body. I can still remember a case of Professor, now Sir Joseph, Lister's, in which he removed an epulis from the upper jaw of an old man, laying open the cheek from the angle of the mouth to near the lobe of the ear, and I saw him shave the man over the external incision in presence of the students on the third day after. Now this wound could not be treated strictly antiseptically, but it was dressed internally with chloride of zinc and externally with boracic lotion, and the edges were kept in apposition by collodion. When the man was shaved the line of incision was visible except at one point, where on the closest inspection the eye could not detect the line of union, and close to the angle of the mouth it was not quite healed.

I have had ragged wounds in horses' quarters, caused by kicks from other horses and usually done many hours before being seen, treated as antiseptically as I thought possible, certainly without the overlying dressings, heal at once from half to three-fourths, and in some fortunate cases nearly throughout their whole length. And in these cases there is often great difficulty in getting the horses secured so that one can get accurate and satisfactory apposition of the edges of the wounds.

True enough, the introduction and advocacy of antiseptic surgery by Lister, and the interest excited thereby have revolutionised human surgery; but for all that it has not led to the Listerian system of wound dressing and treatment, although it has undoubtedly led to a better and more accurate understanding of the causes of failure, and conversely to a clearer and more correct apprehension of the principles which should guide the surgeon in his endeavour after better results.

I cannot but think that the writer of the article in question has been too sweeping in his condemnation of veterinary surgeons; yet, unless those who passed through the colleges some twenty years ago and upwards have taken other means to keep themselves conversant with the advances made in surgery than that afforded by the professional journals, it need not be matter of astonishment that the opinions and practice of many of them are perhaps a little behind the age.

PLEURO-PNEUMONIA.¹

By J. M'FADYEAN, M.B., B.Sc., F.R.S.E., Royal Veterinary College, Edinburgh.

AT the present time there is only one form of pneumonia occurring in cattle of which the Contagious Diseases (Animals) Act takes cognisance, but it is well known that both pleurisy and pneumonia may be set up in animals of the bovine species by other causes than the contagium of the disease termed contagious pleuro-pneumonia. When the clinical history of any given case is known there may be no difficulty in determining whether that case is one of contagious pleuro-pneumonia, but in the discharge of his duties under the Contagious Diseases (Animals) Act the veterinary surgeon has in many cases to decide whether the pneumonic lesions found after slaughter in the lungs of an animal whose clinical history is unknown are those of the contagious disease—in other words he has often to base his diagnosis

¹ A Paper read before the Lancashire Veterinary Medical Association, Dec. 16, 1891.

solely on the anatomical characters of the lesions. To saddle veterinary inspectors with this duty is to assume that the lung lesions in contagious pleuro-pneumonia are absolutely pathognomonic—that there is no other known disease in which similar or indistinguishable alterations of lung structure are found. I propose to discuss whether that assumption in the present state of our knowledge is well founded. And in the first place I must crave indulgence while I describe the morbid anatomy of an ordinary case of contagious pleuro-pneumonia.

At the outset it is to be noted that in the great majority of cases there is, as the name expresses, a combination of pleurisy and pneumonia, but the association is by no means constant. There is abundant evidence to show that the primary lesion is situated in the lung substance, and that the implication of the pleura is a secondary process. The hypothetical germ of the disease appears to enter the lung with the inspired air, and, having penetrated into the lung tissue, it sets up in the area in which it multiplies changes that lead to the peculiar form of consolidation presently to be described. Until this consolidation has at some point advanced up to the surface of the lung the pleura remains healthy; but whenever the lesion, in the process of extension, reaches the superficial lobules at any part, the overlying pleura immediately becomes affected. Passing over for a moment the characters of this pleurisy, let us examine the alterations usually discovered in the lung substance.

The extent of lung affected is of course very variable; the lesions may involve the whole of one lung or the greater part of both, and the only limit in the upward direction is that caused by the fact that a certain amount of healthy lung tissue is necessary for the continuance of life. Theoretically, we ought to expect to find all gradations of extent, down to the very smallest lesions, for in every case the disease must start from a point of microscopic dimensions. Nevertheless, even under the operation of the Slaughter Order, very minute lesions are seldom detected. This may be due to the progress of the lesion being very slow during the first days or weeks of the period of incubation, and more rapid when once it is fairly started, but probably it is in part explainable by the fact that until the lesion has reached a certain extent and invaded a certain number of lobules it does not acquire those features which we regard as characteristic of the disease. In other words, we might find some thickening of a septum, some effusion into a single lobule, or a spot of disease in a bronchus, but we do not know any character that would absolutely stamp any one of these as being caused by the virus of pleuro-pneumonia. The very onset of the disease will not be recognisable until we discover the causal organism, and learn how to demonstrate its presence in the tissues.

When the consolidated part of a pleuro-pneumonic lesion is incised in an ordinary case of the disease, the cut surface presents a marbled appearance. This marbling is determined in the first place by the intersection of prominent pale yellow lines or bands, which correspond to the "veins" in a slab of marble. These, as everyone knows, are sections of the altered interlobular septa. Regarded from a little distance, the septa may appear homogeneous, but when closely inspected they show a variable structure. In some portions of the consolidated area the septa may be described as composed of a

meshwork of delicate connective-tissue filaments saturated with a straw-coloured liquid, which exudes from them as soon as they are cut across. This liquid—the so-called “lymph” of inoculators—is in reality contained in the greatly distended lymphatic spaces and vessels of the septa; and in the latter, vessels which normally are quite invisible to the naked eye may have acquired the diameter of a crow quill. When the liquid escapes from the lymphatics it usually soon forms a soft coagulum. The apparent breadth of the septa varies greatly, but this is partly due to many of them being cut very obliquely, while others are cut straight across. Towards the margin of the consolidated area the thickening of the interlobular connective tissue shades off gradually, but it is usually traceable into the non-hepatised lung tissue.

The so-called lymph is not present in the liquid state in all the thickened septa. Some of the latter are beset, not with fluid lymph, but with coagulated fibrin, and these acquire in consequence a greater firmness and friability than are possessed by the septa in which the lymph is in the liquid state. Moreover, in some parts the septa may be still firmer and actually fibrous in appearance.

Turning in the next place to the lung parenchyma, we find that this presents alterations that form an essential part of the picture. The sections of pulmonary lobules cut in various directions appear as coloured islands surrounded by the pale septa, and if the consolidation is of any considerable extent these islands are not uniform in colour. The shades of colour commonly present are buff or yellow, light red or salmon-coloured, deeper red or flesh (muscle) coloured, and deep dark red like extravasated blood. The density of the parenchyma, like that of the interlobular tissue, is variable, and there is generally a gradual transition from hepatised to normal lung at the periphery of the solidified portions.

In the more solid central portions of the lesion the arteries and veins are often thrombotic, and the bronchi in the same area may be plugged with coagula of various sorts, and show obvious thickening of their walls and contraction of their lumen.

What, it may now be asked, are the microscopic lesions that underlie these departures from the naked-eye appearance of a normal lung?

(1.) The interlobular septa. At the periphery of the affected part, where the first stage in the broadening of the interlobular septa is found, the rich network of lymphatic channels is seen to be distended with a fibrinous coagulum which is in general very poor in cells. The fibrin appears as a cloud of delicate threads and finely granular matter. For the most part the fibrin here seen is of *post-mortem* formation, the solid coagulum having been formed from the fluid lymph that escapes from the cut surface of the fresh lung. Although the fibrinous lymph within the lymph channels is, as already mentioned, poor in leucocytes, these are often numerous at the very margin of the interlobular septum, and I am inclined to think that such a small-celled infiltration is always prominent at the outset of the lesion in the interlobular connective tissue. The broader septa in the more solid part of the hepatised area also owe their increased thickness and firmness mainly to the coagulation of a fibrinous material in their enormously distended lymph channels. The granular character and staining reactions may indicate that this fibrin is already in process of degene-

ration, and frequently it is being invaded from the margin of the septum by leucocytes and young fibro-blast cells. In the still firmer septa of old lesions the fibrin of the earlier stage has been largely removed, and its place has been taken by young connective tissue. All stages in the formation of this—from the round fibro-blast cells to the fibrillated tissue—may be met with in one field of the microscope.

(2.) Parenchyma or lobular tissue. When sections from the peripheral part of a pleuro-pneumonic lesion are examined microscopically, the lobules may be found to be almost normal although the connective tissue surrounding them already has its thickness notably increased through the above-described lymphatic distention; and this statement applies not only to the air vesicles and infundibula but also to the intralobular bronchi. But when the connective-tissue capsule of the lobule becomes greatly thickened the lobule must obviously suffer. Some of the fleshy yellow or pink lobules have been rendered airless (1) through the escape into their most peripheral air vesicles of a fibrinous material identical with that in the lymph spaces of the interlobular connective tissue, and (2) through compression of their more central air vesicles by this same fibrinous exudate in the peripheral part of the lobule and in the connective tissue around the lobule. If the distribution of the fibrinous exudate is not uniform throughout a lobule, then it is most abundant in the peripheral air-cells. These are frequently filled with small fibrinous clots identical with the coagulum in the adjacent interlobular tissue, while the central parts of the lobules, including the bronchioles, are absolutely normal save that they are slightly compressed. A most notable feature of the lesion in the lobules is that it is not at any time associated with marked distension of the pulmonary capillaries, such as is constant in the early stage of other forms of pneumonia. The fibrinous exudate in the air-cells may be of recent formation, or it may already have undergone hyaline or fatty degeneration.

But the alveolar contents are by no means always fibrinous in character. In many lobules the change is distinctly a catarrhal one, air vesicles being filled with round nucleated cells, apparently the progeny of the swollen and proliferating epithelium. This change may affect the lobular bronchi also. Sometimes the catarrhal proliferation is associated with a peculiar transformation of the alveolar epithelium, that having become distinctly columnar in character. The fact that catarrhal changes are common in contagious pleuro-pneumonia has been strongly insisted upon by several writers on the subject, notably by Dr Gerald Yeo and Dr Woodhead; and indeed, it must be known to everyone who has paid any attention to the minute anatomy of the lesions.

In the dark-coloured areas which mark the seat of an infarction the air cells as well as the septa are crammed with blood, and in old lesions the walls of the air vesicles are thickened by the formation of connective tissue.

The occurrence of catarrhal changes within the lobules, and affecting the terminal bronchioles, has already been mentioned, but similar changes are not uncommon in the larger bronchi running in the connective tissue of the lung. Throughout the area of consolidation the peribronchial lymphatics are distended with fibrinous lymph, but, in

addition to that, the bronchial mucous membrane may show pronounced catarrhal changes. Dr Yeo says, "I have never examined a lung affected with pleuro-pneumonia in which there was not a well-marked and characteristic lesion of the bronchial tubes of the part most severely affected. No matter how small the focus of disease in the lung may be, or how little advanced a stage these morbid changes may have reached, there is always definite disease in the corresponding bronchus, extending some distance along the tube, but more or less localised to the diseased neighbourhood." The passage quoted, coming as it does from an accomplished microscopist and one who has studied with great care the morbid anatomy of pleuro-pneumonia, may serve to show the folly of concluding, as some have done, that the discovery of bronchitic changes in any case of pneumonia in the ox stamps the disease as something other than contagious pleuro-pneumonia.

The minute anatomy of the vascular lesions in pleuro-pneumonia may be dismissed in a few words. The lymphatic vessels around the artery and vein share in the engorgement with fibrinous lymph. This may be followed by a small-celled infiltration of the coats of the vessels, thickening and roughening of the intima, and thrombosis with partial or complete occlusion of the lumen.

It has already been mentioned that pleurisy is an exceedingly common but not absolutely constant accompaniment of the lung consolidation. Where the affection of the pleura is recent the free surface of the membrane is covered with a thicker or thinner layer of grayish tough lymph, and the pleural sac generally contains more or less of a turbid yellowish liquid with fibrinous coagula floating in it. In the uncomplicated disease this exudation is neither purulent nor putrid. Where the parietal and visceral pleura are not kept apart by the exudation they become united by the layer of fibrinous lymph, and in process of time this lax union may be replaced by firm adhesion due to the formation of fibrous connective tissue.

The microscopic anatomy of the pleural lesions presents little that is special or interesting. The sub-pleural lymphatics are engorged with fibrinous lymph in the early stage of the process, and the layer of lymph coagulated on the surface of the membrane is generally rather poor in cellular elements. As soon as this layer is deposited, degenerative changes begin on it, and *pari passu* it becomes invaded from its deep aspect by leucocytes and fibro-blast cells. The solid fibrin is thus gradually removed, and young cicatricial tissue is substituted for it, and this, if time permit, may lead to the production of firm fibrous adhesions between the lung and the chest-wall.

The foregoing account of the lesions commonly found in contagious pleuro-pneumonia makes no pretensions to being complete or exhaustive. The omission of any reference to the ulterior alterations, such as necrosis, caseation, gangrene, etc., is deliberate and not an oversight, for the object has been to describe only such changes as are to be found in nearly every case of the disease, in order to be in a position to consider how far these changes, taken separately or in combination, are pathognomonic. But before taking up that question there is one other point that demands consideration, viz., the sequence of the lesions previously described. Incidental allusion has already been made to this, in expressing the opinion that the pleural affection ought

to be regarded as secondary. But taking this for granted, one may ask, what is the order in point of time of the various lesions found in the lung substance? Dr Yeo¹ as the result of his investigations came to the conclusion that the primary seat of the disease is in the bronchi, and he considers the sequence of the lesions to be as follows:

- " 1. Irritation of the mucous membrane of the smaller bronchi, probably by some infective material (specific virus).
- " 2. Chronic ulcerative bronchitis, localised to a few minute bronchi.
- " 3. Occlusion of the affected air-tubes.
- " 4. This produces such changes in the air-cells belonging to the affected tubes that the lobular parenchyma becomes solid.
- " 5. As the bronchial disease progresses, the walls of the air-tubes become thickened and infiltrated with the products of chronic inflammation.
- " 6. The peribronchial lymphatics are implicated by the extension of the infective process from the bronchus, and are soon filled with dense exudation.
- " 7. The block in the lymphatics of the broncho-vascular system impedes the flow of lymph from the corresponding territory.
- " 8. The tributary lymph-channels are thus mechanically engorged, and at the same time they are irritated by infective materials.
- " 9. The inflammation of all the coats of the air-tube gradually spreads towards the root of the lung. Thus we have a deep-seated cone of typical consolidations, traversed by numerous wide whitish lines, corresponding to the swollen interlobular connective tissue (marbling). In fact, the essential features of the disease are established."

Dr Woodhead² arrived at somewhat different conclusions regarding the starting-point and progress of the lesions in the lung. He believes that the primary irritation is not confined to the mucous membrane of the smaller bronchi, but that it may directly or indirectly implicate the epithelium of the air vesicles, and set up catarrhal proliferation. He supposes that the virus passes from the air vesicles (and sometimes from the smaller bronchi) into the surrounding lymphatics, and that the peri-bronchial lymphatics are implicated by extension from the air vesicles as well as from the bronchi. The block in the lymphatics of the broncho-vascular system impedes the flow of lymph from the corresponding territory, and this is followed by the accumulation of fibrinous lymph in the air vesicles. This material finds its way into the smaller bronchi, and then to the neighbouring air vesicles, helping to set up a *catarrhal* pneumonic process by occlusion of some of the smaller bronchial tubes, and by direct irritation.

To determine the precise starting-point of the lesions of pleuro-pneumonia is, for the reason already indicated, difficult if not impossible; but there does not appear to me to be much difficulty in determining the manner in which the disease extends, and the order in which the various elements of the lung tissue become involved when once the disease has obtained a foothold. As regards this point my own observations have led me to conclusions much more in harmony with those of Dr Woodhead than those of Dr Yeo.

I think it is highly probable that the initial lesion in pleuro-

¹ Journal of the Royal Agricultural Society of England, vol. xiv., pt. 1.

² Journal of Comparative Pathology and Therapeutics, vol. i.

pneumonia is in the connective tissue at some part, and, however that may be, there is clear evidence in nearly every case of the disease that the affection spreads by way of the lymphatic vessels and spaces of the interlobular connective tissue (including the peribronchial and perivascular lymphatics), and that the implication of the bronchial and alveolar epithelium is a purely secondary process. Evidence in support of this is met with in every case in which the consolidation is extensive, for while one never finds either croupous or catarrhal lesions in lobules surrounded by healthy connective tissue, there is no difficulty in finding lobules that are absolutely or nearly normal although the connective tissue around them already exhibits marked distension of its lymphatic spaces. Moreover, where lobules are only partially consolidated their central part and the intra-lobular bronchi are always least affected. In short, the lobular disease has a centripetal mode of extension.

The catarrhal affection of the air-cells and bronchi is, in my opinion, always as much a secondary process as the changes in the walls of the blood-vessels, and my experience is entirely at variance with that of Dr Yeo regarding the order in which the bronchial mucous membrane becomes involved. Not only may the intralobular bronchi be found perfectly normal in lobules whose peripheral vesicles are filled with a fibrinous exudate, but even in completely hepatised areas all the bronchi, large and small, may have their epithelium absolutely intact, even to the retention of the cilia.

Neither the nature nor the mode of extension of the lung lesions in pleuro-pneumonia is dependent upon anything special in the structure of the bovine lung, although such an opinion has been expressed by various writers on the subject. The peculiar features of the lesion are ascribable to the specific characters of the as yet undiscovered microbe of the disease, and no further evidence of this need be cited than that all the peculiar characters of the lung lesion are reproduced when the virus propagates in such widely different tissues as those of the mammary gland or the voluntary muscles. In all cases the primary and essential change is a distension of the lymphatic vessels and spaces of the part with fibrinous lymph that is comparatively poor in cellular elements. The lesion may be regarded as a peculiar form of inflammation, comparable to that of erysipelas, but differing from that in the slowness of the vascular disturbance, in the paucity of leucocytes in the exudation, and in the absence of all tendency to suppuration. Indeed, in great measure the lesion when fully established appears more of a lymphatic engorgement than an inflammation, but doubtless this engorgement is associated with a truly inflammatory process at some points in the lymphatic system of the part.

And now we arrive at the all important question, Are the before-described lung lesions pathognomonic of contagious pleuro-pneumonia, or are indistinguishable alterations sometimes produced in the lung of the ox by other forms of irritation? The following quotations may serve to show how divided opinion is on that subject.

Professor Williams in the first edition of his work on *Veterinary Medicine* thus describes the morbid anatomy of contagious pleuro-pneumonia:

“On opening the chest of an animal which has been slaughtered early in the disease, the visible changes are redness of the affected pleural

surfaces ; the substance of the lungs is red and congested ; the redness and congestion being often in patches, whilst the lung tissue for some distance may be more or less healthy until another patch of more or less consolidated tissue is arrived at. The groups of consolidated lobules vary much in colour, some showing the deep redness of recent inflammation, whilst others present signs that the exudation is not only consolidated but is undergoing a gradual metamorphosis—softening—by which it is changed into a whitish turbid fluid, which flows from the cut surface, and to some extent capable of absorption. We have thus the red and the grey hepatisation, the red denoting the early, the grey the more advanced stages of the inflammation.

“Some have supposed that the very extensive exudation into the lung structure, and its marbled appearance, are indicative of some speciality in the inflammatory process: this is not, however, the case, for it is found that an ordinary pneumonia caused by cold, induces the same anatomical condition of the pulmonary parenchyma, the peculiar appearance being due to the anatomical fact that the lungs of horned cattle contain much connective tissue, and that the air cells are separated into groups by such connective tissue. ‘In the ox and sheep the subserous, as well as the interlobular tissue, is very thick, the lobules being distinctly visible. This distinctive conformation explains the speciality of inflammatory diseases of the lungs in those animals.’—(Strangeways).”

Galtier in his *Traité des Maladies Contagieuses* (1st edition, p. 510) after giving a description of the lesions which is much too long for quotation here, says: “The pulmonary lesions of pleuro-pneumonia are not veritably pathognomonic; they resemble those of ordinary pleuro-pneumonia of the ox, but it seems that in the latter the infiltration and exudation are less pronounced and less generalised.”

Putz, in the corresponding German work¹, regards this question as undecided and requiring further study. He quotes the following passage from Dr Pauli: “There occurs in the ox a sporadic lung affection which, both in its external symptoms in the living animal and in its morbid anatomy, is similar to if not the same as contagious pleuro-pneumonia. Spinola’s assertion that marbled hepatisation is exclusively the consequence of the organisation of the bovine lung, and not in itself a peculiarity of contagious pleuro-pneumonia, thus, like so many other statements by that experienced investigator, finds corroboration.

In their excellent text-book on the special pathology and therapeutics of the domesticated animals, Friedberger and Fröhner² describe a sporadic croupous pneumonia of the ox, and thus refer to its differential diagnosis: “The morbid anatomy of the two diseases is essentially different. In croupous pneumonia, on account of the acute course of the disease, the hepatised portions of lung are of the same age, colour, and character, and are thus completely similar; the interstitial system of lymphatic vessels is either not at all involved or only in a state of oedematous infiltration, or, if the process is very advanced, saturated with a thin fibrinous exudate with occasional white blood corpuscles. Pleuro-pneumonia, on the contrary, is characterised firstly by the different age and appearance of the hepatised areas, and

¹ Die Seuchen und Heerde-krankheiten der unserer Hausthiere, p. 84.

² Lehrbuch der speciellen Pathologie und Therapie, 1st Edition p. 248.

secondly by the prominent participation of the interlobular lymphatic apparatus. The latter is shown by the extreme inflammation of the interstitial lymphatics (lymphangoitis), with the formation of thrombi in the interior of the vessels and marked inflammatory infiltration of the interstitial connective tissue, leading to broadening of the latter; the pleura also is, in consequence of this, almost always implicated, in the form of an exudative fibrinous pleurisy."

Dr Salmon, veterinary adviser to the Government of the United States, recently referred to this subject at a meeting of the United States Veterinary Medical Association, and a special interest attaches to his views regarding the differential diagnosis of contagious pleuro-pneumonia. He is reported to have said¹:—"Those who have had experience with pleuro-pneumonia know how difficult it is to make a diagnosis when you have only one of these animals, and no way of tracing the history of that animal. You may find the condition of the lungs which resembles pleuro-pneumonia but which also resembles other diseases. We all know that pleuro-pneumonia produces a peculiar effect on the appearance of the affected lungs; but these peculiarities are also found in lungs which have received their irritation from other germs and sometimes by other agents, so that it can hardly be claimed to-day that these symptoms are always those of contagious pleuro-pneumonia. Of course, when you take these symptoms of pleuro-pneumonia and couple them with other characteristics which we generally see, then we begin to have very positive evidence of the diagnosis."

Many similar quotations might be culled from text-books and journals, but those given will suffice to show that in the experience of some observers lesions identical with those constantly encountered in contagious pleuro-pneumonia are occasionally found in animals whose clinical history warrants the assertion that they never were exposed to the contagion of pleuro-pneumonia, and which, although placed in favourable circumstances, did not communicate the disease to other animals. On the other hand, they show that others of equal experience have never met lesions identical with those of pleuro-pneumonia in animals whose clinical history proved that the disease was not due to contagion, or devoid of contagious properties.

The sharp conflict of opinion on this matter is probably due to two things; (1) to the not infrequent difficulty of tracing the contagion of pleuro-pneumonia, and (2) to loose notions regarding the morbid anatomy of that disease. Regarding the first of these nothing need be said, but the second point demands further consideration.

In most of the recorded cases of so-called sporadic pneumonia with lesions indistinguishable from those of the contagious malady, the authors lay special stress on the "marbled" character of the hepatised lung; but it is very important to observe that a certain form of marbling is in no way peculiar to the contagious pleuro-pneumonia, or even to the bovine species. A marbling due to mere increased prominence of the interlobular connective tissue of the lung is admittedly met with in sporadic pneumonia of the ox, and in some cases even in the horse and pig. But an essential feature in the marbling of contagious pleuro-pneumonia is the variety of colour presented by the ground-work or pulmonary parenchyma, and

¹ *Journal of Comparative Medicine and Veterinary Archives*, 1890, p. 664.

I agree with those who hold that we know of no other disease in which this form of marbling is produced. Moreover, I maintain that we know of no disease save contagious pleuro-pneumonia with lesions similar, as regards their nature and mode of extension, to those described in the preceding pages of this article.

Such statements as that quoted from Dr Salmon are too vague to require any direct refutation, and a sufficient reason has already been given for discrediting the opinion formerly expressed by Professor Williams and others, that the peculiar hepatitis of pleuro-pneumonia is due to the anatomical features of the bovine lung.

The great importance of having clear notions regarding this question has been prominently brought to light by a recent conflict of opinion regarding the nature of certain lesions discovered in the lung of an American ox slaughtered at Deptford in April last.

The veterinary officer of the Board of Agriculture whose duty it was to inspect the lungs of this ox discovered in them lesions which he considered to be those of contagious pleuro-pneumonia; and when the suspected organs were forwarded to head-quarters, this opinion was confirmed by Professor Brown, and Messrs Cope and Duguid. At this stage Mr Wray, one of the American veterinary surgeons stationed in this country to see that United States cattle are not wrongly condemned for pleuro-pneumonia, dissented from the diagnosis made by the officers of the Board of Agriculture; and, having submitted portions of the diseased lung to Professor Williams, he obtained from the latter an opinion corroborating his own. The first public intimation of this was made through the medium of an agricultural paper in the following terms: "Dr Wray came to Edinburgh with portions of the affected lungs in order to have them examined by Principal Williams, of the New Veterinary College, and that expert had declared the disease to be broncho-pneumonia, a non-contagious disease which is quite distinct from the true pleuro contagiosa. So clear was the evidence in support of this declaration that Principal Williams expressed his readiness at any time to demonstrate to any body of veterinary practitioners the distinctive features of broncho-pneumonia detected in these affected lungs, as distinguished from the characteristic lesions of contagious pleuro."

Prior to this, portions of the lung had been submitted to Professor Walley and myself, and in each case the opinion was expressed that the lesions present were not distinguishable from those of contagious pleuro-pneumonia.

It need hardly be said that this extraordinary divergence of opinion immediately attracted much attention in agricultural as well as in veterinary circles, and everyone will admit that it was very desirable that better evidence than the mere publication of these contradictory views should be forthcoming in order to enable the profession and the public to judge where the error lay.

Professor Williams' declared readiness to submit the diseased lung to any body of veterinary practitioners bespoke complete confidence in his own opinion, and credit must be accorded to him for having conceived what was the proper method of settling the dispute. It is, however, greatly to be regretted that he has not as yet fulfilled his promise. It is true that at a meeting of the Scottish Metropolitan

Veterinary Medical Association he did exhibit in support of his opinion a micro-photograph from the lung of the Deptford ox, but the published report of the remarks made by the members at that meeting leaves no doubt that he failed to convince them that the disease was not contagious pleuro-pneumonia. Most people would think that this hesitancy on the part of the members of the Scottish Metropolitan Veterinary Medical Association to declare from the examination of a single micro-photograph that the Deptford ox had not been the subject of pleuro-pneumonia, was highly commendable; but Professor Williams appears to have thought otherwise, for he is reported to have exclaimed that if such evidence was not deemed sufficient they had better throw microscopic examination to the winds.

So far as the profession at large was concerned, the matter thus remained as doubtful as before; and hence, with a view to satisfying themselves, and at the same time providing evidence available to the whole profession, the members of the North of England Veterinary Medical Association determined to accept Professor Williams' offer already quoted. It was accordingly arranged to devote a meeting to the discussion of the subject, and Professor Williams was invited to attend and exhibit whatever specimens he thought necessary to convince the members of that Society that the Deptford ox had been free from pleuro-pneumonia. The author of this paper was invited to attend at the same time and lay before them what might be called the opposite side of the case. This meeting was held on the 20th of last month, but Professor Williams did not attend it. His absence is in a sense regrettable, but it is doubtful whether the outcome of the meeting would have been different had he been present. Professors Hunter and Bowhill, both members of Professor Williams' teaching staff, did attend, and exhibited micro-photographs to prove that the lesions present in the lung of the now famous ox were those of catarrhal pneumonia, and not of contagious pleuro-pneumonia. On the other hand, I submitted to the members a mixed series of preparations—naked-eye, lantern, and microscopic—made from the disputed case and from a number of British cattle killed under the Slaughter Order, while suffering from contagious pleuro-pneumonia. No one present (and this applies also to Professors Hunter and Bowhill) was able to distinguish in this mixed series of specimens those belonging to the Deptford ox; and when subsequently the preparations from this animal were specially pointed out, they were accepted by every member present as examples of contagious pleuro-pneumonia.

It will be gathered from this that Professor Williams' representatives entirely failed to convince the members of the North of England Society that one or two micro-photographs can afford more reliable evidence than a careful naked-eye examination of the diseased lung in the differential diagnosis of pleuro-pneumonia. Indeed, one might put this statement more strongly, and say, that these gentlemen furnished irresistible evidence that they were themselves unable to distinguish pleuro-pneumonia by its microscopic characters; for both identified as catarrhal pneumonia, sections from a typical pleuro-pneumonic lesion in the lung of a cow—one of four animals simultaneously affected in a small byre in Edinburgh.

But there remains to be mentioned additional evidence of great

weight in support of the opinion that the Deptford ox was the subject of pleuro-pneumonia. On the occasion of a visit to the Continent during the past summer I personally submitted portions of the lungs of this animal to Professors Bang (Copenhagen), Schütz (Berlin), Johne (Dresden), Csokor (Vienna), Kitt (Munich), and Lüpke (Stuttgart), and each of these distinguished pathologists without hesitation declared that the lesions present were those of contagious pleuro-pneumonia (*Lungenseuche*).

EDITORIAL ARTICLES.

PROTECTIVE INOCULATION AGAINST BLACK-LEG.

THE Report of the Special Committee of the Midland Counties' Veterinary Medical Association on Black-Leg, which is published at a later page of this number, discloses facts that cannot fail to excite astonishment in the minds of all who have followed recent literature on the methods of conferring artificial immunity against this formidable disease.

In France, Switzerland, and Austria the inoculation of young cattle with a view to protecting them against black-quarter has for some years been carried out on a most extensive scale, and in every case, according to the published reports, with the most gratifying results. The success of the operation has generally been estimated by comparing the mortality among the inoculated and the uninoculated animals pastured in the same localities, and the invariable result thus brought out has been to show that the deaths from black-quarter are many times less among the inoculated than among the uninoculated. In some instances it is alleged that the efficacy of inoculation has been further proved by experimentally ascertaining that the inoculated animals were able to resist the injection of doses of virus that promptly proved fatal to uninoculated control animals.

Before proceeding to examine the results obtained by the Committee of the Midland Counties' Association, it may be well to briefly review what are the ascertained or generally accepted facts regarding the pathology of black-quarter.

When the well-known emphysematous tumour, which constitutes the constant and unmistakable lesion of the disease, is incised, and a little of the sanguinolent fluid that escapes from the cut surface is examined microscopically, there are brought to light a great number of bacilli which bear some resemblance to those of anthrax but are readily distinguished from the latter by their rounded ends and their motility. These organisms are invariably present at the seat of disease, and in view of this constancy they were on all hands accepted