REPORT

ON THE

PATHOLOGICAL HISTOLOGY

OF

EPIZOOTIC PLEUROPNEUMONIA.

BY

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EPIZOÖTIC PLEUROPNEUMONIA.

THIS work was undertaken at the suggestion of Professor Burdon Sanderson, to whom I am indebted for much kind advice and aid in encountering the various obstacles which presented themselves in carrying it out. I have also to acknowledge with gratitude the material assistance I have received from the British Medical Association, at whose expense the inquiry was made, and by whose liberality I have been enabled to present it to the reader in so advantageous a form. Without the faithful reproductions of my drawings, which I owe to the skill of Mr. Noble Smith, my paper would have lost a large proportion of whatever value it may possess.

The material for investigation was taken from twenty-one cases in all, from every one of which specimens were obtained for histological purposes. In every instance, the *sectio cadaveris* was made in my presence immediately after the animal had been killed. There is no room for doubt that the animals were really affected with the disease in question; for, apart from the fact that six or seven of the cases occurred at a farm near London where pleuropneumonia has been enzoötic for several years, I had the advantage of being accompanied at nearly every *post-mortem* examination by a highly competent and experienced veterinary surgeon.

Before attempting to give a description of the morbid changes which are found in the lungs of pleuropneumonic cattle, it is necessary to refer, though here we shall be as brief as possible, to one or two peculiarities in the anatomy of the bovine lung which distinguish it from that of man and of most other mammals.

Normal bovine lung.—One of these peculiarities (which, it may be added, have a direct bearing on the morbid anatomy of pleuropneumonia) is the extreme degree to which the division into separate lobuli is carried. In form, these lobuli are irregularly polygonal, and they vary in size within tolerably wide limits; in most cases, their greatest diameter, when the lung is distended, measures between one and three-and-a-half centimeters. They are joined together by loose connective tissue—so loose, indeed, that but little dexterity is required to separate them completely one from another, without injury to the alveolar substance.

On injecting salt solution, or warm solution of gelatine, by one of the bronchial branches into the alveolar tissue, the fluid which transudes through the walls of the alveoli distends considerably this interlobular connective tissue, even when comparatively little force is used in injecting, so that the lobuli are found separated from one another by crack-like spaces filled with fluid, which measure, usually, three or four millimeters across.

The bronchiole, with its accompanying vessels, which passes to each lobulus, enters it on that aspect which is turned towards the root of the lung, afterwards dividing into a varying number of branches according to the size of the lobule. The branchlets of the pulmonary artery form, at the point where they enter into each individual lobule, typical examples of the so-called *end arteries*, *i.e.*, neither they nor their branches anastomose with the vessels of the neighbouring lobuli; and the importance of this fact, which corresponds with what is known as to the arrangement of the pulmonary vessels in other mammalia, will be seen when the characters of the pleuropneumonic lung come to be described. Another point, which requires notice here, is the arrangement in the bovine lung of the lymphatic vessels. These present certain characteristic peculiarities which appear to be due to the extreme degree to which lobulation is carried, the lobuli forming more or less independent units—each representing, in fact, a miniature lung, which is anatomically complete in itself.

Under the pleura, the lymphatic vessels form, on the surface

of each superficially seated lobule, a somewhat irregular plexus, to which the radiating arrangement of the component vessels gives a roughly stellate appearance. Corresponding with the extent of the pleural surface of the lobules, these plexuses measure from one to three centimeters in diameter. They are rounded, or polygonal, in shape. The vessels, or rather sinuses, of which they are in great part composed, measure usually from a fourth to two millimeters in breadth by from two to eight in length. Joining these larger vessels together are relatively narrow canals, which are mostly provided with valves. Each stellate plexus is in communication with its neighbours through the medium of short constricted channels, the course of the lymph through which is governed by the valves which are contained in their interior. These subpleural lymphatics can be readily injected by the "puncture method", and, in making such an injection, it may be seen that the coloured fluid spreads with great ease through the wide vessels composing any given plexus, but that it passes with much less readiness from one plexus to the others lying in its immediate vicinity. On this account, it is difficult, from any single puncture, to fill with injection fluid more than four or five of these stellate networks. On the other hand, the Berlin blue, or other fluid used, passes readily from the subpleural lymphatics to the lymph vessels which accompany the bronchioles and small bloodvessels; and, *vice versâ*, it is easy to fill completely very considerable tracts of the subpleural lymphatic plexuses by injecting from the peribronchial or perivascular lymphatics; this, it need scarcely be added, is readily managed by inserting and tying the point of the syringe into the loose tissue immediately beside the wall of the small bronchus or pulmonary artery going to part which it is desired to inject.

The vessels which connect the subpleural plexuses with the perivascular and peribronchial lymphatics pass through the interlobular connective tissue, and can be beautifully demonstrated by injecting the lymphatics and the alveolar cavities simultaneously, the former with Berlin blue, and the latter with warm gelatine solution. On account of the presence of these vessels, the interlobular connective tissue which is

situated near the pleural surface of the lung is much more rich in lymphatics than that lying between the more deeply seated lobuli.

In the case of the lungs of most mammals, as is well known,* there are present lymphatic vessels which run under the pleura towards the root of the organ, and whose function it is to convey the lymph from the pleural membrane to the retrobronchial glands. It is important for our subject to notice that in the bovine lung vessels of this kind are not found. In spite of oft-repeated and careful injections of the pulmonary lymphatics, I have never been able to find any evidence in the lung of the cow and calf of the existence of such vessels. Moreover, the course which the injection fluid takes, when the point of the syringe is introduced under the pleura, indicates clearly enough what is the route normally taken by the lymph which enters the subpleural lymph plexuses. As has been said, the Berlin blue passes with the greatest readiness into the perivascular and peribronchial lymphatics. The pleural lymphatic vessels, therefore, behave towards each superficially seated lobule as if it were an independent lung in miniature and pass over its surface to join the lymphatics leaving its root. Each lobule thus, not only in regard to its blood-vessels, but also in so far as concerns its lymphatics, is anatomically independent; and it is scarcely possible in connection with the subject of pleuropneumonia to overestimate the importance of this deviation from the normal rule; for, as will be shewn further on, the morbid process in cases of the disease in question invariably spreads along the lymphatic vessels. If we ignore the above-mentioned peculiarities in the normal anatomy, many characteristics of the pleuropneumonic lung necessarily remain unintelligible.

In so far as concerns their finer histological characters, the lymphatics of the cow's lung, both those situated under the pleura as well as those in the walls of the alveoli and infundibuli, do not differ in any important point from the pulmonary lymphatics of other mammalia. We have nothing to add or

* Klein, *Lymphatics of the Lungs.*

to take away from the description of these which has been given by Dr. Klein, in his invaluable monograph on the subject, and turn now to consider the characters of the lung when it is the seat of the pleuropneumonic process.

Macroscopical characters of the pleuropneumonic lung.—A short reference to the macroscopic characters of the diseased tissue must necessarily precede the description of the minute anatomy which forms the subject proper of this report. For this purpose, we give the notes of two cases in which the lesions present may serve as typical examples of those which are most frequently encountered. These notes were written in part at the time of the *post-mortem* examination, and were completed after a more thorough study of the fresh specimens had been made at the Laboratory of the Brown Institution. Seeing that the general characters of the disease are the same in all affected animals, it has been thought better, instead of encumbering this paper with the notes of the other cases, simply to record the particulars wherein they differed from those two which are given in full.

CASE I.—(Extent of tissue involved comparatively limited.) Dairy cow, aged five years. Time of illness supposed by owner to be about a week. (Such opinions were naturally in all cases of little value.)

Left Lung.—Pleural cavity contains about a pint of serous exudation-fluid. A thin layer of false membrane glues together those portions of the visceral and parietal pleuræ which are seated over the affected part of the lung. The false membrane peels off readily. The subpleural connective tissue is infiltrated with a dull yellowish-white exudation, which hides the margins of the superficial lobuli. The diseased part, which is about the size of a man's head, is somewhat rounded in shape and occupies the lower part of the posterior lobe. A section through the infiltrated tissue shews the characteristic marbling, due to the difference in colour between the reddish-yellow lobuli and the paler exudation occupying the interlobular connective tissue. The colour of the lobuli varies at different parts of the diseased mass; at some parts it is a pale reddish-yellow, at others the tinge of red is more pronounced,

and one portion about the size of the fist has a dark-red, almost black, colour. These varying tinges of red pass gradually into one another at many points; elsewhere, again, the line of division between portions of tissue having different shades of colour is sharply defined. This sharpness of boundary is best seen in the case of the dark-red part, which is roughly wedge-shaped, with its base towards the pleural surface. The interlobular exudation also differs in its characters at different parts. At the margin of the affected mass, it is translucent and slightly yellowish in colour, with whiter points scattered here and there; at this part, it is semi-fluid and escapes in good part on incision.

The interlobular exudation in the more deeply seated portions is dull-white or greyish, with a slight tinge of yellow; and here, also, although it is completely solidified, examination with a lens shews that its surface is not absolutely uniform in colour. The subpleural exudation is seen on section to have the same characters as that occupying the interlobular tissue. The same granular appearance is found on cutting, or tearing, the consolidated alveolar substance, as characterises the human lung affected with croupous pneumonia in the stage of red hepatisation.

The boundary line of the consolidated mass is somewhat irregular, the limit of the affected tissue being at some parts sharply defined, while at others the healthy passes gradually into the diseased substance. The irregularity of the boundary is due, in part, to the fact that the fibrinous exudation extends for a greater or less distance along the vessels and bronchi which pass into the diseased region. This perivascular exudation has the same characters as that which has already been described as occurring between the lobuli.

The artery passing to the dark-red hæmorrhagic portion contains a long firmly adherent clot, which is greyish-red in colour, and is at some parts of a lighter tinge than at others. With this exception, the larger blood-vessels found in the affected mass are healthy in appearance, although their adventitia is infiltrated with fibrinous exudation.

The small bronchi passing through the affected part contain

a quantity of viscid muco-pus. Their mucous membrane is at some points irregularly swollen and paler in colour than usual. The lumen of some of the smaller air-tubes is seen to be almost occluded by this swelling, while in others the mucous membrane appears unaffected.

Right Lung is healthy. The trachea and bronchi, with their branches, excepting those within the limits of the diseased tissue, are healthy in appearance.

Two of the bronchial lymphatic glands are much enlarged, and one or two of their afferent lymph-vessels contain a firm fibrinous plug extending for a few centimeters along their interior. The walls of the plugged lymphatics are thickened. The enlarged glands are firmer than usual and present an uniform smooth surface on section. From the cut surface, there exudes a viscid, slightly yellowish, translucent fluid.

Other thoracic viscera normal.

Abdominal viscera normal.

CASE II.—(The extent of affected tissue was here so great that it is given as a contrast to the above comparatively mild case.) The pleuræ are adherent on both sides over the larger part of their surface.

Left Lung.—Nearly the whole of the posterior two-thirds of the organ is consolidated. The marbling is present throughout the diseased mass, as in the former case, whose general characters it follows closely. About a half of the posterior lobe is dark-red in colour, due evidently to the occurrence of hæmorrhagic transudation. This part has a sharply defined boundary separating it from the paler parts. Scattered through the diseased substance other hæmorrhagic masses are seen, all presenting the same sharpness of margin and varying much in size, some being so small that only a single lobulus is involved. A thick semitransparent fluid escapes on section of the diseased tissue, the quantity, however, varying at different parts of the mass. The outer parts, those next the healthy lung tissue, are the most succulent, nearly the whole of the exudation flowing gradually away when a portion of the tissue is left for some time suspended by a thread over a beaker. At other parts, the amount of the non-coagulated exudation is

relatively small, and the tissue is firmer to the touch and usually paler in colour. These drier parts are, as has been noted, situated in the depth of the diseased mass, but in their immediate vicinity at some places are portions which are much more succulent.

The colour of the alveolar tissue varies as in the previous case, and the exudation between the lobuli and under the pleura has the same character in both instances.

The mucous membrane of the bronchi which are found within the limits of the diseased mass is here and there irregularly swollen, and on the surface of the thickened membrane a few shallow ulcerations can be seen. The adventitia of the vessels and bronchi which pass through the diseased tissue is infiltrated with solidified exudation, resembling in its characters that situated between the lobuli. Those arteries and some of the veins of the hæmorrhagic parts which have been more minutely examined contain firmly adherent clots.

Right Lung.—The whole posterior lobe is occupied by the disease, the characters of which are the same as in the lung of the opposite side. In this lung, also, a large hæmorrhagic mass is found.

The connective tissue round the upper part of the two bronchi, at the point where they join the trachea, is infiltrated with exudation. Their mucous membrane, as well as that of the posterior part of the trachea, is irregularly swollen.

Four or five of the retrobronchial glands are larger and firmer than usual, and present an uniform, gelatinous surface on section.

Heart and large vessels normal.

Left kidney shows a few greyish-white mottlings scattered through its cortical substance, and its pelvis contains a calculus about the size of a bean. Right kidney is normal in appearance. Liver healthy.

These two cases will suffice to show what are the more striking macroscopical characters of the disease. Of my other cases, one presented, in addition to the lung affection, an infiltration of the loose connective tissue of the posterior mediastinum extending from the diaphragm to the retrobronchial

glands, and causing enormous swelling. The exudation here was translucent, semifluid, and slightly yellowish in colour.

In another case, two or three whitish nodules were found in the substance of the liver. Each of these was about the size of a bean, and they were firmer in consistence than the surrounding liver substance. One of them, seated superficially under the capsule, produced a slight elevation on the surface of the organ.

In a third case, one of the kidneys presented in its cortical substance the same irregular whitish mottling which was found in Case II; here, moreover, there was no calculus present in the pelvis of the gland. The corresponding kidney was normal in appearance.

In so far as regards the general characters of the diseased tissue, one case resembles another pretty closely, and on looking over the notes of the cases which formed the basis of this report, I find that they differed from one another mainly as to the particular part of the lung, or lungs, in which the disease was situated, and as to the amount of lung substance which was involved in the morbid process. As to the localisation of the lesion, there seems to be no part of the lung which is free from attack, nor, on the other hand, does the disease show any marked predilection for any particular region of the organ.

The amount of lung substance involved varied greatly in different cases, and the two examples which are recorded above sufficiently illustrate this. Such differences, however, help us but little to a better understanding of the disease, seeing that it was impossible in any given case to tell whether the disease had *ab initio* involved all the tissue found affected at the time when the animal was killed, or whether the morbid process had for some time previously been gradually extending. For, unfortunately, no reliable data were obtainable enabling us to determine the time during which the disease had existed in any of the cases which were examined. And this is only what might be expected, considering the obscurity of the early symptoms and the insidiousness with which the disease seems so often to commence. Nor are there any characters in the lesions present in any individual case from which definite conclusions as to its age may be deduced.

Most writers on the subject of pleuropneumonia seem to agree in holding it to be a subacute or chronic process, and the histology of the disease fully bears out this view. There are also grounds for believing that, while in the great majority of cases the disease, having once affected the lung, spreads more or less gradually, invading the pulmonary tissue by degrees, in some rare cases, on the other hand, having once attacked a larger or smaller tract of lung substance, it shows but little tendency to spread. The morbid process appears to spread much more rapidly in some cases than in others, and on that account the extent of tissue affected cannot in any case be received as a means of discovering the time during which the disease has existed.

It is naturally very important in dealing with the minute anatomy to try to discover, in any given diseased lung, which parts have been first affected, and which more recently, in order to learn through what stages of development the affected tissue passes. Without entering on debatable ground, I may, for convenience of description, speak of the diseased lung substance in three stages.

I. A stage in which the tissue is infiltrated with fluid, or semifluid, gelatinous exudation.

II. Where the exudation is more or less completely solidified.

III. Where the diseased tissue is characterised by being firmer, frequently paler, and usually less succulent, but which is specially distinguished by the fact that the blood-vessels are no longer pervious.

This last has naturally nothing in common with the dark-red hæmorrhagic parts, except that in both the vessels are impervious.

The hæmorrhagic enfarctions, with their resulting anatomical changes, we prefer to treat as accidental complications occurring in the course of the disease. That they do not play any essential part in its pathology is sufficiently proved by the fact that they are completely absent in many cases, and by an examination of the cause of their occurrence in those cases in which they present themselves.

The above-named stages, it should not be forgotten are

purely abitrary divisions which are assumed only for convenience sake. The tissue infiltrated with semifluid exudation, and which is usually, though not invariably, situated at the more external part of the diseased mass, as a rule passes gradually into the tissue where the exudation has become solidified, and that again into that portion of the diseased lung substance where the tissue is firmer, and the vessels are impermeable to injection fluid. On the other hand, as is noted in the two cases which are recorded above in full, in some places the portions of diseased tissue at different stages may be sharply defined from one another.

The third stage is not present in all cases, but its pathological importance has induced us to give it a place along with the other two; for it seems that through this stage the diseased tissue must pass before recovery can take place; that is, if recovery ever do result in cases of pleuropneumonia—a matter which appears not altogether beyond doubt. The macroscopical characters of this third stage are not such as to distinguish it at the first glance from the second stage, though the difference between the two is usually quite evident on more careful examination. My attention was first attracted to this condition of the affected tissue by finding that the blood-vessels were at some parts impermeable to injection fluids. We shall find that its histological structure is sufficiently characteristic.

Liquid exudation.—In turning to the finer anatomy of the disease, the fluid exudation which infiltrates the outer parts of the affected lung substance naturally comes first to be examined. It is rich in albumen, and its yellowish translucent appearance is due to its containing a variable number of red blood-corpuscles and cell elements. These cells have, for the most part, the characters of white blood-corpuscles, *i.e.*, they are pale, slightly granular, are provided with one, two, or three nuclei, and when examined on the hot stage, show an amœboid movement. Besides these, and in variable proportion, there are other cells which are larger, being from twice to three times the size of the leucocytes, or lymphoid cells, as they are often called. These larger cells are usually distinctly granular

with one large bright nucleus; more rarely they have two, or even three, nuclei; their protoplasm is often vacuolated or else contains globules of fat. They also show a sluggish amœboid movement when examined under suitable conditions. These epithelioid cells resemble in their essential characters those which are found in young tissue, or in developing cicatricial tissue, and which Ziegler* found to arise from the development of ordinary leucocytes—an opinion in which he is supported by Cohnheim.†

Ziegler's method of observation consisted in introducing under the skin of the dog small glass chambers of such a kind as admitted of their contents being readily examined with the microscope. These chambers he examined at various intervals of time after their introduction, and found that while at first they contained chiefly pus-corpuscles, these later on were found to have disappeared in great part, their place being taken by the larger cells. Ziegler did not, of course, observe directly the formation of the larger epithelioid cells from the pus-corpuscles, but his method of inquiry leaves little room for doubt that the pus-cells, through assimilation of the surrounding albuminoid matter, and most probably of one another, increased in size, and assumed the characters peculiar to the larger cells. In Ziegler's observations these epithelioid elements underwent further changes, resulting in the formation of typical *giant-cells*. In the pleuropneumonic exudation, on the other hand, these large cells seem mostly too degenerated, and break down, without advancing to any further stage of development; for, as has been noted, they very often contain large fat granules, or their protoplasm is vacuolated; besides which, there is found floating in the exudation-fluid a large quantity of granular *débris* and free nuclei, which are evidently the remains of pre-existing cell elements. It should be added, that between the pus-cells and the larger epithelioid elements all varities of intermediary forms are found.

Minute Anatomy of the diseased lung substance.—Turning now to an examination of the minute anatomy of the affected lung substance, we shall first attempt to describe its rougher histological characters as seen on examining thin sections of the hardened tissue with a low magnifying power.

On examining such sections taken from various places, passing from the healthy tissue into the substance of the dis-

* "Ueber patholog. Bindegewebsneubildung", in *Verhandl. der Würzburger p. med. Ges. N.F.*, Band x, 1876.

† *Vorlesungen über allgemeine Pathologie*, 1877.

eased mass, we find, at the portions which are least affected—those usually situated next the healthy lung substance—that the exudation is seated almost exclusively in the interlobular connective tissue or in that lying under the pleura when the section is taken close to the free surface of the lung. The alveolar substance is still comparatively unaffected although the lobuli may be completely embedded in the surrounding fibrinous exudation. The exudation, at the very outer part of the disease, has, being semifluid, disappeared in good part during the process of hardening, leaving only a few fibrinous bands strengthening the normally present connective tissue fibres which pass from each lobule to its neighbours.

Advancing further into the affected region, we find that these fibrinous bands become rapidly thicker and thicker, until the spaces situated between the lobuli are filled and distended by an apparently compact mass of coagulated fibrine. In this condition the interlobular spaces usually measure from two to four millimeters across. We have said that this coagulated exudation was apparently compact; but more careful examination shows that running through it there are a number of wide channels, whose walls, when seen in cross section, present an irregularly rounded outline. These canals, which are readily overlooked, on account of their lumen being almost invariably filled with fibrinous coagula, vary much in width at different parts, but as a rule they measure somewhere between an eighth and half a millimeter in diameter. They can, in fresh specimens, be injected with coloured fluid by the *puncture method* without great difficulty, and their nature thus more closely examined. In specimens so prepared, it is easy to convince onesself that these canals running through the coagulated exudation are in direct communication with the perivascular and peribronchial lymphatics—that they are, in short, provisional lymphatics destined to carry off the lymph from the diseased tissue. In their arrangement these canals vary much at different parts of the affected lung substance, but they form usually a tolerably rich network between the lobuli. This network is closest near the pleural surface of the lung, and closest of all in the exudation lying under the pleura.

That they are not the same lymphatics which are present in the normal lung between the lobuli and under the pleura is evident from the fact that, in the latter situation, their course is entirely different from that of the normal subpleural lymphatics, whose arrangement, as has been pointed out, is very characteristic. It has been said that they are usually filled with fibrinous coagula, and it is between these latter and the wall of the canal that the injected colouring matter finds its way.

In fig. 1, which represents a vertical section taken from a superficially seated lobule and seen with a low magnifying power, these provisional lymphatics are well shown. On the right-hand side of the drawing, it can be seen that the coagulated exudation which occupies the subpleural connective tissue *(a)* is pierced by a number of channels *(b b)*, which are partially occluded by fibrinous coagula. The dark lines running between these clots and the walls of the lymph-vessels represent the Berlin blue with which the specimen had been injected *(puncture method)* previous to its being hardened.

At the lower part of fig. 1 is represented a part of the interlobular exudation (*c*), in which one lymphatic vessel (*d*) is seen in transverse section. Towards the middle of the section, and near its upper left hand corner, can be seen the perivascular and peribronchial exudation (*f*), in which are situated several lymphatics containing Berlin blue.

In tissues which have reached what we have called the *third stage* of the disease, the above described lymphatics can no longer be injected, or else the injection passes only for a very short distance along their interior. Even here, however, the outline of the closed tubes can be seen *in situ*, they having evidently become impermeable at the time when, on the cessation of the capillary circulation, no more lymph remained to be carried off.

Independently of the presence of the lymph channels, the appearance of the interlobular exudation when seen in thin sections with a low power objective, is by no means uniform, from the fact that the meshes of the network formed by the fibrinous threads are closer at some parts than at others. This

want of uniformity in the fibrinous matter lying between the lobuli, which is rendered strikingly apparent by the variation in depth of the bluish tinge which logwood imparts, is evidently due for the most part to the gradual and irregular manner in which the coagulation of the fibrine has taken place.

Leaving for a moment the *interlobular* exudation, we turn to notice shortly what are the rougher histological characters of the *intralobular* tissue within the limits of the affected part. It has been said that, before the alveolar substance proper becomes involved, the disease products are found invading the connective tissue between the lobuli and under the pleura. Simultaneously with its appearance in these latter situations, and also, therefore, before the alveolar substance is affected, we find the exudation occupying the tissue surrounding the intralobular blood and air tubes. This condition is illustrated by the specimen from which fig. 1 was taken, and in which it can be seen that the air cells are still free from evident affection.

The perivascular and peribronchial exudation resembles in its general characters that which is found between the lobuli, *i. e.*, it consists for the most part of fibrinous coagulum. It is generally found that, although the vessels and bronchial branches are completely surrounded by a thick layer of exudation, their lumen at first remains unobstructed. In those tissues, however, which have advanced well into the *second stage* of the morbid process, and *à fortiori* in the *third stage*, the intralobular bronchioles most usually contain fibrinous coagula-

Throughout both the first and second stages, the perivascular and peribronchial lymphatics remain pervious to injection fluid. After the exudation has invaded the loose connective tissue of the affected region, and thus compressed to a greater or less degree the alveolar substance, this latter is finally found to be the seat of well-marked morbid changes.

Using a higher magnifying power, we return to the examination of the interlobular exudation. This we now see contains a varying number of cell-elements. These are for the most part young or pus cells, such as were described as being present in the fluid exudation when examined in the fresh condition. Confining our attention for the moment to the

diseased tissue in the second stage of development, we find that, at some points, the interlobular exudation is infiltrated with cells in great numbers, while at others these are only thinly scattered through the fibrinous coagulum. The key to this inequality of distribution of the pus-corpuscles is to be found in the presence of the improvised lymph-channels, which have already been described ; for it is round their walls, and in the substance of the coagula which they usually contain, that the cells are most thickly crowded. At many parts, indeed, they are so closely packed, and the intercellular substance has become so much attenuated, that the term *lymph-adenoid* might justly be used to describe the morbid tissue. At other parts, again, the tissue is not so completely flooded with cells, and the wall of the lymph canal may be surrounded by only two or three layers of pus-corpuscles, while in the exudation between the canals only isolated cells, or groups of two or three, may be found scattered. The cells themselves, however, whether they are present in large or in small numbers, are identical in appearance ; they are typical pus-cells, and differ in nothing from the cells found in tissues in the condition of simple inflammation. It is only in their distribution, and, as we shall find, also in their life history, that they are characteristic of the disease in question. Fig. II is intended to illustrate the fact that these cells are mostly found crowded round the wall of the lymph-channels, and in the meshes of the fibrinous clots which these usually contain in their interior.

At some parts, we find the interlobular exudation presenting the characters of developing cicatricial tissue. This is most commonly found near the outer boundary of the affected mass, but by no means invariably so ; of all places it is most commonly present near the pleural surface of the lung, and most so in the subpleural exudation. And it is on account of its cell-elements tending more readily to form cicatricial tissue that the subpleural exudation distinguishes itself from that lying between the lobules.

The newly formed connective tissue usually presents a laminated appearance on section, being composed of layers or

bundles of fibrous tissue within tervening spaces, to the sides of which flattened cells are applied. This form of tissue, from its resemblance in structure to that of the cornea, has been named by Köster* *keratoid connective tissue.* When seated under the pleura, however, the newly formed tissue may be supplied with blood-vessels which have reached it from the parietal pleura passing through the partially organised pleuritic false membrane. This pleuritic false membrane must not be confounded with the subpleural exudation—that lying under the visceral pleura: the former differs in no essential particular from the fibrinous exudation found between the layers of the pleura in ordinary pleuritis.

Besides the lymphoid cells, the interlobular and subpleural exudation contains a varying number of epithelioid elements. These are more commonly found under the pleura than between the lobuli; and are, generally speaking, most abundant at those parts where the tendency to organisation is most marked.

At many parts, the cells which are contained in the exudation show signs of degeneration. This is especially the case in that condition which we have termed the *third stage.* Here the lymphoid cells are usually dull, seem often to be breaking down, and their nuclei stain badly with logwood or carmine, while the epithelioid cells contain vacuoles in their protoplasm, which has often become coarsely granular or contains large fat-globules. With these latter cells also the dull appearance of the cell substance, and the difficulty with which the nuclei can be stained with colouring agents, lead to the belief that they have died before the animal was slaughtered. Cells showing signs of degeneration are also found during the *second* or even the *first stage*, while it is the rule in the *third stage* to find them either much degenerated or necrosed. In this latter stage, also, we often find the place of the cell-elements occupied by large numbers of unequally-sized angular granules, which are distinguished by their being tinged readily with carmine or logwood. They are usually found

* Köster, *Sitzungsber. d. niederrheinische Ges. zu Bonn*, 1875.

surrounding the wall of the lymph-channels, which in this stage are impervious, or in their interior. The presence of these granules, their unequal size and angular shape, together with their characteristic grouping in rings round the wall of the lymph-channel, form one of the most striking peculiarities seen on examining stained sections of pleuropneumonic lungs. They are not confined entirely to the *third stage*, though in it they are most common, but occur here and there throughout the tissue in the *second stage* of the diseased process.

Leaving the disease products found under the pleura and between the lobuli, there present themselves for consideration those changes which occur within the lobuli, and which have only as yet been referred to in their rougher characters.

The perivascular and peribronchial exudation within the lobuli resembles in many points that found between the lobules. In both, the same fibrinous exudation is present, with its unequal and irregular appearance, due to its irregular deposition or coagulation; in both there is the same peculiarity as to the distribution of the cell-elements, which have the same characters in both situations. In the tissue which we now consider, the cells are, however, on the whole more numerous, and in perhaps the majority of specimens examined the characters are those of *lymph-adenoid* tissue. In the exudation round the intralobular vessels and bronchi, too, the same tendency to organisation is found, the appearance of the cicatricial tissue being the same as in the interlobular exudation.

The lymphatic follicles which are found in the course of the perivascular and peribronchial lymph-vessels, and the importance of which in the pathology of tuberculosis has been demonstrated by Burdon Sanderson and Klein, become in pleuropneumonia early affected. They are found enlarged, the contained lymph-cells being increased in number, while the margin of the follicle is rendered indistinct and irregular by the presence of irregularly scattered cells. The usual appearance of the exudation round the vessels and bronchioles is represented in fig. III, which is taken from a specimen in the *second stage* of the disease.

In the *first stage* of the morbid process, it is the rule to find the inner and middle coats of the intralobular blood and air tubes unaffected. In the *second stage*, however, these structures frequently present a varying degree of cell-infiltration. In so far as concerns the bronchioles, while the muscular coat usually retains its normal appearance, the mucous membrane is often found infiltrated with fibrinous and cellular exudation to such an extent as to cause considerable increase in its thickness. The cells here found, being of the same kind as those which are present in the interlobular exudation, need not again be described.

No matter how seriously the mucosa be involved, it is most common to find the epithelial layer unbroken, although their dull appearance, and the imperfect degree to which their nuclei can be tinged with colouring agents, show that the cells have undergone some molecular change—most probably necrosis. The number of cells found in the interstices of the mucous membrane varies greatly at different parts of the diseased tissue, and here and there both it and the muscular layer appear scarcely affected.

As regards the arterioles and venules of the lobuli, it is usual in the second stage to find both the media and intima but little changed, while at the same time the lumen is unobstructed; at some parts, however, which are comparatively rarely met with, these structures are infiltrated with a varying number of cell-elements. It is still more rare to find any evidence of proliferation of the endothelium of these smaller vessels. Such proliferation is, however, found not infrequently at those parts which are the seat of the hæmorrhagic infarctions, and in these cases it is the rule to find the lumen of the vessel occupied by a fibrinous clot. In the *third stage*, the vessels usually contain a finely molecular substance, imbedded in which a few scattered nuclei can generally be seen; the endothelial layer is, however, unchanged in appearance.

In the *third stage*, it should also be observed that the cell-elements of the exudation, which is situated in and around the walls of the intralobular blood-vessels and air-tubes, are usually more or less degenerated, the cells being at some parts

replaced by granules, which are often angular, and which are stained more deeply by logwood or carmine than the surrounding molecular or fibrinous matter. At other parts, the cells retain their form, but are distinguished by the imperfect way in which they take on the staining of colouring agents; elsewhere, again, they have the same characters as those found in the *second stage*.

We now come to the description of the morbid changes which are found in the alveolar substance proper. The characters presented by this tissue vary greatly at different parts of the diseased mass, under the influence of causes which in some cases escape even the most careful investigation, whilst in others, the variations in appearance are manifestly due to the stage to which the morbid process has attained, or to the varying degree of organisation or degeneration which the exuded cell-elements have undergone.

At the edge of the affected part, there is often some degree of capillary congestion. This is rarely, however, a marked feature, which is only what might be anticipated, seeing that the rich liquid and solidified exudation must necessarily raise considerably the extracapillary pressure, so that, on the blood-pressure within the capillaries falling with the death of the animal, the blood will be forced out of the vessels. It is probably on this account that the diseased tissue, even near the margin of the affected part, not infrequently appears anæmic.

The air within the alveolar cavities is at first replaced by the thick fluid, rich in albumen, which has been repeatedly referred to, and which disappears during the process of hardening the specimen for the purpose of cutting sections. At this early stage, the walls of the alveoli show little change beyond some swelling of the contained nuclei and of the epithelial cells with which they are more or less completely lined.

In the deeper parts of the affected mass, we find the alveoli filled with coagulated matter, which varies in character at different places. At some parts, the air-cells are occupied by typical fibrinous coagula, in the meshes of which are seen a few scattered leucocytes and red blood-corpuscles, while the

walls present no striking change in appearance; the characters of the tissue, in short, are those found in the stage of red hepatisation of croupous pneumonia of the human lung.

At other parts, again, of the same lung, the contents of the alveoli may be composed almost entirely of cell-elements, which are usually partly epithelial and in part young cells containing a varying number of nuclei. At some places, the epithelial, or it is perhaps better to call them epithelioid, cells predominate along with a number of granules of varying size; at other parts, again, only lymphoid cells are found, which may be either closely packed together and filling up the cavity of the alveolus, or which may be more or less thickly scattered through a finely molecular substance. Or, again, the cavities of the alveoli may contain little else than this finely molecular matter, scattered through whose substance, however, there are usually one or two epithelioid cells or leucocytes. Between these different appearances, all varieties of intermediary conditions are encountered.

The walls of the alveoli and infundibula are also affected in a different manner, and to a varying degree, at different parts. They retain occasionally an almost normal appearance, while elsewhere, on the other hand, they may be greatly thickened and their interstices flooded with lymphoid cells. As a general rule, they are found somewhat thickened with a moderate number of young cells scattered through their substance.

Figs. IV, V, and VI, represent the appearances which are most commonly encountered in the histological examination of the alveolar tissue. Of these three, that condition which is illustrated by fig. VI is, perhaps, the one which is most frequently met with.

Sometimes the walls of the alveoli are found covered with a continuous layer of epithelial cells, as is represented in fig. VII; more rarely, this layer is double, two rows of cells being seen (when the wall is seen in section) superposed, and still to all appearance firmly attached to the alveolar wall. At other parts, the wall of the air-cell or infundibulum is thickened by the growth in its substance of connective tissue, as in pulmonary cirrhosis. Elsewhere, again, a condition of considerable

interest is met with, in which the alveolar and infundibular septa are thickened by strong bands, composed entirely, or almost entirely, of smooth muscular fibres, which can be readily recognised from their characteristic staff-shaped nuclei. Fig. VII is intended to illustrate this condition.

It is only within recent years that attention has been called to the fact that the lung of man, as well as of most of the domestic animals, contains unstriped muscular fibres in the septa of the alveolar tissue. So far as I can learn, Rindfleisch* was the first to demonstrate the existence of such fibres in the parenchyma of the normal lung, and their hypertrophy in certain morbid conditions. He describes looplike bundles of muscular fibres proceeding from the bronchioles at their point of entrance into the infundibula, and extending along the walls of the latter as far as their fundus, while at right angles to these loops, and running round the infundibula, are from two to four ring-shaped muscular bands lying in those alveolar septa which project furthest into the infundibular cavity. These bands he found hypertrophied in cases of the so-called "brown induration". These observations were confirmed by Stirling, Orth,† and by Buhl,‡ the latter of whom, in that form of cirrhosis which results from desquamative pneumonia, found a hypertrophy of the muscular bands in question. In some cases, indeed, he found the muscular elements so greatly predominating as to justify the term "muscular cirrhosis", which he applied to the condition. Rindfleisch,§ on the other hand, found no increase of the muscular fibres of the pulmonary parenchyma in the cases of chronic desquamative pneumonia which he examined. In various domestic animals, more especially in the lung of the cat, Eberth‖ found a very

* Rindfleisch, "Die Muskulatur der kleinen Bronchien und des Lungenparenchyma", *Centralblatt*, 1872, s. 65.

† Orth, "Zur Kenntniss der braunen Indurationen der Lunge", in *Virch. Arch.*, Band lviii.

‡ Buhl, *Lungenentzündung*, 2te Auflage, s. 58, 1873.

§ Rindfleisch, "Die chronische Lungentuberculose", *Arch. für klin. Med.*, Band xiii, 1874.

‖ Eberth, "Ueber Hyperplasie der Muskeln des Lungenparenchyma", *Virch. Arch.*, Jan. 1878.

marked increase in the muscular fibres of the infundibular septa, both in simple chronic pneumonia and in those forms of which result from the presence of various parasites. We cannot, therefore, lay much weight on the presence of this muscular hypertrophy in cases of pleuropneumonia, in which its occurrence is not rare; for it would seem that, in various chronic inflammatory processes affecting the lung, it has only to be carefully sought for in order to be demonstrated.

In those parts of the diseased tissue which present the characters of what has been called the *third stage*, the morbid changes found in the alveolar substance are, with some exceptions, fairly uniform. Here the air-cells are occluded by coagulated fibrinous or molecular matter which adheres firmly to their walls, and through which are scattered here and there a varying number of leucocytes and epithelioid cells. These cells are either partially broken down, or present an unchanged outline; but both they and the tissues forming the alveolar walls are characterised by their dull appearance and by the imperfect degree to which their nuclei can be stained with hæmatoxylin or carmine. There is no disintegration of the tissue, but the often ragged outline of the alveolar septa, and the peculiar molecular change which they have undergone, and which gives the whole section a dull uniformity of character, form an *ensemble* which is strikingly in contrast with the appearances seen on examining sections taken from parts in the *second stage* of the morbid process. It requires no great histological experience to be able to tell at the first glance that the tissue in this condition has become necrosed.

In the dark red hæmorrhagic parts, except that the capillaries are distended with red blood corpuscles, and that a large number of these are found within the alveolar spaces, the characters of tissue are the same as those found in other parts of the affected mass.

The irregularity of the boundary line between the healthy and the diseased lung tissue is, as has been said, due in part to the presence of pointed process or extensions of the disease into the limits of otherwise unaffected regions of the lung. These extensions outwards of the exudation vary in shape

and length in different cases and at different parts (in one case the morbid process could be traced stretching without interruption from the posterior lobe, where the disease proper was situated, as far as the retrobronchial glands), but they have this in common, that they occupy the perivascular and peribronchial connective tissue, and point, therefore, towards the root of the lung. Such processes are never found extending under the pleura for any distance, as we might expect were the course of the lymphatics of the bovine lung the same as that which they take in man. This tendency to spread along the lymphatics is one of the characters wherein the disease in question differs from simple pneumonic processes.

The exudation found surrounding the vessels, both beyond the general margin of the diseased mass as well as within its limits, is very uniform in its characters in different cases, and differs in no important particulars from the interlobular exudation, so that its description need not detain us further. The degree, however, to which the blood-vessels themselves and the bronchial branches are affected requires some notice, as it varies much at different parts.

The musculosa and intima of the vessels are often found altogether unchanged in appearance, and this, although, the adventitia be much infiltrated and swollen with the exuded material. In rarer instances, the middle and inner coats are found containing numerous cell-elements in their substance, and sometimes fibrinous coagula are seen between the deeper layers of the inner coat ; the swelling, however, resulting from the presence of this fibrinous exudation is never, so far as I have observed, great in amount. Where the intima is deeply affected it is not rare to find the endothelial lining of the vessel replaced by a layer of young cells, and not unfrequnetly three or four such layers may be seen. This latter condition is represented in fig. VIII. This is what is almost invariably found in those vessels which pass to the hæmorrhagic parts, at some point or other of their course, and the situation of which is easily discovered from the presence, at the affected part of the vessel, of a firm clot occupying and more or less completely occluding its lumen. It is scarcely necessary to add,

that the clot in such cases most probably resulted from the diseased condition of the inner wall, and especially from the affection of the endothelial lining.

The branches of the bronchi which pass to the diseased region are more commonly affected than the accompanying vessels. The muscular coat and mucosa are occasionally found unchanged, but usually these tissues are the seat of cellular infiltration varying greatly in amount. The mucous lining of the air-tube is almost invariably more deeply affected than the muscular coat. The histological characters of the disease affecting the air-tubes are in no important manner different from those of the other affected tissues which have already been described. The bronchial wall is swollen from the presence in its interstices of cell-elements and fibrinous exudation. This condition is illustrated by fig. IX. The cells in the less deeply affected parts are situated chiefly round the lymphatic follicles which are swollen, with ill-defined margins. At parts where the disease is more advanced, the cells are more thickly scattered through the mucosa. They are often found crowded round the walls of the racemose glands, the cells of which may be unchanged in appearance, but are often, on the other hand, broken up and their place within the gland tube taken by lymphoid cells. The epithelial lining of the bronchi are usually intact to all appearance, except at those parts where the superficial ulcerations of the mucous membrane which have already been spoken of are present. These ulcerations seem to result from the pressure on the tissue exercised by masses of cell elements, or by a large amount of fibrinous exudation.

It was noted in treating of the macroscopic characters of the disease that, in one case, the posterior mediastinal connective tissue was found greatly swollen with gelatinous semifluid exudation. The examination of this with the microscope showed it to be identical in structure with that of the exudation between the lobules.

Having finished our description of the histology of the diseased lung, we turn to notice the appearances found in the lymphatic glands which receive the lymph from the affected region of the lung.

On examining thin partially-washed sections taken from the swollen glands, we find that, situated in the *lymph sinuses*, are many of the epithelioid cells which have been described as occurring in other diseased parts. Such cells are not discoverable amongst the smaller cell-elements of the *lymph cylinders*. In most specimens, the follicles of the cortical substance are found enlarged, their margins at the same time being more indistinct than usual. In some cases, we find a thickening of the fine fibres that run across the *lymph sinuses* from the trabeculæ to the *lymph cylinders*, together with an increase of the nuclei which are seated at their nodal points. I am not, however, inclined to place much importance on this, for Dr. Klein informs me that he has observed a thickening of these fibres and a moderate increase in their nuclei, in lymphatic glands which have been taken from perfectly healthy cattle ; besides which, although this appearance was very marked in some cases, it was altogether absent in other pleuropneumonic glands. In those cases where the afferent lymph-vessels contained thrombi, the wall of the lymphatic was much thickened, its substance being flooded with young cells, the contained clot being usually also rich in cell-elements. In fig. X, a transverse section through such a lymphatic vessel is represented.

I have but little to add, in concluding this part of the report, concerning the few, and on the whole rare, changes which occur in the kidney and liver.

In the two cases where the kidney was affected, the histological characters of the diseased gland were the same in both instances. The changes found consisted in the presence between the tubules of the cortical substance, of lymphoid cells disposed irregularly, at some parts being massed together in large nnmbers and displacing or causing absorption of some of the tubules, while, at other places, groups of two or three cells were seen in the interstices between the tubuli. The tubuli themselves appeared unaffected. The disease occurred in scattered patches, some portions of the cortical substance being quite normal in appearance. The medullary tissue in both cases showed no abnormality.

In the liver, the small whitish masses were also due to the

presence of leucocytes occupying the interstices of the tissue. They were most crowded at those spaces left by the rounded angles of neighbouring lobuli. At some parts, they were found in the interior of the lobuli, the cells of which had in part disappeared.

At various parts of the diseased lung, appearances were found which seemed, at first sight, to be due to the presence of micrococci in clumps, situated chiefly in the interlobular exudation. The granules composing these little masses were nearly equal in size, and they took on the same coloration as micrococcous masses when the section was stained with logwood. None of them, however, resisted the action of caustic potash ; nor, in spite of careful and systematic examination, have I, in any single case, been able to convince myself of the presence of micrococci or of bacteria in the diseased tissue. It is well known, from the observations of Klein and Zeigler, that micrococci may be thickly scattered through a tissue and yet be unrecognisable, seeing that, although the characters of these organisms when collected in masses are well enough defined, yet when scattered they escape our means of investigation. Such organisms may, therefore, be present in pleuropneumonic lungs, though it is doubtful if we have good *à priori* grounds for believing them to be so.

The examination of the fluid exudation in the fresh condition has also invariably given negative results in so far as the search for micrococci and bacteria was concerned.

CONCLUSION.

In the foregoing pages I have confined myself to the description of facts concerning which there was scarcely room for difference of opinion ; but I have not considered it advisable to finish my report without indicating a few of the more evident conclusions which may be drawn from the histological anatomy of the disease. As has been pointed out, we have here to do with a morbid process which presents many points of resemblance to simple inflammation of the lung. The marbled appearance which is seen on section of the diseased lung substance, and many other of the macroscopical cha-

racters are manifestly due, not to any peculiarity in the morbid process, but to the special conformation of the bovine lung. As we have seen also, in its histological characters, the diseased tissue presents many analogies with that affected with simple pneumonia. In so far as the changes in the alveolar substance are concerned, these, at some parts, copy so closely the appearances found in ordinary croupous pneumonia, that it would be impossible, from a simple examination of a section under the microscope, to say whether the specimen had been taken from a case of simple pneumonia or from one of contagious pleuropneumonia. And, in fact, many writers on the subject, *e.g.*, Roll* and Bruckmüller,† consider the disease to correspond exactly with the simple pneumonia of other animals.

The changes in the alveolar tissue, however, in cases of pleuropneumonia are characterised by their want of uniformity. We find at different parts of the same lung changes which, if they were uniform, would serve to distinguish the process as a croupous inflammation; at others, again, the characters of catarrhal pneumonia are those which predominate; elsewhere, the formation of cicatricial tissue makes cirrhosis the striking feature; at still other parts, the rich hypertrophy of the smooth muscular fibres of the pulmonary parenchyma copies the muscular cirrhosis of Buhl; while, finally, the dense infiltration by young cells of the alveolar walls, while the cavities of the air-cells are crowded with epithelial elements, gives an appearance identical with that found in cases of "tubercular pneumonia". So that *the absence of uniformity in the morbid changes found in the alveolar tissue serves to characterise the disease.*

It has been noted that the most usual condition of the diseased alveolar tissue is one in which the walls of the air-cells are more or less thickened and infiltrated with young cells. These cells, in so far as their appearance is concerned, differ

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* Roll, *Lehrb. d. Patholog. in Therap. d. Hausthiere.* Wien, 1860.

† Bruckmüller, *Lehrb. d. Patholog.* Zootomie. Wien, 1862.

in no particular from ordinary pus corpuscles. We find them in great abundance everywhere through the affected mass, and they form indeed the most striking feature in the morbid histology of pleuropneumonia. Their life history, however, is peculiar, and seems in great part governed, as might be anticipated, by the amount of nutrient fluid present in the tissue in which they are situated, and by the degree of intensity of the irritant factor, whose presence is presumably the cause of their exudation or development. At some parts, we find them forming cicatricial tissue, while at others they have broken up, leaving only slight traces of their former existence. The fact that so much fibrinous coagulum is present throughout the diseased tissue may, reasoning from Schmidt's observations, be taken as evidence that a large number of leucocytes have become disintegrated. But perhaps of more importance than the modifications which these cells undergo is the absence in them of certain degenerative changes.

They frequently disintegrate, but they never shew any traces of caseation, nor of mucous degeneration. This is especially noteworthy in the case of the enlarged lymphatic glands, which never shew any tendency to caseation: a form of degeneration which is so common in them in other chronic inflammations of the lung. The cell elements in syphilis, in glanders, in lupus, and in tuberculosis, resemble in appearance the ordinary young cells found in cases of simple inflammation; but they are distinguished in the case of each of the diseases mentioned by the changes which they undergo: in syphilis it is the mucous degeneration ; in tuberculosis it is caseation, etc., etc.; and so also with the cell elements in the case of pleuropneumonia. *The cells which are found richly infiltrating the diseased tissue resemble in appearance those found in ordinary non-specific inflammation, but they are characterised by the changes which they undergo.*

The distribution of these cells in the interlobular exudation, and the peculiar manner in which they are grouped round the lymph channels, serve also to distinguish the morbid process.

It is scarcely possible to discover in what manner the disease first commences, what tissue it first attacks, seeing that it is

practically impossible to obtain specimens at a stage of the affection sufficiently early for us to be able to elucidate this point by direct observation. For although, after the disease has made progress enough to enable a diagnosis to be made, and the animal is slaughtered, a given tissue may be more deeply or more extensively affected than another, that forms no sufficient ground for believing that in it the disease has first fixed its root. I cannot, therefore, understand on what Professor Yeo has based his assertion that the disease first attacks the mucous membrane of the smaller bronchi. It is, of course, probable enough that the disease does so commence, but one requires something more than a probability to serve as the foundation for a definite assertion of this kind. For my own part, I should imagine that the disease more probably first attacks the peribronchial connective tissue; but I should not feel justified in asserting it as a definite fact. As Professor Yeo's* investigations are by far the most important and interesting researches on the pathology of pleuropneumonia with which I am acquainted, I believe that I cannot do better than quote, in his own words, the conclusions to which he has arrived. He says:

"To recapitulate, then, the various items in the pathological sequence may be thus enumerated:

"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 smaller 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.

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* *A Report on the Pathological Anatomy of Pleuropneumonia*, by Prof. Gerald F. Yeo. London, 1878.

"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-tubes gradually spreads towards the root of the lung."

As will have been gathered from the contents of the foregoing pages, it is impossible for me to endorse the views contained in this *résumé*.

It is difficult to draw absolute conclusions as to the later progress of the disease—as to what would result if the animals were allowed to live. That cure may result is asserted by various writers, and the method is supposed to consist in the affected part, which necessarily must not be of large extent, becoming dry, firm, and bloodless, and being finally separated from the healthy lung substance by a layer of cicatricial tissue. Cases not very rarely occur, in which the lungs of cattle believed to be healthy are found, on their being slaughtered, to contain a firm, dry, encapsuled part into which neither blood nor air had penetrated, and which presents a well marked marbled appearance on section. I have not been able to obtain any such specimen, nor have I found any published account of the microscopic appearances found on examination of the dried capsuled part, and cannot therefore be certain that it represented the remains of a healed pleuropneumonic mass. The peculiar kind of dry gangrene which distinguishes what I have called the *third stage* may very probably represent the first step towards this form of cure, which seems the only form that can take place, when once the disease is fairly established in the pulmonary tissue.

The anatomical features of the disease which I have attempted to describe in the preceding pages are so sharply defined and so characteristic as to leave no room for doubt as to the classification of the morbid process. It is a typical example of a "specific inflammation", or, as Klebs has proposed to name this class of diseases, of an "infection tumour". It takes its place naturally, therefore, beside syphilis, glanders, tuberculosis, etc.

In conclusion, I must express my thanks to the veterinary

officers of the Privy Council, to whom I am indebted for enabling me to obtain the greater part of the material for examination. To Dr. Klein, also, I must express my gratitude for the many friendly hints which he has given me and which, from his great experience in this and indeed of most other kinds of histological work, were highly prized.

EXPLANATION OF THE FIGURES.

FIG. I.—Vertical section from pleural surface of diseased lung. Enlargement 10 diameters. The lymphatics have been injected with Berlin blue.

a. Subpleural connective tissue infiltrated with exudation.

bb. Subpleural lymphatics containing coagula. The dark parts the coloured injections.

c. Interlobular exudation, with

d. Lymphatic vessel cut across.

e. Alveolar tissue.

f. Perivascular and peribronchial exudation with injected lymphatics.

FIG. II.—Interlobular lymphatic vessel, seen cut across, with large numbers of young cells around and within its walls.

a. Fibrinous exudation.

b. Wall of lymphatic.

c. Mass of lymphoid cells. (Obj. iv Hartnack, eye-piece No. ii, tube drawn out.)

FIG. III.—Exudation round interlobular vessels and bronchus.

a. Alveolar tissue.

b. Artery.

c. Veins.

d. Bronchial branch. (Obj. No. iv Hartnack. Ocular No. ii.)

FIG. IV.—Condensed alveolar tissue. (Obj. No. vii Hartnack. Oc. No. iii.)

FIG. V.— Do. do. (Obj. No. vii Hartnack. Oc. No. ii.)

FIG. VI.— Do. do. (Obj. No. vii Hartnack. Oc. No. iii, tube drawn out.)

FIG. VII.—Portion alveolar septum, *a*, containing *(b)* newly formed smooth muscle fibres, and covered at some parts *(c)* with a continuous layer of epithelial cells. (Obj. No. viii Hartnack. Oc. No. ii, tube drawn out.)

FIG. VIII.—Vertical section from the wall of branch of the pulmonary artery, showing the endothelium replaced by young cells. (Obj vii Hartnack. Oc. No. ii.)

FIG. IX.—Vertical section of mucous membrane of bronchus of second order.

a. Epithelium.

b. Mucous gland.

c. Muscular layer.

d d d. Young cells infiltrated at various parts of the tissue. (Obj. iv Hartnack. Oc. No. ii.)

Fig. X.—Section through an afferent lymphatic vessel passing to a diseased lymphatic gland.

a. Thickened wall of lymphatic.

b. Coagulum in lumen of vessel.

c. Connective tissue swollen and containing a large number of young cells.

d. Artery cut across.

Fig. I.

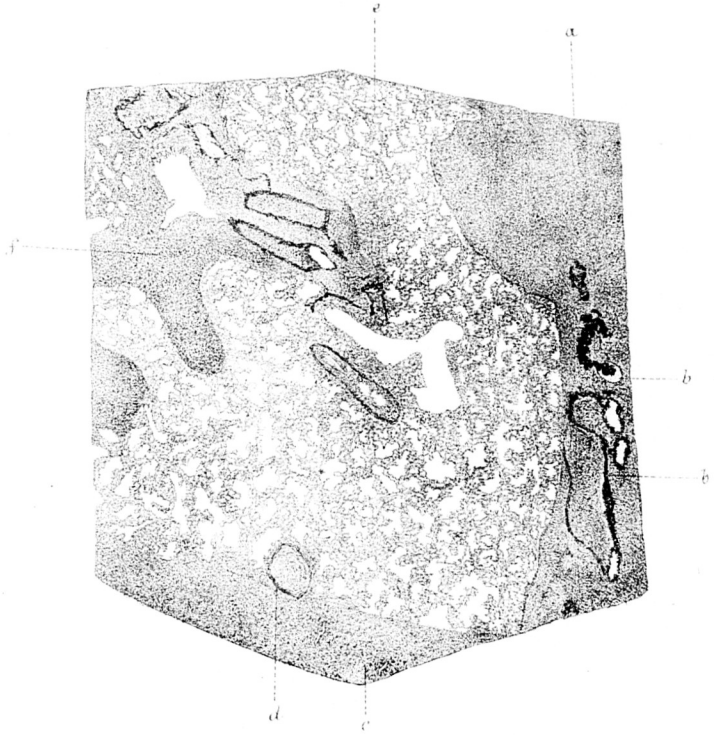


Fig. II.

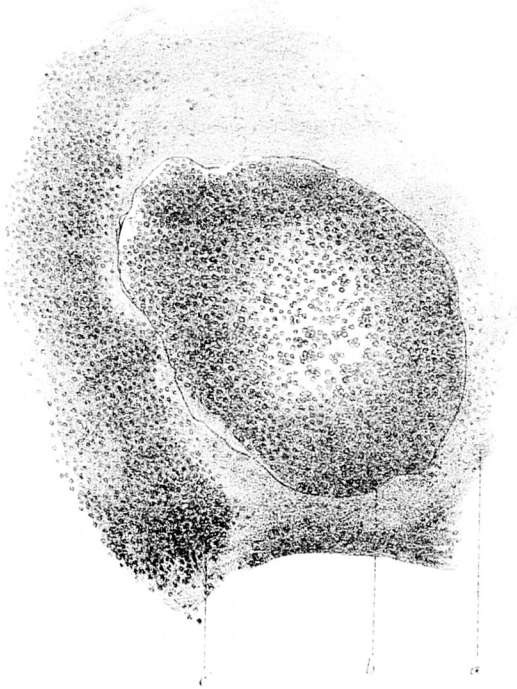
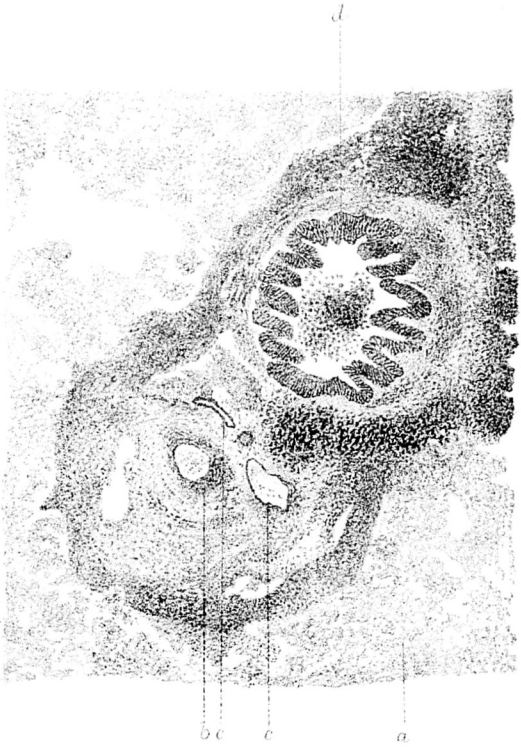


Fig. III.



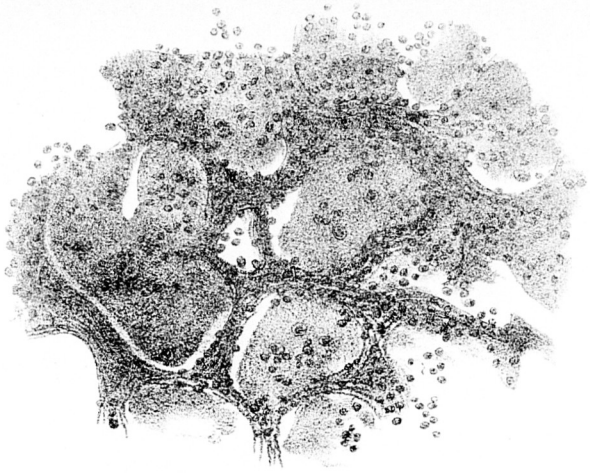


Fig. IV.

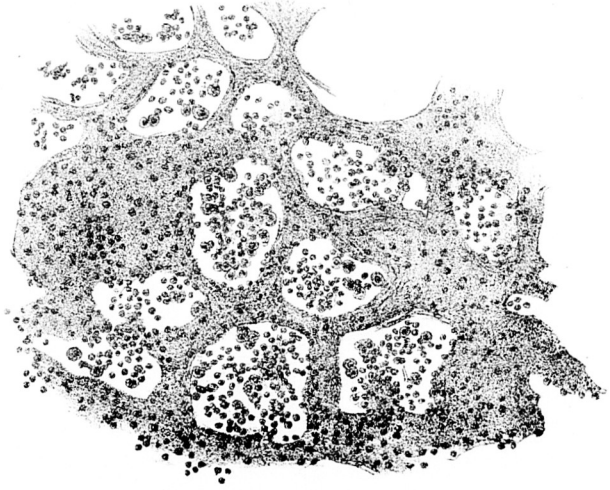


Fig. V.

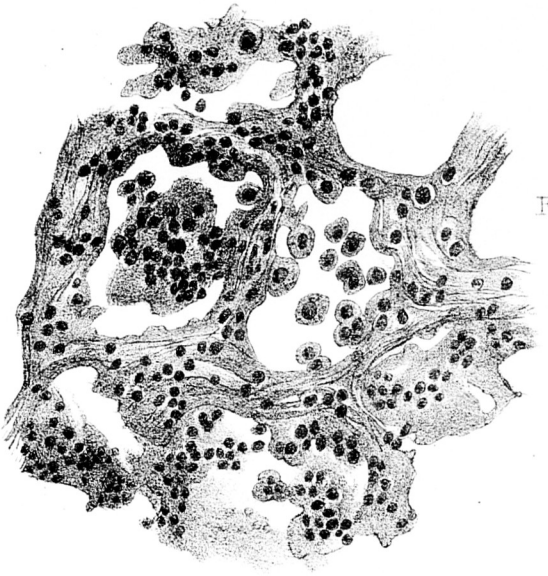


Fig. VI.

Fig. VII.

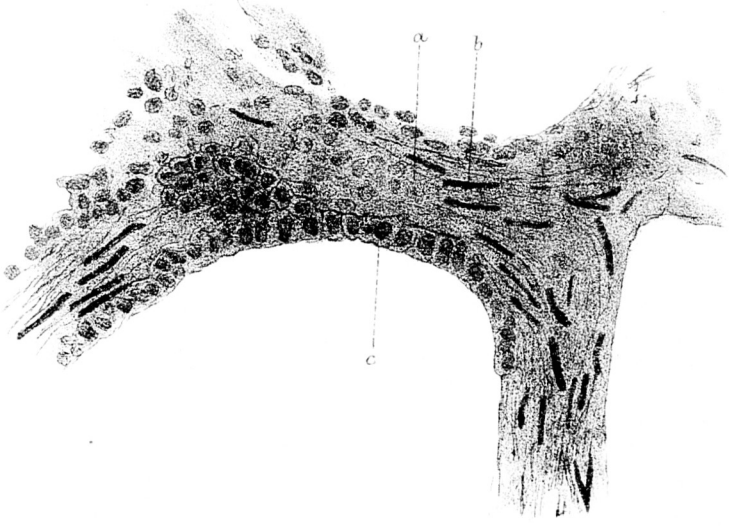


Fig. VIII.

Fig. IX.

Fig. X.