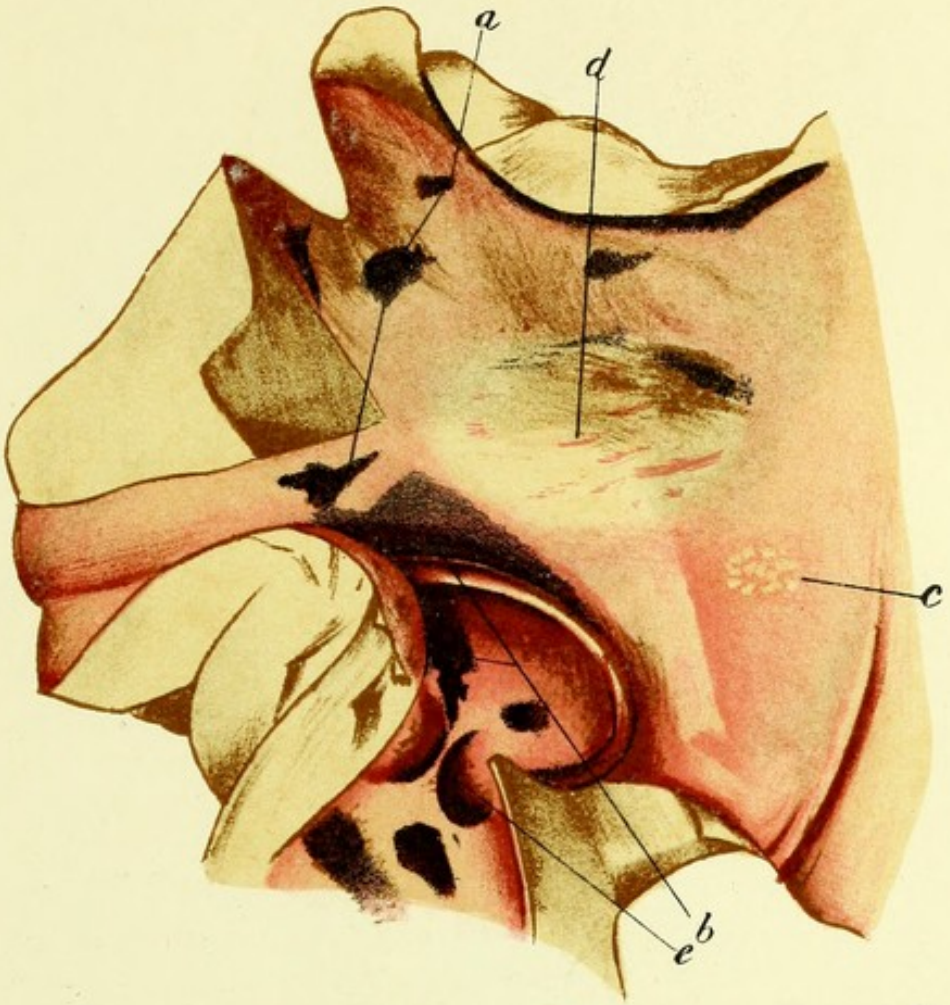

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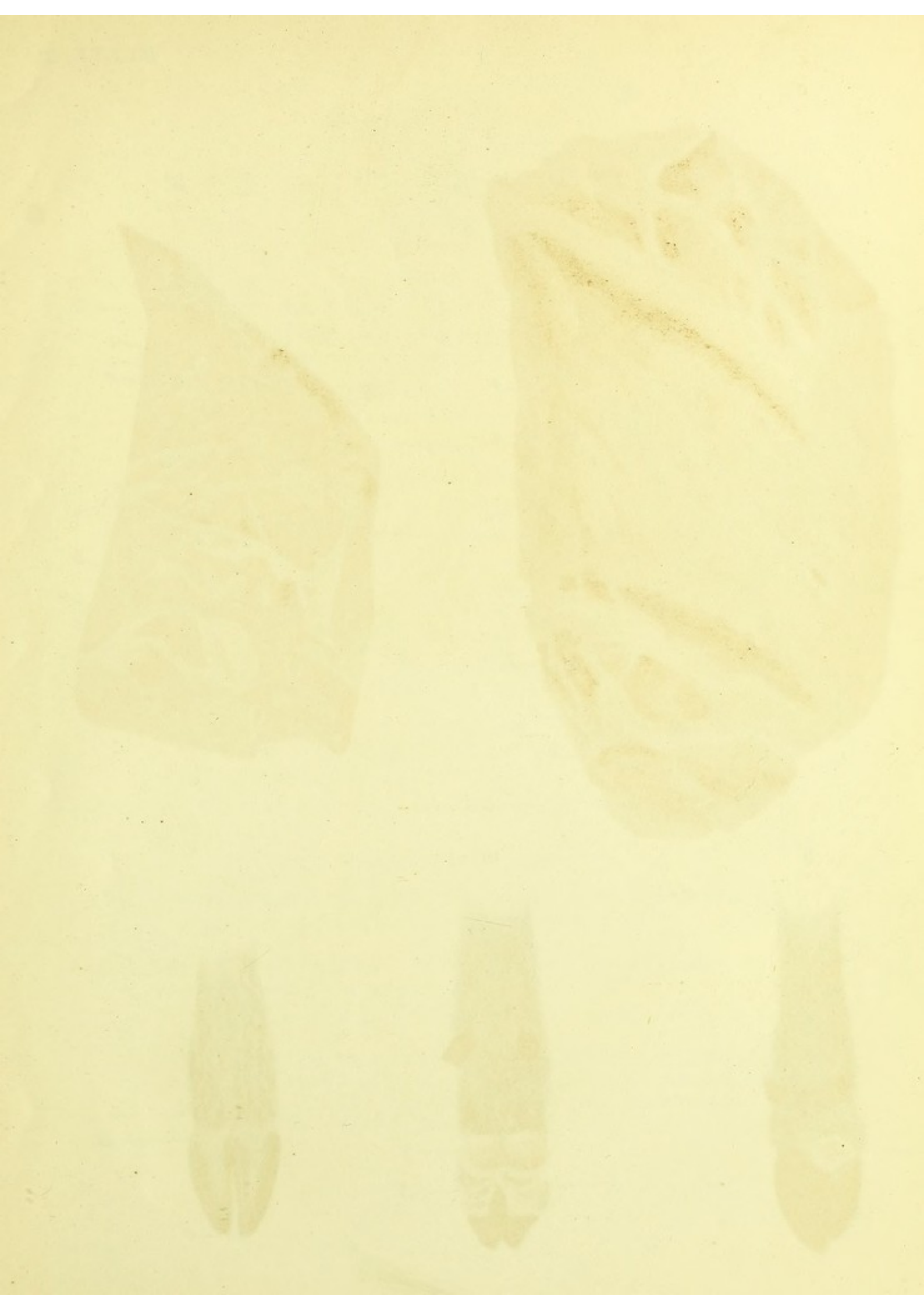

PLATE III.

FIG. 8. *Section of encysted Lung* (P. p. zy.), about six weeks after attack.

1. Adjacent healthy lung, with interlobular infiltration, and slight lobular consolidation,—the latter resulting from intralobular serous infiltration and pressure.
2. 2. 2. Encysting fibrinous band or ultimate cyst wall.
3. 3. Cavity between walls of cyst and dead structures (chasm of segregation).
4. 4. 4. Encysted dead lung (sequestrum).
5. Obliterated blood-vessel longitudinally sectated.
6. Artery transversely sectated, and with coagulum of blood in interior.
7. Collection of small abscesses from degeneration of portions of exudate,—also encysted.
8. Red hepatisation of adjacent lung,—with interlobular hyperplasy.

FIG. 8A. *Section of Lung of Ox;* showing catarrhal pneumonia.

a. a. Vesicular exudations,—some of which were softened.

b. b. Alveolar exudations,—all firm, but tending to caseation.

c. c. Interlobular exudations (hyperplasy).

d. d. Divided bronchial tubes,—the lining membrane of which is of a dark-red colour, as result of inflammation.

e. e. Lobular (red) hepatisation.

(Contrast with Fig. 38, Plate VIII., and Fig. 1, Plate I.)

FOOT-AND-MOUTH DISEASE.

FIG. 9. *Foot of Sheep* (Eczema Epizootica); showing (1) state of heel after the rupture of the vesicle,—the scarlet portion being surrounded by inspissated pus.

FIG. 10. *Foot of Sheep;* showing (2) heels with the skin connecting them in an intensely hyperæmic condition,—exposed by reflecting the horn at 3.

The redness extends round the coronet to the front of the foot, as seen in Fig. 11.

FIG. 11. *Front view of Foot of Sheep;* showing extension (*a*) of red line of Eczema Epizootica between hair and hoof, towards the orifice of the biflex canal at *b* and the interdigital space; with granulations projecting from the toe at *c* as the result of Foot-Rot.

PLATE IV.

FOOT-AND-MOUTH DISEASE.

FIG. 12. *Posterior view of Leg and Foot of a Cow* (after Fenwick); showing the inflamed state of the connecting skin of the supernumerary digits.

a. a. The inflamed skin around the supernumerary digits; and

b. The interdigital space after the bursting of the vesicle, with the jagged white edge of the lacerated skin *c.*

FIG. 13. *A portion of the Tongue of a Cow a few days subsequent to the bursting of a vesicle* showing the intensely scarlet colour of the mucous membrane after the forcible removal of portions of the layers of brown epithelium.

FIG. 14. *Small portion of the Dental Pad of Ox* (after Fenwick); showing the most frequent position of vesicle and the appearance of the part after the vesicle has been ruptured.

FIG. 15. *Tongue of Ox* (Fenwick); showing at *a* the state of the sub-mucous tissues several days after the bursting and removal of the epithelium forming the wall of the vesicle.

b. Everted edge of the boundary of the vesicle.

c. An ulcer nearly healed.

FIG. 16. *Teat of Cow in advanced stage of Eczema Epizootica.*

a. Base of teat,—the skin corrugated and thickened.

b. Scab in its natural condition.

c. Excoriated surface, from which the scab has been forcibly removed.

d. Large crust or scab extending over point of teat and blocking it up, composed of dried pus, effused lymph, epithelial scales, hair, &c.

FIG. 17. *Portion of Skin of Udder of a Cow;* showing the appearance of the ulcers in the convalescent stage of Cow-pox.

a. A small eschar nearly healed.

b. Larger one, with the white line of the cicatrix surrounding the attached scab.

c. Cicatrix after the removal of the scab.

FIG. 18. *Section through a Gastric Ulcer* (Ox)—Foot-and-Mouth Disease; showing that the diseased action does not extend past the mucous membrane.

PLATE IV

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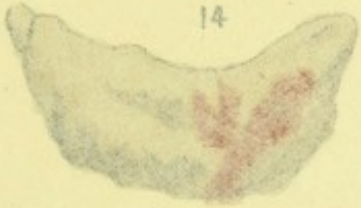

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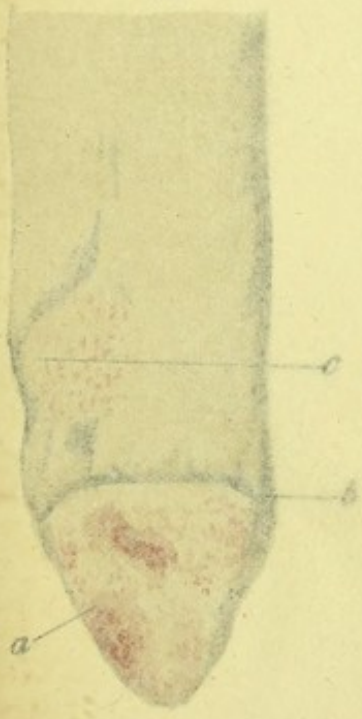

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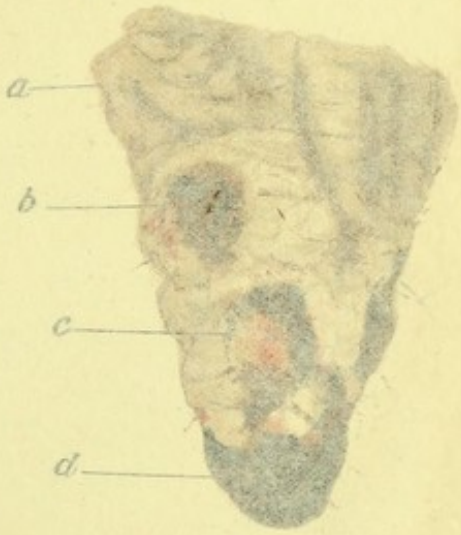

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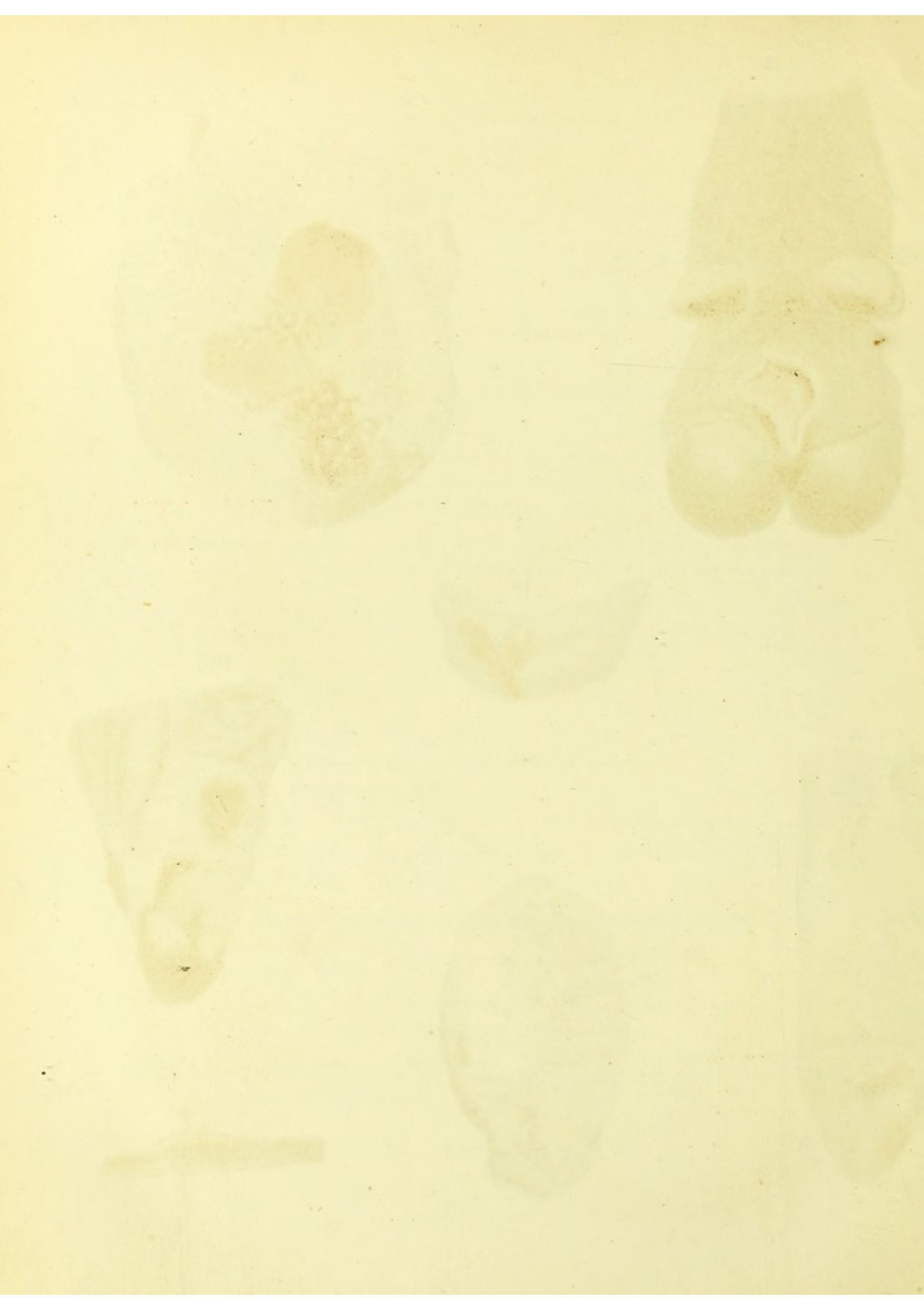

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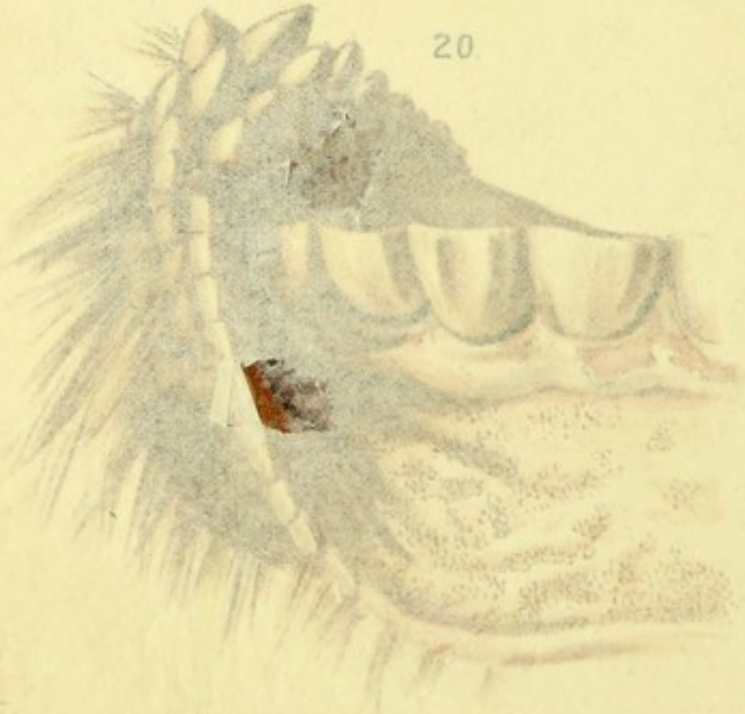

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PLATE V.

CATTLE PLAGUE.

FIG. 19. *Palate, Papillæ of Cheek, Dental Pad, and Upper Lip at the sixth day.*

The epithelium of the palate is exfoliated in patches; some of the papillæ denuded and red, others of their natural colour and appearance.

The patches of red on the dental pad—when the vascular layer of the mucous membrane is exposed—are edged with raised margins of thickened epithelium.

The drawing was taken from an animal just dead, and accurately represents the appearance during life.

(From Third Report of C. P. Commissioners, 1866.)

FIG. 20. *Section of Lower Jaw—under lip everted;* showing characteristic epithelial eruption of the mucous membrane of the lip; also the characteristic peeling off—at a more advanced stage of the disease—of the same membrane from the gum, exposing the red surface beneath.

(From Dr. Smart's Report.)

FIG. 21. *Section of Lower Jaw and Under Lip of a Calf, about four months old;* showing the denuded condition of the mucous membrane.

(From Third Report of C. P. Commissioners, 1866.)

PLATE VI.

CATTLE PLAGUE.

FIG. 22. *Vulva of Cow;* showing at

a. The mucous membrane swollen and highly congested.

b. Eruption on the membrane.

c. Ropy discharge from the vagina.

(From Dr. Smart's Report.)

FIG. 23. *Folds of Rectum of Cow;* showing the highly congested and hæmorrhagic condition of the mucous membrane.

(From Dr. Smart's Report.)

FIG. 24. *Section of Ileum* (small intestine); with

a. Peyer's patch.

b. Chronic enlargement of solitary glands.

c. Hæmorrhage of mucous membrane.

d. Mucous membrane partially degenerated.

(From Third Report of C. P. Commissioners, 1866.)

FIG. 25. *Section of smooth portion of Rumen* (1st stomach); showing sloughs in process of separation.

(From Third Report of C. P. Commissioners, 1866.)

PLATE VI.

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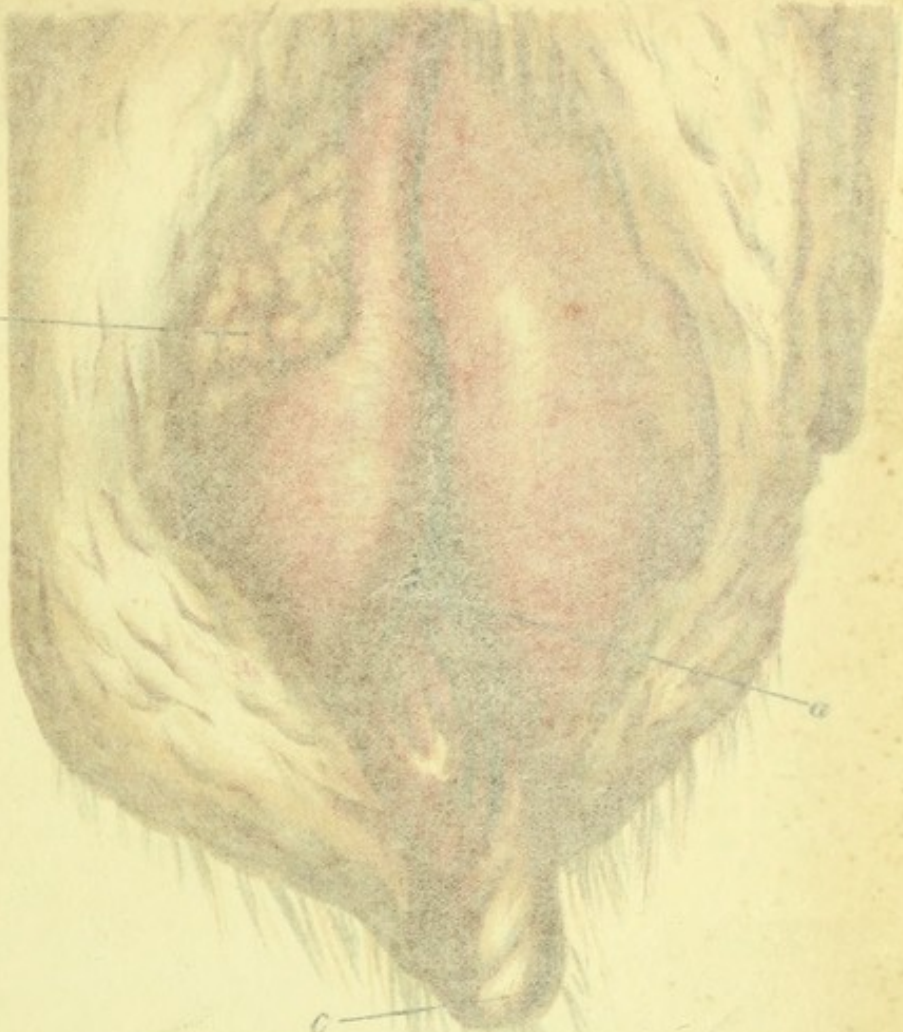

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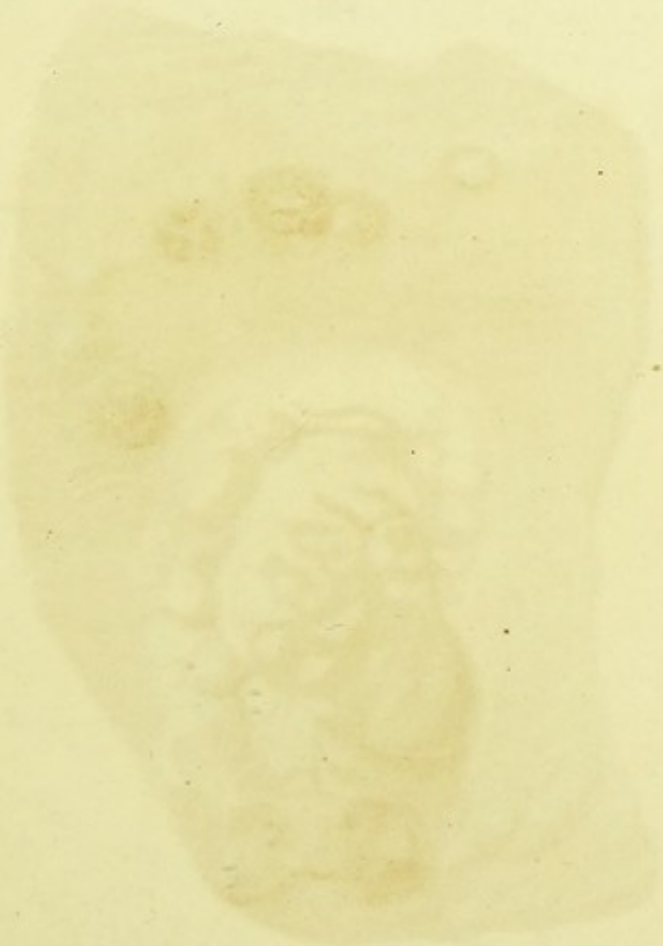

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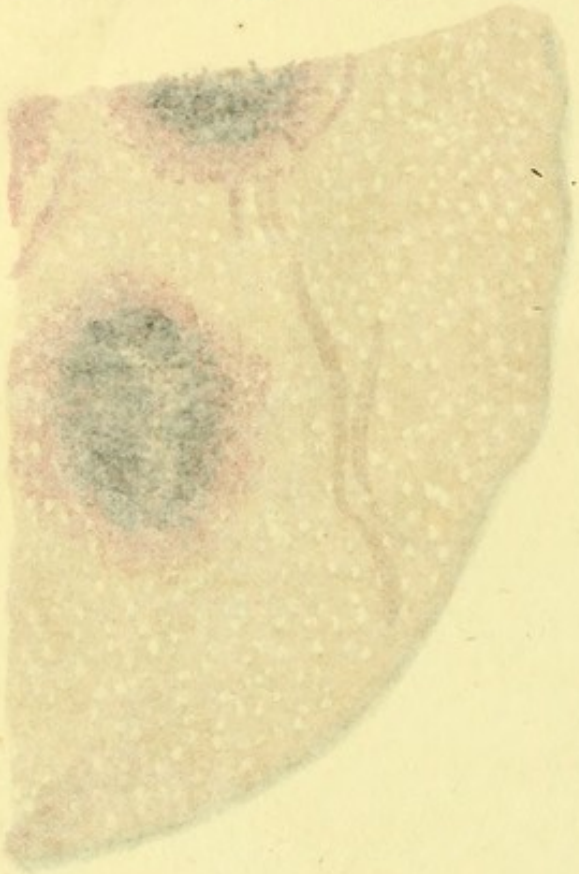

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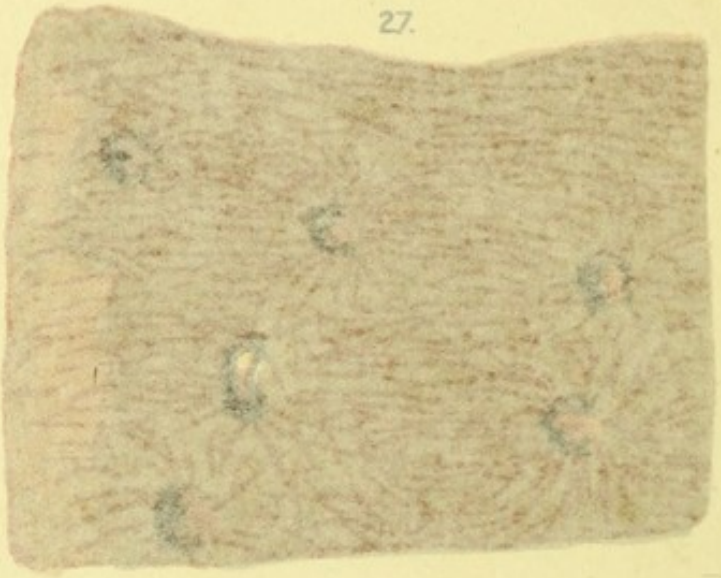

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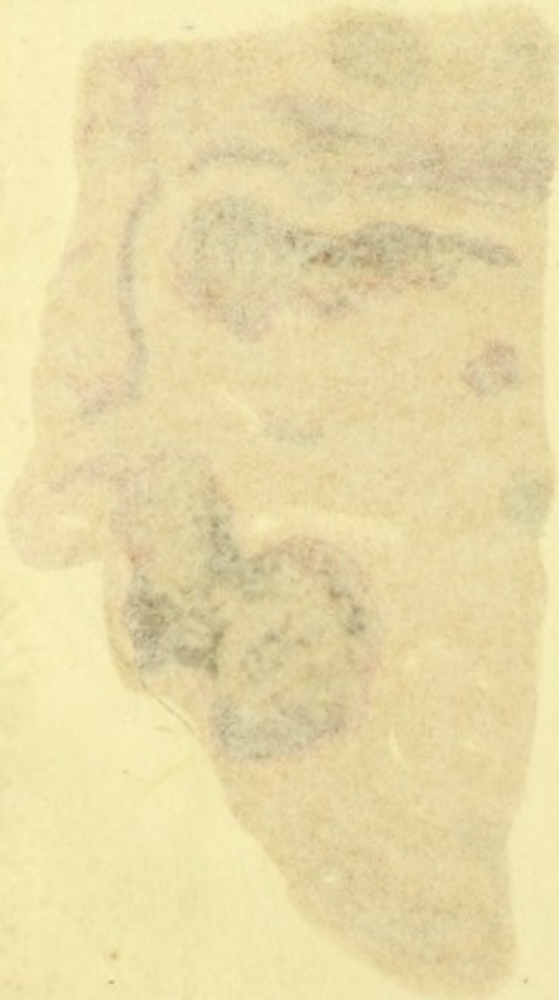

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PLATE VII.

CATTLE PLAGUE.

FIG. 26. *Section of Omasum* (3rd stomach); showing the formation of sloughs surrounded by a vascular ring.

(From Third Report of C. P. Commissioners, 1866.)

FIG. 27. *Section of Pyloric Extremity of Abomasum* (4th stomach); showing cicatrices or scars after ulceration,—the red lines indicate new vessels.

(From Third Report of C. P. Commissioners, 1866.)

FIG. 28. *Section of Pyloric Extremity of Abomasum;* showing sloughs, with a patch of congestion.

(From Third Report of C. P. Commissioners, 1866.)

FIG. 29. *Skin of Udder and Teat;* showing eruption to a very unusual extent.

a. Diffused redness of the skin.

b. Vesicles in various stages.

c. Crusts in different stages.

(From Third Report of C. P. Commissioners, 1866.)

PLATE VIII.

TUBERCLE.

FIG. 30. *Section of Liver (Cow), with circumscribed and diffused tubercle.*

a. a. Liver substance of an orange-yellow colour,—characteristic of tuberculous liver in the ox.

b. b. Two spots of a red colour, as the result of recent tubercular inflammation.

c. A mass of diffused tubercle deposited on the coats of the portal vein, *e*; with masses of circumscribed yellow tubercle, *d.*

FIG. 31. *Section of Lung of Ox, with tubercle in various stages.*

a. a. a. Small masses of tubercle.

b. b. b. Large masses of yellow tubercle, caseation.

c. A large mass commencing to liquefy and break down in the centre, with the condensed areolar tissue forming the so-called cyst or wall, *d.*

The light-blue portion at *e* represents condensed connective tissue, and the lines *f*, interlobular connective tissue; *g g*, parenchyma, or lung-substance.

FIG. 32. *Section of Lung—Progressive Fibrosis, with secondary degeneration in patches, from a five years' old Ox.*—History, unknown; but body was anæmic and emaciated, pleural adhesions existed, as also pleural granulations in patches, and both lungs were much increased in size, some portions of the lung-structure exhibiting vascular spots—stellar hyperæmia, on sectation. The lung was heavy and scirrhous; the interlobular tissue hypertrophied and indurated—nearly cartilaginous—in patches. Cause of change, sub-acute Pneumonia (probably lobar and lobular); but cause of the Pneumonia uncertain (not Pleuro-Pneumonia Zymotica). *The white lines* represent the interlobular septæ; *the yellow patches* the lobules; which presented the yellow colour depicted, and were in some places tolerably resistant, in others soft, and easily removed with the knife, while in other parts small yellow masses were seen, *like tubercle*, composed of degenerated exudate. Under the microscope the lobules were seen to be made up of extremely fine and abundant fibres, numerous epithelial cells, leucocytes, and a few exudation corpuscles. Fibrosis was progressing rapidly, and extending to the sound portions.

a. Two alveoli, from which softened portions have been scraped.

b. Portions of lung in which the change was commencing.

c. Artery.

d Bronchial tube.

e. Vein.

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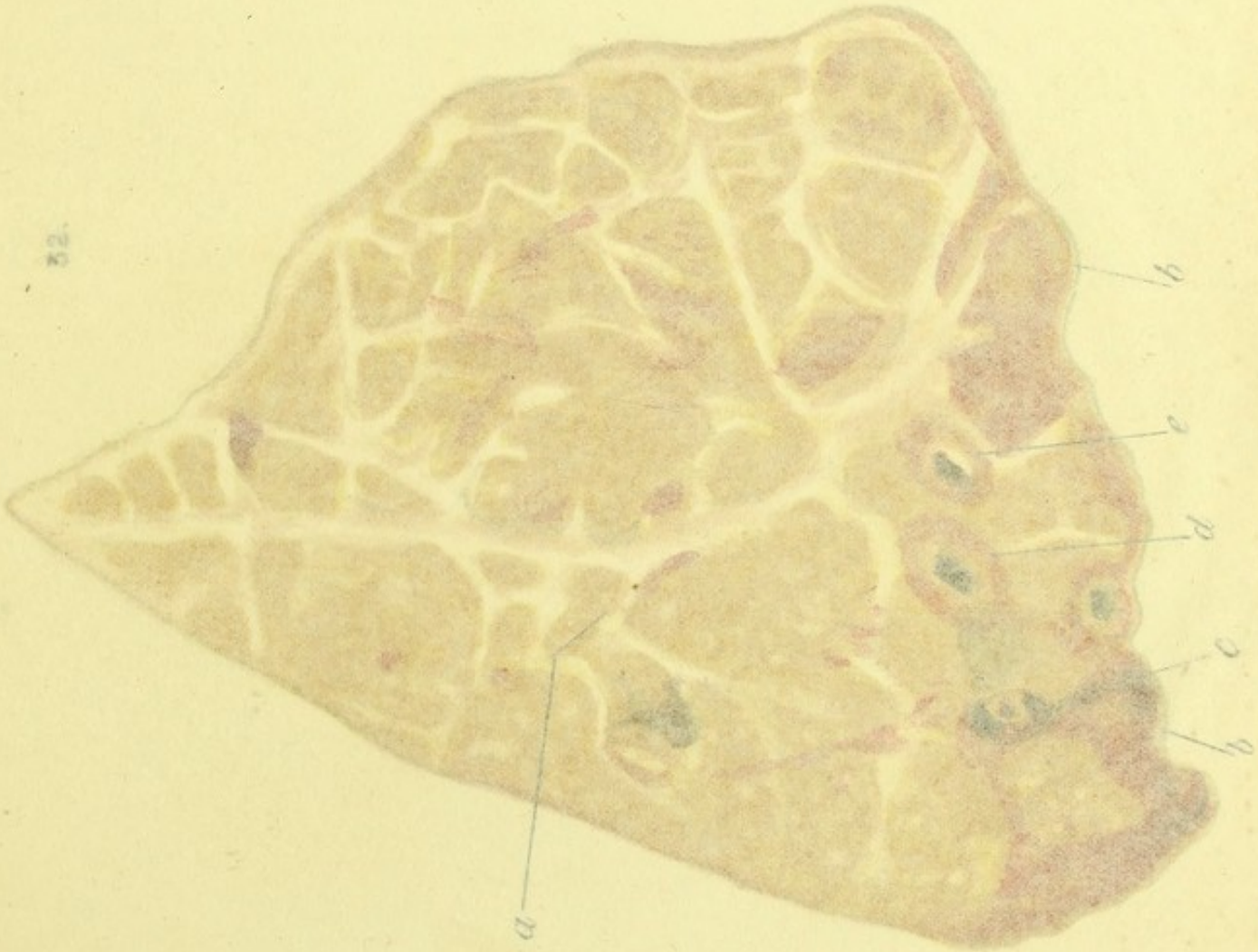

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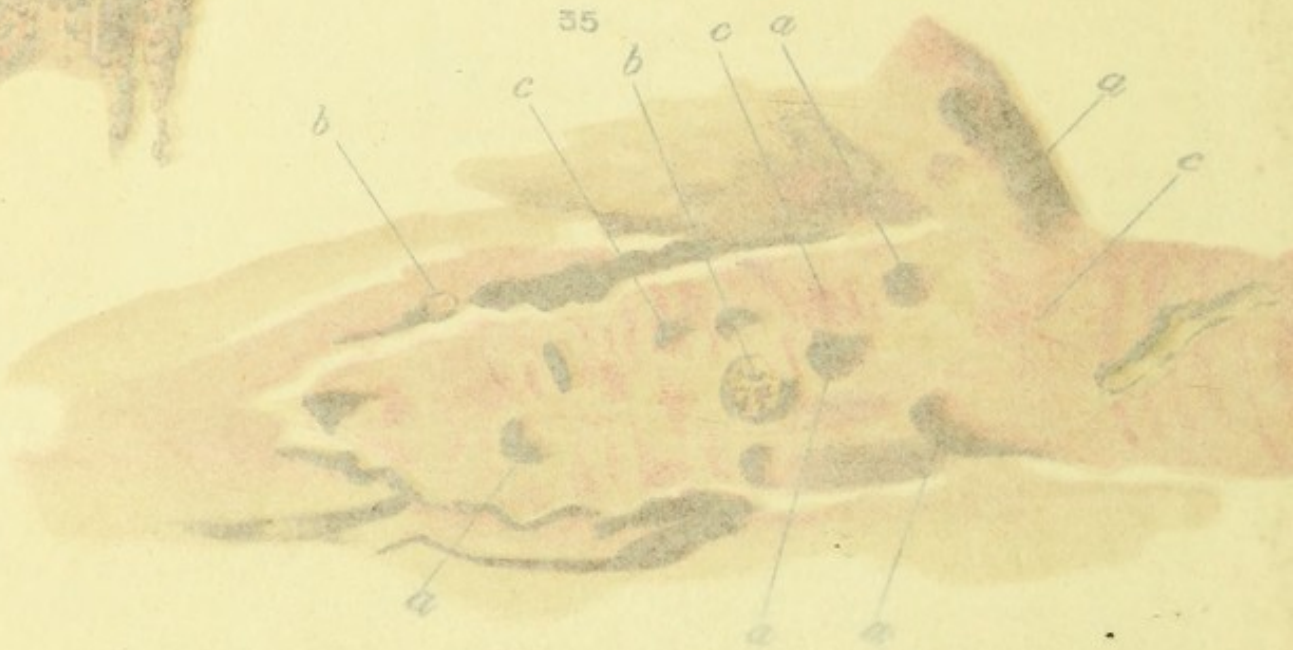

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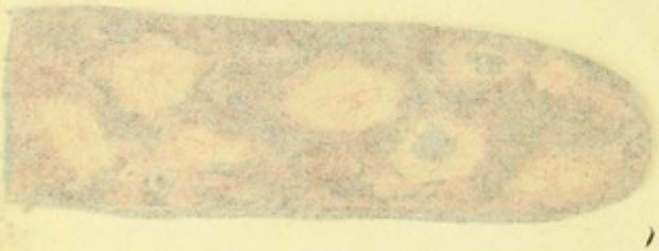

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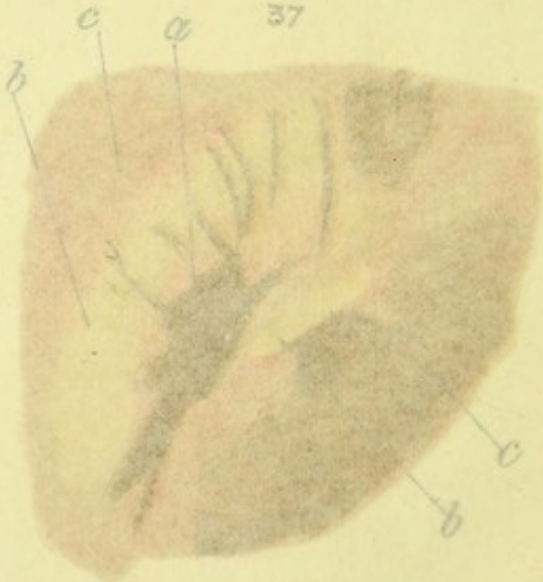

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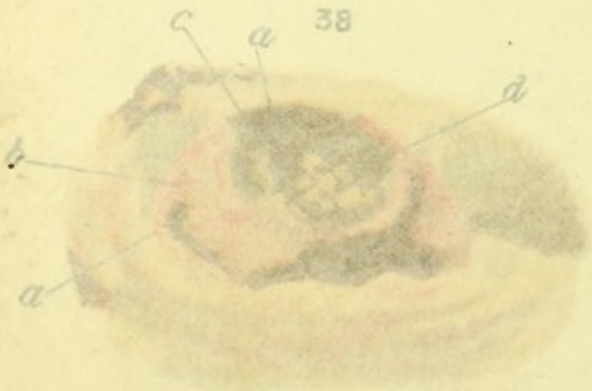


PLATE IX.

TUBERCLE.

FIG. 33. *Portion of Pulmonary Pleura* (external view), with grape-like masses of miliary tubercle connected together by fibrinous bands.

FIG. 34. *Dorsal Spine of a Cow* (Tubercle), bulged by the pressure of inflammatory exudations. Compact structure of bone thinned and absorbed. The small yellow masses (*a*) represent the tubercular deposits.

FIG. 35. *Portion of Trachea and large Bronchia of Cow* (Tubercle).

a. Openings of smaller bronchia.

b. Spherical masses of yellow tubercle underneath the mucous membrane, elevating it and causing it to bulge into the interior of the tube.

c. New cicatrices formed after the healing of tubercular ulcers, showing the vascular plexus of new capillaries radiating from the centre.

FIG. 36. *Section of Spleen of Horse* (Lymphadenoma); showing the circular shape and somewhat grey colour of the new growth, also the absence of any attempt at isolation from the spleen structure.

The radiating red lines illustrate the capillary vessels so frequently seen permeating this form of new growth.

The black portion shows pigmentation.

(Somewhat diagrammatic.)

FIG. 37. *Cicatrix in Lung after evacuation of a tubercular abscess.*

a. Cicatrix or depression seen on external surface of the lung.

b. *b*. Diffused sub-pleural tubercular deposit.

c. *c*. Lung.

FIG. 38. *Tubercular Ulcer of Lung, communicating with a Bronchial Tube.*

a. *a*. Circumscribing lymph-sac.

b. Vascular walls of sac after evacuation of tubercular matter—internal surface.

c. Communicating aperture with bronchial tube.

d. Small portions of yellow tubercle still adhering to the lining of the sac.

PLATE X.

TUBERCLE, ZY. P. P., AND PYÆMIA.

FIG. 1. *Larynx of Cow* (Tubercle) laid open; showing—

a. Elevation of mucous membrane by subjacent tuberculous nodules.

b. b. b. Masses of yellow tubercle exposed by ulceration and absorption of the mucous membrane.

c. Edge of ulcer after eruption of tubercle—intensely hyperæmic.

FIG. 2. *Section of Lung of a Cow* (Chronic Tubercular Phthisis).

a. Bronchial tube and artery.

b. Pulmonary lobule,—carnified by compression of surrounding tubercular deposits and new growths.

c. Large mass of obsolete tubercle; partly caseous and partly cretaceous.

d. d. d. d. Tracks of new tissue (hyperplasy of the interlobular connective) round the destroyed lobules, invaded, in parts, by small tubercular masses.

e. e. e. Masses of fat deposited under pleura.

f. Tracts of pigmentation.

FIG. 3. *Section of small portion of Lung of Ox;* showing degenerated multilocular hydatid cysts.

FIG. 4. *Small portion of Lung of Ox* (Zy. p. p.); showing degeneration of the interlobular tissue in oval patches (*a. a.*) of a yellowish-green colour; *b. b.* the compressed lobules.

FIG. 5. *Small section of Lung* (Miliary Tubercle); showing—

a. a. The circumscribed, somewhat circular, masses of tubercle.

b. b. The increase in the interlobular connective tissue.

c. c. Lobules carnified by compression (to contrast with Fig. 4).

FIG. 6. *Section of Lung of Ox* (old Zy. p. p.); showing a somewhat rare condition, in which the vitality of the lobules has been destroyed, while the interlobular tissue has retained its vitality.

a. a. a. Hyperplastic interlobular tissue.

b. b. b. Destroyed lobules undergoing caseation, liquefaction, and gradual absorption, without the formation of an abscess.

c. Segregating band.

FIG. 7. *A large Polypoid Tubercular Growth from the serous covering of the rumen.*

a. Portion of external coat of rumen.

b. Hæmorrhagic condition often seen in these growths.

c. Apex of growth of a yellowish-red colour.

FIG. 8. *Section of Flesh of Ox;* showing the dark colour and iridescent appearance so characteristic of the flesh of animals in cases where there has been deficient oxidation or great excitement.

FIG. 9. *Section of the base of left Lung of a Cow;* showing the extensive alterations which have taken place in all the structures as the result of Zy. p. p.

a. Ring of a bronchial tube.

b. Mucous and sub-mucous thickening.

c. Imperfect chasm, partially separating the membrane from (*d*) a plug of consolidated croupous exudation.

e. e. Walls of an artery longitudinally divided.

f. Partially degenerated thrombus filling the artery.

g. Prolonged thrombus from proximal extremity of *f.*

h. Thrombus continuing along a branch of the artery *f.*

i. A much larger vessel longitudinally divided.

j. Branch from same plugged by a recent coagulum.

n. Branch unplugged.

k. k. Irregular patches of granulation on inner surface of vessel.

l. Small ecchymosis under coats of vessel.

m. The same, with partial detachment of the vascular wall from adjacent structures.

o. Remains of a small lobule hepatised and slightly pigmented.

p. Remains of a small lobule, red hepatisation.

The yellowish-coloured intervening portions represent the hyperplastic and indurated connective tissue with, here and there, red streaks indicative of vascularisation.

FIG. 10. *Transverse section of lower portion of Trachea* (Zy. p. p.); disease having been located in anterior lobes.

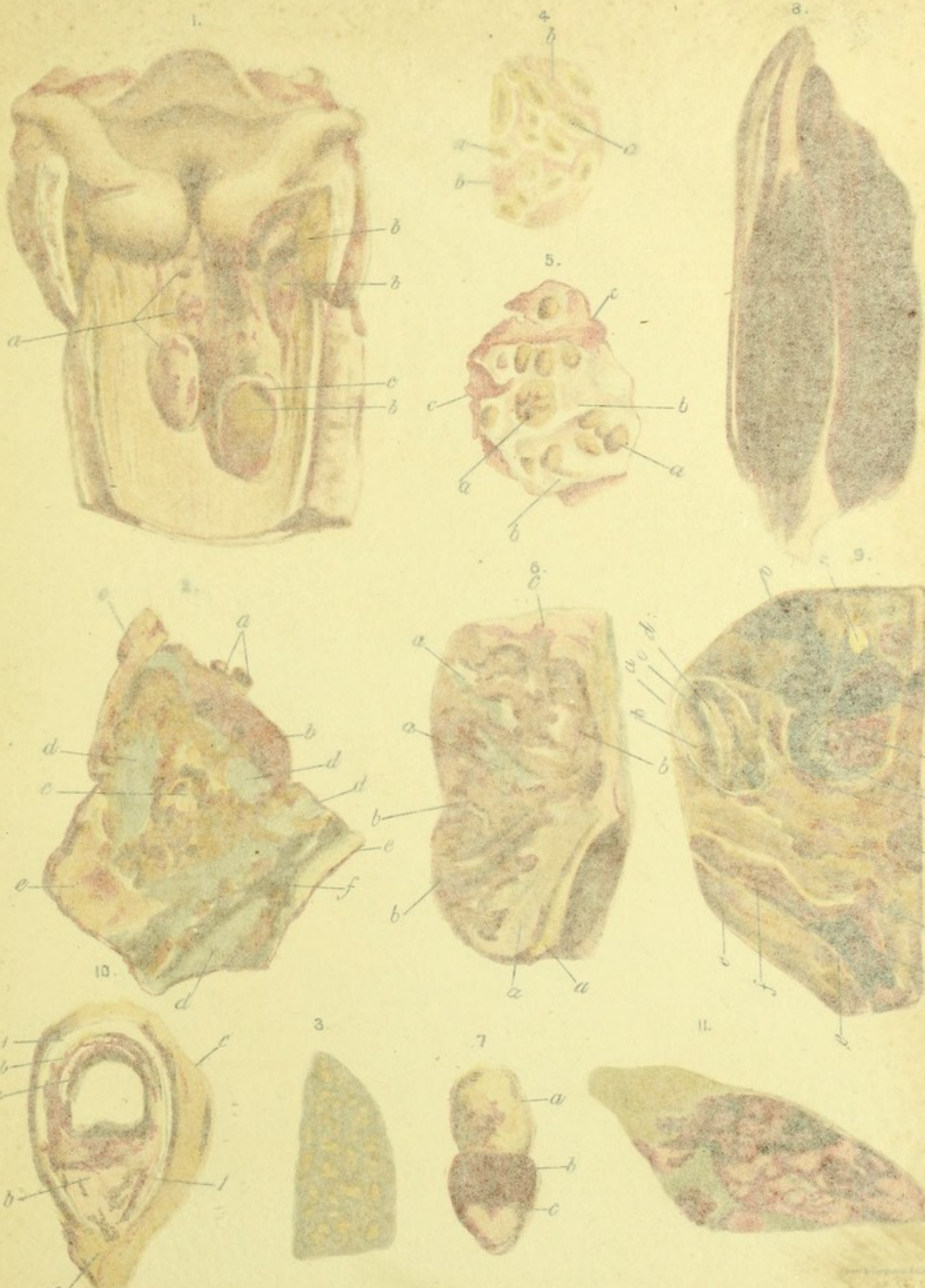
a. Mucous membrane — dark in colour, thickened and softened.

b. b. Sub-mucous tissue—much thickened, and very vascular.

c. c. External connective tissue of trachea—much thickened.

1. 1. Tracheal ring.

FIG. 11. *Section of Lung of Cow* (Virulent Pyæmia).—*The greyish coloured patches* represent numerous pyæmic abscesses and caseous nodules; *the dark intervening patches* the intensely hyperæmic condition of the lung-structure so characteristic of Pyæmia; *the greenish portions* indicate Gangrene.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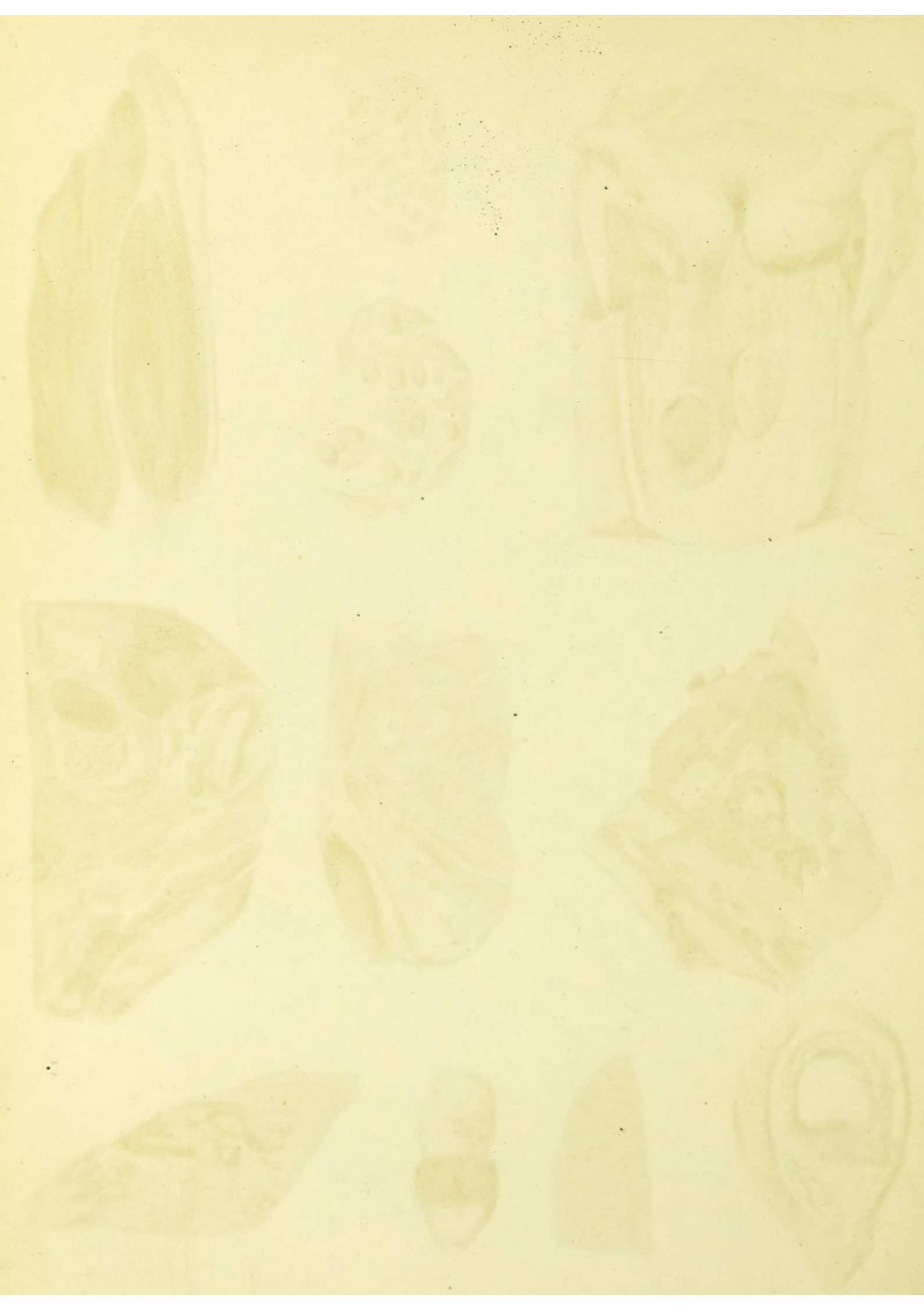

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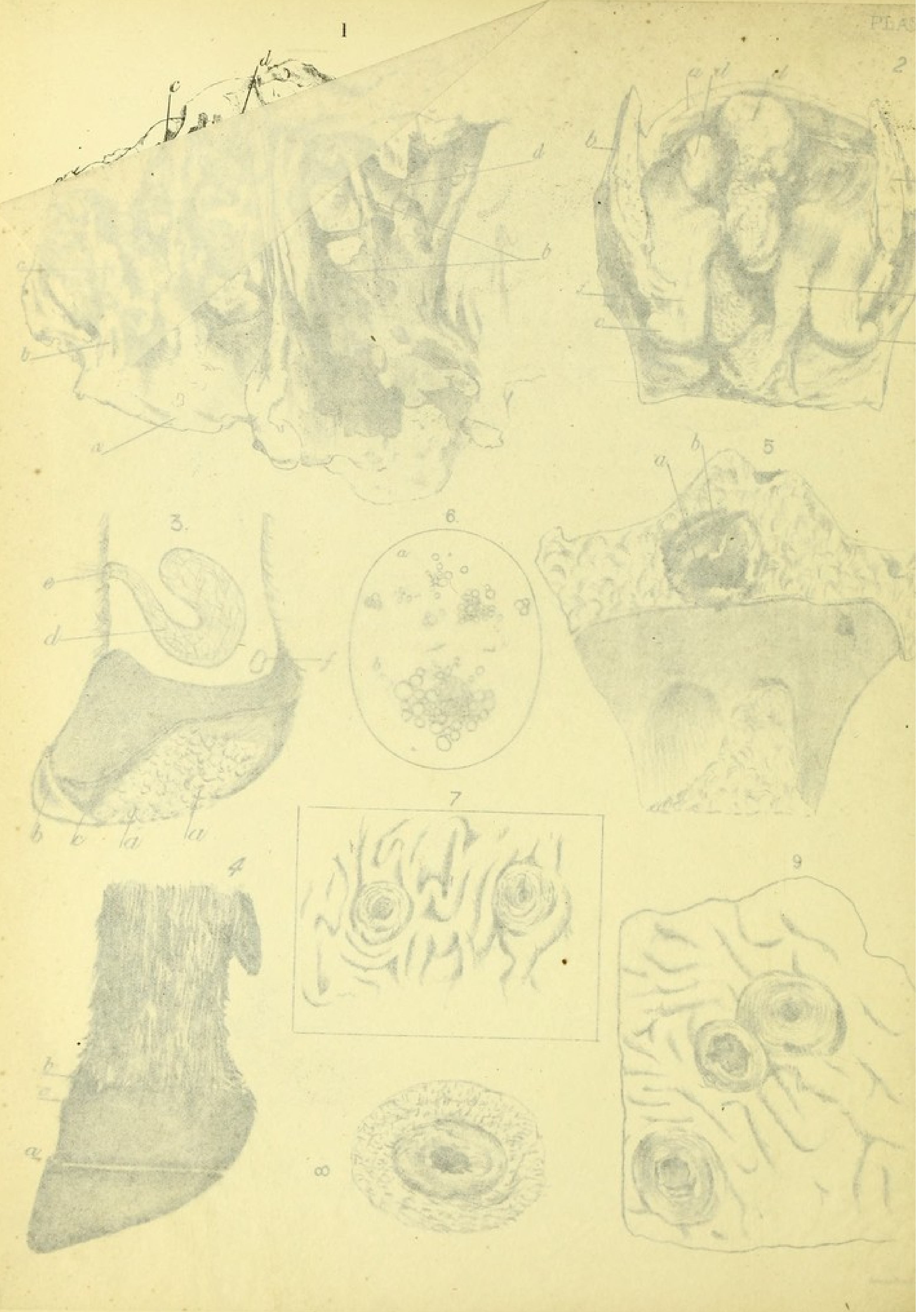

PLATE XI.

FIG. 1. *Section of Old Pulmonary* (P. p. zy.) *Cyst.*

a. a. Wall of the sac.

b. b. Vascular bands formed by organised lymph (granulations) running through the dead mass and connecting the opposite sides of the sac.

c. c. Portions of dead lung remaining in the cavities formed by the vascular bands.

d. d. Cavities from which all the dead matter has been removed.

FIG. 2. *Larynx of Cow* (laid open, longitudinally, from below); showing (*d. d.*) one large and one small tuberculous tumour situated at the posterior part of the organ; and at *e.* prominence of the mucous membrane, as the result of a tumour underneath, which contrast with the natural condition at *f.*

a. First ring of trachea.

b. b. Divided cricoid cartilage.

c. c. Apices of arytenoid cartilages.

FIG. 3. *Internal surface of right digit of Sheep's Foot;* showing Foot-Rot.

a. a. Ragged appearance produced by removal of the horn in small masses as a result of inflammation of the laminæ, and consequent interference with nutrition.

b. Excavated appearance of toe due to loss of crust and bulging of the free edge of separated horn.

c. New crust growing down to form the toe.

d. Interdigital pouch with its contained hairs, several of which are seen projecting from its orifice at *e.* The severed interdigital connecting ligament is seen at *f.*

FIG. 4. *Foot of Sheep* (Foot-and-Mouth Disease)—outside view; showing—

a. Separation of old hoof about two or three months after recovery;

b. A prominent granulation in the seat of a secondary vesicle; with

c. Partial separation of the new hoof around it.

FIG. 5. *Section of Head of Tibia of Young Ox;* showing a large tubercular excavation at *a.*, and a communicating aperture with the stifle joint at *b.*

FIG. 6. *a. Milk of Cow* (Foot-and-Mouth Disease). Aggregation of corpuscles.

b. Milk from a cow shortly after parturition, showing the natural globules of various size, with a large granular colostrum corpuscle.

FIG. 7. *Ulcers on mucous surface of large Intestine of Pig* (Enteric Fever of Pig).

FIG. 8. *Ulcer on border of posterior part of Tongue of Cow* (Foot-and-Mouth Disease).

FIG. 9. *Ulcers on mucous surface of Rumen of Cow* (Foot-and-Mouth Disease).