Discussion on the pathology of phthisis pulmonalis.

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DISCUSSION ON THE PATHOLOGY OF PHTHISIS PULMONALIS.*

ON A CASE OF PHTHISIS AB HÆMOPTOE.

By W. T. GAIRDNER, M.D.

Any one who has followed the course of recent researches on tubercular disease, especially those which have got importance and celebrity in Germany from the great name of Virchow, must be aware that many new questions have been raised; and among these are the relations of tubercle to inflammatory processes, which, according to Laennec's views, are secondary to the tubercle, but which some of the Germans are teaching us are primary. That is a very large question, and it is not necessary that it should be entered on at present; but every one who is familiar with the able lectures of Niemeyer on phthisis is aware that he presents this subject in a light to which most of us find it rather difficult to accommodate all our clinical and practical opinions; and in particular, he utterly denies the tubercular nature of a great many of the processes concerned in phthisis pulmonalis; further, he presents the relation of the inflammatory processes in the lung to tubercle in quite an inverted manner to that which Laennec's views involved, and makes it a particular

* This discussion was held in the Pathological and Clinical Society of Glasgow, on the evenings of February 8th and 25th, and March 8th. On the first of these evenings Dr. Gairdner and Dr. Joseph Coats read the papers which follow.

point of his doctrine to support the suggestion implied in the phrase—"Phthisis ab Hæmoptoe" (which was also a very ancient view of the origin of consumption), that the bleeding is the first step and the consumption the second. He tries to bring it into accord with Virchow's views, and thinks that the blood, being poured out into the bronchial tubes, is sucked back into the alveoli of the lungs, or is extravasated directly into the alveoli, and being there caseates, and in this process of caseation gives rise to products which infect the system, and thus originate miliary tuberculosis, which, according to him, is the only true tuberculosis. Now, the pathology of Laennec was precisely the opposite—that the tubercles were there, or at any rate the tubercular tendency was there, and that the blood-vessels of the lung bled as a result of it; thus the hæmoptysis was the result of the tubercular tendency, and therefore took place at various stages of the progress of the disease, and thus the bleeding was usually, if not always, a secondary change, arising from previous tissue changes in the lung or its blood-vessels.

The seeming discordance of these two views gives to every case of apparent "Phthisis ab Hæmoptoe" great interest at present; and though it cannot be presumed that any one case will settle the question, yet the case now before the Society may be considered as presenting some features which were worthy of attention.

The peculiarity of the case is that in a young man two separate considerable hæmorrhages occurred at intervals, with so little disturbance to the system that, but for the alarming character of the hæmorrhages themselves, he would not have been in the Hospital, and would not have taken any medical measures whatever. Of course, only a brief account of the case, which is reported very fully in the *Journals* of the Ward, can be here given.

Patient was a boy of 17, a rivet heater; admitted into the Western Infirmary on 24th Sept., 1877, with no characteristic physiognomy, unless it was a tendency to a florid, or slightly livid complexion, which rather gave one the impression of heart disease than of tubercle, and, in fact, a doubt was entertained as to whether the hæmoptysis was due to the heart or lungs. There had been a hæmoptysis immediately before admission, of uncertain but of considerable amount, and it was found that there had been a preceding hæmoptysis some time before of still greater amount, but which had only temporarily interrupted him in his occupation. The symptoms were almost

nil after the blood had come up; it could even be said that, but for the distinct instructions of the physician, he would not have been in bed. He had no sense of pain or difficulty in breathing; he had absolutely nothing to complain of. But while there was this entire absence of palpable symptoms, there was an extensively diffused crepitant râle on the left side of the chest, chiefly over the lower part, which left no doubt that the hæmoptysis was connected with it. There was also a reduplication of the second sound of the heart over the pulmonary artery, which led either to the inference that there was cardiac disease, or pulmonary disease leading to obstruction of the circulation through the lungs. These signs were singularly persistent; the crepitant râle, loud and distinct over the lower lobe, continued for weeks, during all which time the lad hardly suffered from a single symptom that was worthy of the name. His temperatures also, probably the most delicate physical test of a state in any way allied either to inflammation or tuberculosis, showed singularly little disturbance. They were at first taken only twice a day, and from 25th September to 1st November they rose on one occasion to 101° F., and on one other to 100° F., but with these two exceptions they were almost absolutely normal throughout those three weeks following the hæmorrhage. At a later period they began to show slight oscillation, and on 3rd December a sudden and exceptional rise took place to 102·2° F., and for weeks after that the temperatures were little if at all in excess of the normal, up to the beginning of January; so that for three months of the most careful recording, there were only the most rare exceptions to the general statement as to the temperature being mostly within normal limits. The pulse and respiration were also almost perfectly quiescent. During this time, after the first alarm of the bleeding had been got over, the lad was out of bed, going about the ward, assisting in the work, and making himself useful, and only kept in hospital because it was thought desirable to watch him; the examination of his chest was thus rather thrown into the background. After he had been six weeks in the house a new examination was made. Here it should be stated that on his first admission he was carefully examined every day, and the upper lobe of the lung was adjudged to be perfectly sound, the respiratory murmur over it being even puerile, while the lower lobe presented the crepitus above mentioned, and also dulness on percussion. I must say, therefore, that considerable surprise was

felt on coming back to the physical examination some weeks afterwards, on discovering the metallic sounds characteristic of a considerable excavation over the upper lobe, where immediately after the hæmoptysis the respiratory murmur had been abundant and the percussion good. There was no reasonable doubt of these facts; so that it was perfectly clear to me, that while this lad was walking about the ward with pulse, temperature, and respiration normal, with scarcely any appreciable expectoration, and no pain, a cavity had developed, of size sufficient to produce the most marked physical signs, in the upper part of the lung. Briefly, the rest of the case may be said to be as follows: from this time, by exceedingly slow stages, the patient still making no complaint, hardly a patient at all, going home and coming back again, for he was three times in the Infirmary, the disease gravitated into a case of very chronic ordinary phthisis, and came to be undistinguishable in character from an average case of very slowly developed tuberculosis; and ultimately the lad died.

At the *post-mortem* examination the left lung was found firmly adherent, and an enormous cavity was found in the upper lobe, into which several large bronchial tubes opened; the tissue was condensed throughout in the lower lobe with smaller cavities. The right lung was free from adhesions, and there was no considerable condensation, much less any cavities, but it was dotted over with frequent dark nodules, which the microscope showed to be undoubted miliary tubercles. Miliary tubercles were also found, though not very abundantly, in the liver and spleen. It was certainly a case where one would have been strongly inclined to doubt the tubercular nature of the disease, as observed without the microscope, for it had a good many of the characters of cirrhosis of the lung, but from the microscopic sections from the right lung, some of which are shown by Dr. Coats to-night, there was no doubt as to its tubercular nature. This lung was very adherent. In the right lung there was an entire absence of adhesions.

The tubercular character of the disease ultimately is thus clearly established, and the question is, With what pathology of tubercle does this case best agree? It seems to be one of the cases that comes nearest to the general doctrine of Virchow, the doctrine of a secondary tuberculosis, of a "phthisis ab hæmoptoe," as expounded by Niemeyer; but if it is to be accepted as such, it differs from Niemeyer's description in details, especially as regards the inflammatory changes and symptoms usually following a hæmoptysis. He says, the

blood being in the parenchyma of the lungs or in the alveoli leads first to an inflammation; and that he has often witnessed a development of high fever and pain, with symptoms of inflammation, after such a bleeding as we had in this case. In the patient whose lung is now before us, it would perhaps be too much to say that the bleeding took place without any inflammation; but this at least may be safely said, that whether the tubercular tendency preceded or followed the hæmoptysis, it seems to have come on without anything of the characters of the inflammation as gauged by the symptoms.

It may here be suggested that cases of hæmoptysis like this one are not the only cases in which it is known that blood is poured out into the lung; and it always appeared to me a difficult point to meet, on the modern theory of hæmoptysis giving rise to tubercle, that cases are very numerous where hæmoptysis and also hæmorrhagic condensations take place, but where it is very rare for tubercles to follow. Hæmoptysis as the result of mitral disease, "apoplexy of the lung" as Laennec called it, is the very type of a case where one would expect, on this theory, the blood stagnating in the air cells of the lung to caseate, and thus give rise to secondary or miliary tubercles; and yet it is very rare to find mitral disease associated with tubercular disease. Then, again, hæmorrhage often occurs into other organs or parts in which tubercle never follows, as in bruises, in scurvy, in embolism, even pulmonary embolisms, &c., or in hæmorrhage into the brain, where the rupture of a blood-vessel and the formation and organisation of a blood-clot is often survived for many years, and where caseation hardly ever does result as a consequence, and tubercle is still more rare. Certainly neither inflammation in the ordinary sense of the word, *e. g.*, abscess, nor caseation, nor tubercular meningitis, nor general miliary tuberculosis, can be said to be at all a common result of an old apoplectic clot becoming encysted; and yet, on the theory that extravasated blood *per se* is apt to caseate and give rise to tubercular disease, one would expect tubercle in one form or another to be among the well known and familiar pathological incidents of cases where an apoplectic clot has remained for years, has undergone gradual fatty degeneration, and has by slow degrees become to a great extent removed. So also it is not rare, though not, perhaps, so common, as in the case of the brain, to find hæmorrhagic condensation in the lungs, from cardiac disease or embolism, undergoing a great variety of chronic changes, more or less allied to inflammation, and even to ulcer-

ation and gangrene; but caseation in such cases is almost unknown, and tubercles, whether primary or secondary, are extremely rare, more rare perhaps than in any other kind of death from chronic disease in any organ.

ON PHTHISIS PULMONALIS, ESPECIALLY ITS RELATION TO TUBERCULOSIS.

By JOSEPH COATS, M.D.

In approaching the study of phthisis pulmonalis we have two things to consider, in the first place the exact nature of the anatomical changes, and in the second place what may be called the proper pathology of the disease. At the very outset of the inquiry we are met with the question, Is phthisis pulmonalis a tubercular disease? and this leads to the further questions, What are the characteristics of tubercular disease? How are we to recognise tuberculosis?

In order to answer these questions, we must take, to begin with, undoubted cases of tubercular disease, and endeavour from them to find what are the essentials in their pathology. For this purpose we shall take, in the first place, acute general tuberculosis, and afterwards an undoubted case of local tuberculosis, and endeavour by the comparison of these two to determine the essential features of tuberculosis.

The General Pathology of Tuberculosis.

In acute miliary tuberculosis we have a disease running its course in a few weeks with high fever, and leading to a fatal issue apparently in very much the same way as a case of typhoid fever, either uncomplicated or complicated only with pulmonary catarrh. According to my experience, in a large proportion of cases this disease is mistaken for typhoid fever during life. On examining the body a condition is found which can, I think, be fairly designated an eruption. Myriads of little grey bodies are found in the most diverse organs—in the lungs, kidneys, liver, spleen, sometimes in the muscular tissue of the heart, in the membranes of the brain, and elsewhere. An eruption has occurred, apparently simultaneously, of what in the meantime we may designate miliary tubercles, in all these organs. The eruption is perfectly symmetrical,

both lungs, both kidneys, both halves of the membranes of the brain, &c., are equally affected. In the individual organ also the tubercles are planted uniformly in every region of it. In the lung, for instance, there is no localization at the apex or elsewhere, but from apex to base there is a homogeneous distribution of the nodules. So is it in the liver; the tubercles here are mostly too small to be visible to the naked eye, but making a microscopic section of any part of the organ one is certain to meet with them.

Now a lesion which has thus a symmetrical distribution, which occurs simultaneously in a great variety of organs, and which presents all the characters of an eruption, must be due to the presence of some poisonous agent in the blood. This is confirmed by the clinical characters of the disease, which are not entirely referrible to the local conditions. In a recent debate on syphilis in the Pathological Society of London, Jonathan Hutchinson insisted on the view that a symmetrical disease is a blood disease, and adduced the authority of Paget and Budd in support of this view. In the present case it seems impossible to escape the conclusion that there is some virus carried by the blood to these various organs and producing the lesions there.

It may be important here to refer to the histological characters of this lesion, the miliary tubercle. The structure is virtually identical in all situations. In the case of the lung, for instance, as appears in the sections which I have placed under the microscopes, the tubercles are seen as rounded solid tumours in the midst of perfectly vesicular tissue. So in the liver there are rounded bodies appearing in the midst of the hepatic tissue. The tubercles are situated in the connective tissue of the organs. In their finer structure they present giant cells in their central parts, and these giant cells contain multitudinous nuclei, largely distributed towards their margin. At their peripheral parts the giant cells present processes which form a reticulum, in the meshes of which are smaller cells, some of them epithelioid in size and appearance, some of them with the ordinary characters of small round cells. These characters are often obscure in their finer details on account of degenerations and the complications about to be referred to.

While these small round tumours, the true tubercles, are present in the various organs mentioned, they are not the only pathological condition. It is important to observe that in almost every case there are evidences of concomitant inflammation. In the case of the lung the inflammation manifests itself in the form of a catarrhal exudation in the air vesicles,

and also by an inflammatory infiltration of the connective tissue of the lung. Around each tubercle there is commonly an inflammatory zone, and the tubercle as seen by the naked eye includes inflammatory products along with the true tubercular growth. In the case of the pia mater the inflammatory manifestations are highly developed, and the tubercles are so concealed by the inflammatory exudation as to be usually rather difficult of detection by an inexperienced person. An important question comes up here as to the relation of the inflammation to the tubercles. Is the inflammation produced by and secondary to the tubercles, or are they both due to the same poisonous agent? This question will come up afterwards and need not be fully dealt with here.

Turning now to a case of local tuberculosis, we have to consider what the exact pathological processes are in an undoubted case of this kind. Local tuberculosis of the kidney, sometimes called phthisis renalis, affords a good example. In this disease there is, let us say, first in a single calex, a tuberculosis of the apex of one of the pyramids of the kidney, that is to say, tubercles are formed in this situation. These tubercles, of essentially the same structure as those in acute miliary tuberculosis, have a tendency like most tubercles to undergo caseous metamorphosis. Caseous metamorphosis means death or necrosis of structures, and here as the caseous material is at the surface, it is carried away and an irregular ulcer forms. This ulceration increases by a repetition of the same process. Tubercles are formed in successive crops outside the ulcer in the neighbouring kidney tissue, and the ulceration spreads by the successive disintegration of the tubercles and tissue involved. But this disease advances not only in this direction. It progresses down the ureter, causing ulceration of its mucous membrane, the caseous material sometimes forming a continuous layer on the surface of the prolonged ulcer. In this way the entire ureter may be converted into an ulcerated tube with a caseous lining. The bladder is also affected, and we have again ulcers produced by the formation of tubercles in the mucous membrane, and extending by the formation of successive tubercles peripherally, and their disintegration. The formation of tubercular ulcers may extend to the other ureter, but the process apparently finds some difficulty in ascending against the current of the urine, but it may spread to the vesiculæ seminales and vasa defferentia.

Now in such a case as this we have a local disease presenting characters exactly parallel to what we find in acute general tuberculosis. We have here a virus not conveyed by the

blood, but carried, on the one hand, inward, possibly by the lymphatics, and on the other hand, along a surface, and producing as its result the formation of these minute bodies, virtually identical with the miliary tubercles of the other disease. It is true that here the tubercles are variously altered; they are in different stages of caseation and disintegration, and not in the fresh and nearly uniform condition of those which are virtually of simultaneous production. Yet they are the same rounded bodies with the giant cells in their central parts and smaller cells peripherally, and it need hardly be said that even in acute miliary tuberculosis a caseous necrosis is always more or less present.

Here, again, the tubercles are accompanied by inflammatory manifestations. In such a ureter as that I am referring to—and I have had in my mind a particular case, from which I show you a piece of the ureter—there is great inflammatory thickening, and the tubercles are in great part buried in the midst of inflammatory cells, so that their boundaries are obscured, and they are mainly recognisable by their giant cells.

It may here be remarked that the tubercles themselves, in their structure, present analogies to inflammatory products. In inflammation we have round cells like those of the periphery; we have larger epithelioid cells in granulation tissue, and even the presence of giant cells is not unknown in granulations. Tubercles present analogies to inflammatory products in another respect. It is well known that inflammatory new-formations tend to form connective tissue; the natural termination of the granulating wound is the cicatrix, a connective tissue structure. As we have already seen, tubercles frequently have a tendency to undergo caseous metamorphosis, but in the lungs they often become converted into solid fibrous bodies, forming a dense non-vascular glistening connective tissue. Although in some respects analogous to inflammatory structures, tubercles are not to be regarded as simply inflammatory, any more than syphilitic gummata are to be considered inflammatory, although presenting at least as much analogy in structure to inflammatory products.

It need hardly be added that the same question arises here as in the former case in regard to the exact relations of the inflammation. We can only say in the meantime that the formation of tubercles and the inflammatory new-formation are again concomitant.

So far, then, it is apparent that in a typical case of local tuberculosis we have an "infective" disease. I have long been

in the habit of using this word as equivalent to the German word "infectiv," and as bearing a distinctly different meaning to infectious; and I was glad to notice a few years ago that Dr. Burdon Sanderson has arrived at a similar use of the word infective. An infective process is one in which lesions are to be traced to the direct action of a virulent agent, whether that agent is introduced from without or formed inside the body. Acute miliary tuberculosis is an infective disease because each miliary tubercle owes its origin to the action of an infective particle. And so local tuberculosis is infective, because we have again evidence of some infective material acting directly on structures, and producing the particular form of lesion called a tubercle.

The Anatomical Conditions in Phthisis Pulmonalis.

Leaving now these preliminary observations which I have thought necessary in order to clear the ground, we turn to the more specific subject of discussion—namely, the Pathology of Phthisis Pulmonalis.

If we define phthisis pulmonalis as an emaciating disease involving destruction of the lung-tissue, our first task should be to determine what are the exact anatomical conditions which lead up to the formation of cavities in the lung, the formation of cavities being perhaps equivalent to the destruction of lung tissue, and forming, let us say, the criterion of the disease.

There are undoubtedly two distinct forms of the disease, and two modes in which cavities may form. It is not asserted that these two forms are absolutely separated in the actual case, but on the whole, we have two groups and two distinct anatomical conditions.

In one of these forms, and perhaps the commoner, the disease, in its purely anatomical aspects, is to a great extent, but not entirely, a catarrhal inflammation of the lung. Beginning as a rule in a series of finer bronchi, it extends to the lung-alveoli. The inflammation here manifests itself by the filling up of the lung-alveoli with large round cells—the derivatives of the alveolar epithelium. The physical result of this is that the portions of lung concerned are deprived of air; they are solidified, and these solidified pieces often, by their mere shape, suggest the form and arrangement of the ultimate lobules of the lung. They are grey in colour, and sometimes slightly pigmented. The stroma of the lung is also involved, and it is generally difficult to disentangle the various

elements. The products accumulated in the air vesicles after a time present a peculiar change. They undergo caseous metamorphosis, and not only they, but simultaneously the portion of lung tissue involved in the condensation. The significance of this process has not, I think, been sufficiently elucidated. In caseous metamorphosis there is not merely the drying in and fatty degeneration of inflammatory products but there is the actual death not only of these products, but of the piece of lung in which they are contained, so that the process warrants the name of caseous necrosis. If you examine a microscopic section of a piece of lung in which this process has occurred, you find simply a homogeneously granular appearance, in which you may be able vaguely to make out the outlines of the air vesicles, but very often not even that, the whole tissue being involved in an indiscriminate necrosis. We are here more immediately concerned with the anatomical details, so I do not pause to discuss fully the pathology of this process; but I would in the meantime emphasize the fact that sometimes this caseous necrosis is almost an acute process. Sometimes the disease runs rapidly on through the preliminary stage of condensation to that of caseous necrosis, without there being almost time for drying in to occur, and the cheesy material formed is rather a soft moist cheese. It is customary to describe inflammations having a tendency to caseous metamorphosis as scrofulous, the inflammatory products in these cases having a peculiar tendency to retrograde changes, but no mere degenerative tendency on the part of the products of inflammation will explain the marked necrosis of these products and of the lung tissue besides.

The caseous necrosis having occurred, the products may lie long unchanged, and may even remain as an obsolete piece of tissue, becoming encapsuled like a foreign body, then partly absorbed and partly impregnated with lime salts. More commonly, however, the caseous material after a time softens. It may be, as Hamilton suggests, that this softening is akin to the chemical change involved in the "ripening" of cheese, in which, according to M. Duclaux, "the main decomposition which takes place is that certain of the albuminoids, insoluble in water, become soluble." However this may be, the caseous material softens and breaks down, forming a cavity. If the piece of tissue be small, then a small cavity is the result, but probably neighbouring portions of the lung have been similarly affected, and the cavities increase in size by coalescence of several. Doubtless, also, similar processes are occurring successively around the forming cavity and so increasing its size.

In the other form of phthisis the anatomical conditions are very different from these, and we have an example of it in the case which was the immediate occasion of this discussion, and which I again bring before the Society. The condition which is most pronounced here is that commonly designated cirrhosis of the lungs, and in its more immediate anatomical features it consists in a new formation of connective tissue in the lung, with the usual contraction of the connective tissue—it is in fact an interstitial inflammation of the lung.

We may take the description of the anatomical conditions in this case as indicating the usual appearances in an advanced stage of this disease. The report book contains the following account of the state of the lungs and heart:—

"There is great shrinking of the left lung, so that the mediastinum is drawn greatly to the left, and the edge of the right lung passes in some parts fully an inch and a half to the left of the middle line, and the heart is drawn considerably to the left.

"The heart is moderate in size, but the right ventricle is considerably enlarged, forming the apex of the heart. The valves are normal, but the tricuspid orifice admits four fingers.

"The right lung is adherent almost throughout, but especially over the upper lobe. Here the adhesion is exceedingly firm, and the coalesced layers of pleura are greatly thickened. In this upper lobe there are numerous cavities, all of them smooth walled and with more or less of the sacculated form, without any distinct projecting trabeculæ. These cavities are sometimes quite distinctly in the form of bulbous dilatations of the bronchi, and are always directly continuous with one or more bronchi. [On examining the walls of the cavities there is found a great preponderance of inflammatory tissue, but with ciliated epithelium in abundance. The presence of this ciliated epithelium is determined in some of the most typical of the cavities.] Outside the cavities the upper lobe presents dense pigmented connective tissue, and there is not a trace in this lobe of normal air vesicles, nor is there any caseous material present in any part.

"The left lung is firmly adherent throughout, there being the same excessive adhesion over the upper lobe as in the other lung, and a still greater thickening of the pleura, which here reaches about half an inch in thickness. In the upper lobe there are also cavities distinctly bronchiectatic and dense pigmented tissue outside them. It is noted that the pigmentation does not pass into the thickened pleura, but stops short by an abrupt line at the sub-pleural tissue.

"It is to be noted that in both lungs the lower lobe is almost free from cavities, but there are isolated patches of condensation in the form of groups of nodular projections. Examined roughly in the fresh state, these are found to present rounded bodies supposed to be tubercles and containing distinct giant cells."

There are now one or two points which must be considered here in relation to the more immediate anatomical conditions. Hamilton has recently in the *Practitioner* given an account of the processes concerned in this disease, with much of which I am in full accordance; but there are certain parts of this account with which I cannot agree, and while taking advantage of his excellent descriptions, I do not rigidly follow his account of the processes.

There are three principal situations in which connective tissue is present in the lung—namely, under the pleura, between the lobules, and around the bronchi, so that we may speak of sub-pleural, interlobular, and peribronchial connective tissue. These are all in intimate connection by means of the lymphatics, which communicate so thoroughly throughout these parts that the connective tissue in these three situations may be regarded as one. As evidence of this, Hamilton has shown that foreign material, such as dust inhaled, if it finds its way into the peribronchial connective tissue, is carried about in the lung and deposited in all three situations.

In interstitial pneumonia there is great new-formation of connective tissue, in the usual fashion of productive inflammation, in all these three situations. Just as in the liver the inflammatory new-formation occurs where there is existing connective tissue, the so-called capsule of Glisson, so here the inflammation produces primarily a thickening of the existing connective tissue. There is great thickening of the sub-pleural and pleural connective tissue, great peribronchitic new-formation, and the interlobular connective tissue is converted into thicker bands. But the inflammation does not confine itself to these structures, and the walls of the lung alveoli are invaded, and become thickened. The bronchial mucous membrane, on the other hand, becomes the seat of catarrhal inflammatory changes, and forms a muco-purulent discharge.

The new-formed connective tissue, like other inflammatory connective tissue—like the cicatrix—has a marked tendency to contract, and the flattenning of the chest which is so marked in this disease is a result of this. By the formation of the connective tissue and its contraction there is great

destruction of the proper vesicular tissue of the lung, and in this way masses of firm tissue come to occupy the place of lung tissue. The dragging in of the chest wall does not by any means fully compensate for the great shrinking which occurs, and there are two conditions which must be taken into account in this connection—namely, emphysema and the formation of cavities.

Localised emphysema or dilatation of the air vesicles is of frequent occurrence. It is an emphysema which has no special localization, but in a section of the lung, areas of emphysema will alternate with tracts in which there is nothing but connective tissue. In the emphysematous parts the walls of the dilated air vesicles are frequently thickened. There is no difficulty in explaining this emphysema; it is simply compensatory to the shrinking of the lung tissue so frequently referred to already.

The formation of the cavities is a more complicated process, but it may be said that in this disease dilatation of the bronchi is the essential factor in the formation of the cavities. To a certain extent the bronchiectasis is compensatory like the emphysema. It is to be remembered that the bronchi are no longer in their normal condition. The mucous membrane is infiltrated with inflammatory cells and softened; the peribronchitic tissue is also inflamed, and except where there is a concentric contraction, it may be expected to yield readily. Then, as has been so well shown by Hamilton, there are agents at work which often have a direct effect in pulling the bronchial wall outwards. We have seen that the new-formation of connective tissue is mainly around the bronchi, in the interlobular septa and pleura. The contraction of this connective tissue has often a direct effect on the bronchial wall. The pleura is fixed to the chest wall by firm adhesions, and the contraction acts on the chest wall, dragging it inwards. But it also acts on the bronchial wall, dragging it outwards, the chest wall and the bronchial wall being thus mutually approximated.

But there is another method by which bronchial dilatation occurs, and which I believe is often at the origin of the cavities. In the case under discussion, I found this method perfectly illlustrated. The inflammation of the connective tissue in this disease generally begins in the peribronchial tissue; it is a peribronchitis. It is not, however, a general peribronchitis, but the lesion occurs in a number of isolated spots, resulting in a series of local thickenings often pigmented, so that, in an early stage of the disease, the lung

may be studded with dark grey nodules which are mainly peribronchitic. The new formed connective tissue, by its contraction, narrows the calibre of the bronchus, and may even obliterate it. Behind this obstruction the secretion stagnates, and there is a progressive dilatation in which the conditions already alluded to take part. Some of these small cavities often look as if they were completely cut off from all connection with the bronchi. In the lung under consideration I was at first puzzled with one such, and it was by carefully following up a recess of it that I found an undoubted occlusion of the tube. The walls of such cavities produce a muco-purulent material which fills them.

Cavities of large size form by the dilatation of the bronchi, but they can always be distinguished as bronchiectatic by considering their frequently sacculated form, the continuity of their lining membrane with that of the bronchi, and the nature of the process around them. There is no evidence around them of breaking down of lung tissue but of new formation of connective tissue. Examined microscopically the walls of the cavities are very different from those of mucous membranes, but epithelial elements are still to be recognised abundantly. The proper wall of the cavity is, in fact, formed of altered epithelium with occasional groups of ciliated columnar cells. In the case shown, it was only necessary to scrape the internal surface of one of these large cavities to obtain numerous ciliated epithelium cells.

The Relation of Phthisis Pulmonalis to Tuberculosis.

Having now, as fully as the time at my disposal would admit, described the anatomical processes concerned in these two forms of phthisis pulmonalis, we have to consider what relation these conditions have to tuberculosis. Is tuberculosis associated with these processes, and in what way? Few will deny that tubercles *may* be present in both these forms, but the relation which tuberculosis bears to the essential process is matter of discussion. We must rigidly keep to our original ideas of tuberculosis. It is an infective disease in which certain histological structures called tubercles are developed along with accompanying inflammatory products.

That phthisis pulmonalis may possess an infective nature is demonstrable in almost every case of catarrhal, or perhaps more correctly, caseous phthisis in which the disease has gone on to the formation of cavities. I make it a point to examine the mucous membrane of the bron-

chial tubes in such cases, and, if the cavities are of any considerable standing, there is almost, without exception, tubercular ulceration of the mucous membrane. The mucous membrane near the cavities is usually almost continuously ulcerated, but, on passing downwards, isolated ulcers, obviously tubercular in character, are found, and even isolated tubercles. The tuberculosis of the mucous membrane often extends to the main bronchus, and even to the trachea, and I have seen deep ulceration of the trachea even exposing the cartilages in such cases. The tuberculosis is also accompanied by an acute inflammation of the mucous membrane, which I have seen even coated with an inflammatory false membrane. Again, it is in cases of large cavities, due to breaking down of the lung tissue, that ulcers form in the intestines. In a case which occurred a few days ago, the cavities were recent, and we had just the beginnings of tuberculosis of the intestine, in the form of nodules, many of them caseous, in the closed follicles, and in one or two cases the formation of a small crater-shaped ulcer. I believe that in the broken down lung tissue the tubercular virus is carried to the mucous membrane of the bronchial tubes, and is swallowed. In this way it is brought into direct contact with the mucous membranes, and produces its usual effects. I do not usually find any tubercular ulceration of the mucous membrane of the bronchial tubes or tuberculosis of the intestine in cirrhosis, or in connection with bronchiectatic cavities.

But in cirrhosis of the lung there is equally good evidence of the presence of the tubercular virus. In the case which Dr. Gairdner has just described, I found abundant miliary tubercles in that lung which is described as the sound one (as compared with the other), and I have placed sections under the microscopes which show this quite unequivocally. I found also undoubted tubercles in the liver and spleen, although they were not so numerous as in the usual cases of acute miliary tuberculosis. In this case then there is evidence of tuberculosis, the virus having even got into the blood, and infected the other lung throughout, as well as the liver and spleen.

No one will deny that tubercles often occur in the course of phthisis pulmonalis, but the important questions remain, Is tuberculosis an integral part of the processes in ordinary cases of phthisis? Or is it always secondary, as we may suppose the ulceration of the bronchial mucous membrane and the ulcers of the intestine to be? On this subject the views of different observers diverge, and it will be necessary to enter now on debateable ground.

It may be stated at the outset that there is no doubt that some cases of cirrhosis of the lung occur, and run their course without any suspicion of tuberculosis. I have here the lungs from a case of so-called Potter's phthisis, in which, apparently from the irritation of dust inhaled, there has been a great new-formation of dense pigmented connective tissue. I have also seen a case in which there seemed reason to believe that syphilis was the cause of the cirrhosis, what I took to be gummata existing in the midst of the new-formed connective tissue.

But in cases where tubercles are actually present, and present in the parenchyma of the lungs, whether in the one form of phthisis or the other, a difference of view exists as to their exact relation to the pathology of the disease. Those who hold the divergent views are agreed that the appearance of these tubercles is evidence of the existence of an infective process, the presence of a virus or ferment, so that in the case of both, the views are strictly consistent with what we have set down as the criterion of tuberculosis. But on the one hand it is asserted that *the tuberculosis is secondary to the inflammatory process, and the ferment or virus is produced by the caseous material*, while on the other hand it is believed that *the whole process is a tubercular one from the first, and that inflammatory products and tubercles are equally the result of the irritation of the tubercular virus.*

The most recent available exposition of the former view, is that given by Dr. Hamilton in his papers in the *Practitioner*. He states in the most definite way, "Where tubercle occurs in the lung, or in any other organ, it is always preceded by a caseous source of infection," and he even uses the expression "caseous virus," evidently in the belief that in the caseous material a process of chemical decomposition occurs, resulting in the formation of a ferment. Now, there is much that is enticing in this view, because, especially, it seems to dissociate two things which appear at first sight quite distinct, namely, the ordinary inflammatory lesions and the tubercular. There is no doubt that a virus does exist in caseous material of a phthisical lung, and the virus is conveyed along the mucous membrane of the bronchi, and also by the lymphatics of the lung, producing tuberculosis in the one situation or the other.

For myself I may say that I originally approached the subject of phthisis pulmonalis strongly imbued with this view of its pathology. I felt it to be necessary to keep clearly in view the characteristics of tuberculosis as exhibited in general and local tuberculosis; and Dr. Gairdner knows that I have

somewhat persistently refused to call anything tubercular which might by any chance be simply inflammatory, and have usually answered, "I don't know" to his question, "Is this tubercle?" when put at the *post-mortem* table before microscopic examination.

I have examined lungs in phthisis with the definite object of finding a non-tubercular catarrhal process, but I have been driven to the conclusion that *in all cases of caseous phthisis tubercles are inseparably mixed up in the morbid process.* I do not say that tubercles are invariably to be found in every microscopic section of a caseating piece of lung, but there are few exceptions, and these can be explained on the principles to be presently referred to. It is the same with cirrhotic phthisis. Here also, it seems to me, tubercles are inextricably mixed up in the process, and as in the case I have brought before you to-night they may be present without any caseous material being discovered after diligent search.

Take this lung, which was removed from a case examined *post-mortem* yesterday, as one example out of many. You see that the central parts of the lung—namely, the lower part of the upper lobe, and the upper part of the lower lobe, are the seat of an extensive "frog-spawn like" condensation. The disease is obviously advancing, and more and more of the lung is getting involved in a process which, beginning in what appears to be a simple condensation, soon passes on into a caseous condition. There are some signs of softening in the caseous material, but no cavity has formed, and even in the other lung, which was much more extensively involved, only one small cavity was discovered. Here is, obviously, a comparatively acute process of a progressive character, and microscopic examination shows that it is a tubercular process, or at least a process in which the formation of miliary tubercles is inextricably involved. The whole process in this lung seems to me a homogeneous one, and it happens that the patient has died at a period when it is nowhere far advanced. Are we to suppose that, at a still earlier period, there was a simple catarrhal pneumonia leading on to caseation and producing tubercles secondarily? When we see a process which is a consistent whole, and in that whole a particular element is an integral part, there must be strong evidence to convince us that that element is a secondary one. It seems to me much more reasonable in this case to suppose a virus acting on the lung tissue and inducing both inflammatory lesions and tubercles. The virus is self-propagating, and so the disease has a continuous tendency to spread.

I would here recur to the question already incidentally referred to of caseous necrosis. It is usual to explain this process by the occlusion of the alveolar capillaries from the pressure of the accumulated catarrhal products in the alveoli. Now, I cannot accept this explanation as satisfactory. The uniformity with which this process occurs, the manner in which it extends from definite centres, and the fact that lung tissue and catarrhal products die simultaneously, seem to me to be inconsistent with this view. We know that occlusion of vessels will produce a process essentially similar, as we often see a cheesy looking wedge in the kidney from embolism of the renal artery. Here there is a sudden deprivation of blood, and the condition may pass directly into that of the pale infarction; which is a necrosis in many ways similar to caseous necrosis. In the case of the lungs it is difficult to believe that a pressure from within the alveoli is sufficient to obstruct the vessels, not only of the alveolar wall, but also of the interlobular connective tissue, which becomes involved in the caseous necrosis. We are to remember that the material for the formation of the catarrhal products is obtainable only from the blood-vessels, and it seems inconsistent to suppose that these products will go on increasing till they finally occlude their own source of supply. It seems to me more reasonable to suppose that an agent acts on the tissues producing the catarrhal processes and the tendency to necrosis, the capillary circulation being impossible when the damage to the tissue has reached a certain degree.

Turning now to the cirrhotic form of phthisis, I have carefully examined in various cases what I took to be the parts in which the disease was in its earlier stages. In the case before us I chose the isolated hard nodules which exist apart from the general mass. Here the process consists in its more general aspects in a new-formation of connective tissue, with a marked tendency to contraction such as we see in other cases of cirrhosis. But *wherever there is cirrhosis there are tubercles, and wherever there are tubercles there is cirrhosis.* The two processes are indissolubly connected the one with the other, and the tubercles take part in the transformation into connective tissue, becoming converted into hard dense nodules. Here then we see again tuberculosis and inflammation associated, and in the present case as well as in others I could see no source of caseous infection.

It will be seen, then, that in both forms of phthisis the inflammatory process and the tuberculosis are concomitant, and though the inflammatory products may be in the anatom-

ical relations the more prominent, yet they are no more predominant in the lung than are the inflammatory products in tubercular meningitis, and no one hesitates about calling that a tubercular disease. I think we are forced to the conclusion that tubercles and inflammatory products are both the result of the irritation of the tubercular virus, and it is even conceivable that the inflammatory processes might be produced without the miliary tubercles.

It may here be asked, How is it that the tubercular virus produces two such various lesions as catarrh with caseous necrosis and interstitial inflammation? This is a question which it is very difficult to answer. We can only say that the tubercles themselves show very different tendencies in the two conditions. In the catarrhal form the tubercles undergo caseous metamorphosis in common with the inflammatory products. In the cirrhotic form they undergo fibrous transformation in common with the inflammatory new-formation. In these respects it will be observed the tubercles again show their affinity to inflammation.

It may here be noted that tubercles, either by caseous necrosis or fibrous transformation, tend to become obsolete. The individual tubercle becomes obsolete, but the virus is obviously self-propagating, and the tendency is to the production of fresh crops of tubercles and fresh inflammatory lesions; but even the virus may cease to be produced, and in that case the disease will undergo a spontaneous cure, the tubercles naturally becoming obsolete. We know that calcareous or pultaceous material is often found at the apices of lungs surrounded by cicatrices while the rest of the lung is normal.

In conclusion, it may, I think, be said that in tuberculosis we have an infective process. In general tuberculosis this manifests itself in a general infection of the system, the infective material being conveyed by the blood. In local tuberculosis the infective material is obviously self-propagating, so that the lesions extend from the original focus to neighbouring parts. In phthisis pulmonalis we have again an infective process, the lesions extending as a general rule continuously from one part of the lung to another. These lesions are in their general features inflammatory, but when examined in detail there are evidences, in the histological structure, of tuberculosis, and even the inflammatory lesions are probably the effect of the tubercular infection. Caseous necrosis is of frequent occurrence, involving tubercles, inflammatory products, and lung tissue, and the occurrence of this probably points to

the existence of some particularly virulent agent, and is not explicable on the view of a mere secondary degeneration of inflammatory products.

The question of the source of the virus has not been entered on here, mainly because it has been my desire to keep within the bounds of matters of which I am personally cognisant. It is clear, however, that all persons and all tissues are not equally susceptible to the virus, just as all persons are not equally susceptible to the viri of typhus, or any infectious fever. It is more for clinical observers to determine under what circumstances the virus is introduced, and what determines the susceptibility of the individual.

Adjourned Discussion—*25th February.*

Dr. Foulis remarked that, in the brief time at his disposal, it was not possible to give a view of the whole of so complicated a subject as tuberculosis and its relation to phthisis, and he would content himself with referring to a few points. One of these was the infectious nature of tuberculosis, which, since the time when Villemin produced a generalised tuberculosis in the guinea pig, by injecting into the peritoneal cavity tubercular matter, had been the subject of much experiment. The result of this experiment was to show that the disease could be introduced into the bodies of animals in various ways. Chauveau succeeded in infecting animals by feeding them with food mixed with tubercular tissue. Cohnheim, by introducing particles of tubercular tissue into the anterior chamber of the eye, induced, after 20 to 30 days, a local tuberculosis of the iris, which then spread to the rest of the body. And Tappeiner, causing dogs to inhale for a certain time air in which pulverised tubercular tissue was suspended, induced in them a tuberculosis of the lungs, and afterwards of the other organs. It was noteworthy that his experiments, when repeated with pulverised calf's brains, and with matter from scrofulous glands of the neck, gave negative results. All these experiments showed that the tubercular taint could be introduced into the body by different paths, but that once in, it spread all over it like any other virus. In the case of man the facts pointed in the same direction; and there were cases which pointed strongly to the transmission of the disease from the lower animals to man, *e.g.*, from the cow. On the table were the parts from the body of a boy æt. 3½ years, who had died after a comparatively short and sudden illness, in

which the first symptoms were abdominal swelling and sickness, followed by cough. On *post-mortem* examination, the peritoneum was found studded over with yellowish-white nodules in immense numbers; some very minute; others larger, up to the size of a pea and over, while in places there were masses of the same firm, yellowish-white tissue. These nodules all presented the same aspect, and even the most minute were yellow and opaque, and surrounded by a zone of congestion. The mesenteric glands were very large, firm, and yellowish-white; the bronchial glands similarly affected, but to a lesser extent; there was a yellow nodule half an inch in diameter in the wall of the left ventricle of the heart; another in the left suprarenal capsule; and several in the superficial layers of the liver. With this there was evidence of pleurisy on both sides; and at the base of each lung an area of greyish-red solid pneumonic tissue, much larger in the left lung than in the right. It was noticeable that in the middle of the left lung the lung tissue was solid, but œdematous, and that in this œdematous area were two or three small irregular cavities opening into bronchi, and full of yellow purulent fluid. In the apex there were several groups of more translucent greyish-white tubercles amid a reddish and crepitant lung tissue. In the ileum, a single small ulcer, on the peritoneal area of which was a group of minute grey miliary tubercles. Allowing now for the absence of direct and detailed proof, there was even in this case such a degree of resemblance to the Perlsucht disease of the cow as to suggest the idea of a direct communication, perhaps by means of the milk as described by Gerlach. It might be said that the caseous material was the original starting point of the disease, but it was not enough to say so; for how many cases were there not in which caseous masses failed to infect the system, while on the other hand it could hardly be denied that there occurred instances of acute tuberculosis where a minute inspection of the body failed to reveal the existence of caseous masses. There must therefore be a specially infectious character in the tubercular material, whereby it played the part of a particulate virus, whose particles lodged in the various organs, and there gave rise to tubercles. This was not mere mechanical irritation, for in the cases of injection of powdered cork or cinnabar into the peritoneum, there was indeed a local eruption of miliary tubercle-like nodules, but there was no general infection of the system; and so in the lungs the miliary nodules which were caused by dust particles were limited to the lungs, and did not spread further. What, then, was the peculiarity of the

infectious miliary tubercle? Attempts had been made, by Langhans and others, to find a pathognomonic feature in the *giant cell,* so often found in tubercle. But the existence of giant cells in many widely differing morbid structures rather interfered with this view; and besides this, there was a growing belief among a certain class of pathologists that the giant cell in tubercle was often not a cell at all, but a section of a vessel, either lymphatic or blood-vessel, distorted, distended, and filled with granular *debris* and the nuclei of its own endothelium. If this view were correct, it chimed in with the theory of a particulate virus in tubercle, for it was easy to follow the theory of impaction of the virulent particle in the lymphatic or blood capillary, and the formation of the tubercle around that spot. The giant cell being disallowed as a pathognomonic feature in tubercle, nothing remained which the microscopic examination at present was capable of revealing; and, therefore, Cohnheim had fallen back on the impracticable dictum, that only by inoculation experiments in suitable animals can we finally affirm whether a particular tubercle be of the true infectious sort or no.

As to the share taken by tubercle in the formation of cavities in the lungs, and of ulceration in the bowels and kidneys, that depended on the amount of additional irritation in these organs, whereby a large surplus cell growth was set up, in consequence of which there was a more ready breaking up and loss of tissue. In parts of the body away from contact with air, or urine, or fæces, the tubercle did not break up in this way, but was, if the patient lived, either removed by absorption or underwent fibrosis, and became harmless. Tubercle was only one cause of lung cavities, which could sometimes be traced to bronchiectasis, in which case they were of very limited extent, or to loss of tissue in pneumonia; but it was certain that cavities did sometimes take their origin in softened and broken down true miliary tubercle.

Mr. D. J. Hamilton (Edinburgh) said—The first duty I have to perform, Mr. President and gentlemen, is to thank you for inviting me to come to hear and to take part in the discussion of this evening. The subject is one which is full of interest both for the pathologist and for the physician, and the value of having clear ideas concerning it cannot be overestimated.

It seems to me that, in order to start any discussion upon the subject of tubercle and phthisis pulmonalis, it is necessary to define in exact terms what we mean when we use these

words. They are employed so loosely, and with such wide and diverse significations, that any argument about them will surely end in confusion unless we settle what lesion we are to call phthisis pulmonalis, and unless we can give something like a rational definition of what a tubercle is.

I presume that the members of the Society will agree with me that the body which we generally understand as tubercle is typically seen in the different organs in general tuberculosis of children and adolescents, consecutive to the cheesy softening, say, of an enlarged lymphatic gland of the neck. Such tubercles are found in the lung, liver, spleen, kidney, peritoneum, pleura, meninges, and elsewhere. Granting, therefore, that these bodies are typical instances of tubercle, what I propose to do is to take the structure of one of these as our model, and to call bodies similarly constituted by the name of tubercles, and to discard all other bodies from this nomenclature which do not possess such a composition.

In this course I believe I am thoroughly justified from the fact, which in my experience has never failed, that, if properly examined, all the nodules occurring in such cases have identically the same histological structure and mode of origin. It may happen that the development of this structure might approach perfection more in some organs, or in particular nodules in a certain organ, than in others, but, nevertheless, if a series of the nodules in any organ be systematically examined, essentially the same composition and mode of growth can be observed in each. A question has been raised of late as to whether the mere histological features of a tubercle can be taken as a test of identity. My reply to that is that if we can define histologically what a cancer, a sarcoma, a fibrous tumour, or a myoma is, then the same applies with even more force to the detection of a tubercle.

The appearance of the body, which I will call tubercle, is, that it is rounded in shape, about the size of a mustard seed, grey or yellow in the centre, somewhat fibrous or even cartilaginous in texture, and when examined microscopically it is found to be an isolated and sharply demarcated mass of new formed tissue. In its centre or at its sides are invariably, if the tubercle is not too old or too young, one or more giant cells. From their periphery processes of fibrous tissue are given off, which, by dividing and subdividing, form a surrounding reticulum. Within the meshes of this reticulum, or lying flatly upon it, are connective tissue corpuscles, which bear the same relationship to the fibrous wall of the reticulum on which they lie, that they bear to a bundle of ordinary fibrous tissue.

The reticulum is usually somewhat condensed at the periphery, thus constituting a spurious capsule. This limits the growth of the body, and gives it the rounded appearance which is so characteristic. One nodule does not fuse with those adjacent to it, although it may be connected to them by an intervening septum of fibrous tissue. Finally, this body, so far as I have seen, is always preceded by a softening caseous mass either in the tubercular organ itself or situated in some distant part.

The term phthisis I employ in an exclusively local sense, not as referring to a general marasmus, the result of a lung disease, but merely as indicating a local destruction of the lung of a peculiar nature. This destruction of the lung results from caseous catarrhal pneumonia. Softenings of the lung may owe their origin to so many different causes that this restriction is absolutely necessary. The organ may be the subject of a so-called fibroid phthisis, a coal miner's phthisis, or a stone mason's, or needle grinder's phthisis. Or it may be a gangrenous phthisis, or a phthisis due to gradual obliteration of a branch of the pulmonary artery. All these I exclude from the category of pulmonary phthisis, for the very good reason that they represent processes essentially different in their causation. I would also specially emphasize that bronchiectasy is frequently, very frequently, mistaken for phthisis resulting from catarrhal pneumonia. It need hardly be said that I exclude such mere bronchial widening from this designation. Phthisis pulmonalis, as I intend speaking of it to-night, is the destruction of the lung which results from catarrhal pneumonia.

Having thus stated what I mean by the terms *tubercle* and *pulmonary phthisis*, let us examine what the conditions are under which tubercle arises in the lung.

It is either the primary disease *of the lung*, or it is secondary to some lung disease which is not tubercular. As an instance of primary tubercle, we may take the case familiar to every one in the child, where an eczœma of scalp is the commencement of the history, an enlargement of the cervical glands follows, and where death from general tuberculosis finally occurs.

In such a case both lungs will be universally studded throughout with exemplary tubercle nodules, grey or slightly yellow in the centre, isolated, and having all the other characteristics previously enumerated. The cervical glands will be found to be cheesy. In such circumstances the tubercle is the only disease of the lung. It may be otherwise healthy. It looks as if so many parasites had been scattered throughout it.

The other condition under which tubercle of the lung is found is where it is secondary to some caseous deposit which is not in itself tubercular. This caseous deposit may have various modes of origin. A catarrhal pneumonia is the commonest. Gummatous areas of cirrhotic lung tissue also induce it. Enlarged and cheesy bronchial glands, especially enlargement of those small glands which are continued far into the lung substance, as the so-called lymph-adenoid deposits. These frequently become swollen in children after the bronchitis and catarrhal pneumonia of measles, and when they caseate are one of the commonest causes of general tuberculosis.

In such lungs the tubercles have an entirely different distribution, although they are structurally the same as those found in the primary disease; and the difference in their lines of distribution is owing to the channels by which they are propagated. Both are due to the irritation of the caseous matter which has been absorbed from the primary source of infection; but, in the case where this infecting source is situated without the lung, the caseous matter is carried into it by means of the blood-vessels; while, if localized primarily in the lung itself, the lymphatics are the channels by which it is conveyed.

Such being the case, it is evident that, in the primary form, the general distribution of the tubercles is owing to the fact that particles of this caseous irritant, if we may so call it, are circulating with the blood current, and are carried indiscriminately, as regards distribution, into the lung and other organs. In the instance of tubercle accompanying a softening deposit of caseous catarrhal pneumonia, there may be general tuberculosis elsewhere, but in the lung the tubercle has a local distribution, owing to the neighbouring lymphatics having absorbed the caseous irritant.

Now, I hold that it matters not whether the caseous irritant gets into a blood-vessel or into a lymphatic-vessel. In both cases it will equally well give rise to a tubercle. All that seems necessary for the growth of a tubercle is the caseous irritant and an endothelium or connective fibrous tissue, that is to say, a meso-blastic structure. The reaction of the one on the other is capable of developing this neoplasm. The most favourable endothelia are those of the capillary vessels and the small lymphatics.

I have previously defined phthisis pulmonalis as that softening of the lung which results from catarrhal pneumonia. There are three distinct stages in the disease—as distinct as the stages of a croupous pneumonia.

The first is the stage of acute or sub-acute catarrh, the second is the stage of caseation, and the third is that of phthisical softening. In the first stage, the air vesicles of certain lobules are filled with catarrhal fluid. This fluid is made up of mucus, with great numbers of large cells derived from the proliferation of the nuclei of the pulmonary epithelium.

In the second stage, this fluid becomes richer in cells, poorer in mucin constituents, and it caseates. The walls of the air vesicles containing it also participate in the caseation. The cause of this cheesy degeneration is the gradual obliteration, as shown by injected specimens, of the capillaries supplying the part, from the pressure exerted upon them by the accumulated catarrhal products.

In the third stage, the caseous necrotic mass softens or ripens in the centre, and a phthisical cavity results.

In other organs having a tubular structure and lined by epithelium, there is an analogous process of caseous catarrh and phthisis. The so-called genito-urinary phthisis and phthisis of the testicle are instances of this.

The general impression is that phthisis of the kidney and tubercle of the kidney are the same disease in different stages, but I hold that this is entirely erroneous. The so-called phthisis of the kidney does not commence as a deposit of tubercle, and a primary deposit of tubercle in the kidney does not lead to a phhthisis any more than a primary deposit of tubercle in the lung induces a phthisis of that organ. Phthisis of the kidney commences just as catarrhal pneumonia does, in an accumulation of epithelial products in the urinous tubes. This epithelium, instead of being voided, as usually happens, becomes impacted in the urinous tubules. It dries and (along with the surrounding tissue) caseates just as in catarrhal pneumonia. The caseous mass then softens, and a phthisical cavity results. Tubercles may now form in the neighbourhood, just as they do in a phthisical lung. They are secondary to the primary catarrh.

Tubercle of the kidney, when the primary disease of the organ, usually does not become excavated to form a cavity. A nodule may soften in the centre, but the *debris* is soon absorbed, and the cavity closes by cicatricial contraction. It is a purely local deposit. Phthisis of the kidney, however, involves large masses of the kidney substance, whole groups of tubules, and the softening may be so general that nothing but the capsule and the pelvis actually may be left. In the case of primary tubercle of the lung the same holds good. It does not give

rise to a phthisis, but rather, in the course of time, to a cirrhosis of the organ.

In contrast to pulmonary phthisis as above defined, let us briefly examine some of the other morbid processes in the lung which also go by this designation.

One of these is named "fibroid phthisis." This disease is due to chronic interstitial pneumonia, and the so-called phthisis is nothing more than a bronchial dilatation—a bronchiectasy. The cavities so formed are constantly mistaken for cavities due to lung disintegration. They are frequently very large, so that they may involve the greater part of an upper lobe. In some instances of this disease an obliterative thickening of the inner coat of a branch of the pulmonary artery may occur. This, in certain cases, produces a local destruction of lung substance of limited extent, but the space so left being invariably in the midst of a mass of cicatricial tissue closes by surrounding contraction. In true catarrhal phthisis it is not so. It is rare, if it ever happens, that a truly phthisical cavity closes in this way.

In the coal miner's lung a disintegration sometimes takes place, known as a phthisis. A sloughy cavity is formed, accompanied with great destruction of the lung. This softening, however, is never caseous in its nature. It is due simply to the pressure caused by the accumulated foreign particles upon the small branches of the pulmonary artery which they surround. It so presses on some of them that in severe cases I have seen the lumen of the artery entirely occluded. The result is that a slough of the lung tissue follows.

In the stone mason's lung the so-called cavities are usually dilated bronchi. The stone dust seems to be much more irritating than coal dust or soot, and induces a cirrhosis of the organ. The cirrhotic tissue then contracting, pulls the bronchi open on principles well recognised.

The absurdity of including all these different sources of cavity formation under one common designation, therefore, becomes apparent, and leads to endless confusion.

Having already absorbed so much of the time of the Society, I feel that perhaps I have said enough on this very wide subject, although there still remains a great deal of interesting material which might form subject for debate.

Before concluding, however, I would, with your permission, say a few words on a subject already broached by Dr. Foulis—namely, the transmissibility of tuberculosis.

Villemin's experiments conclusively proved that the contents of a cheesy gland, if inoculated, are capable of inducing a

general tuberculosis. The later experiments of Orth and others have proved beyond any reasonable doubt that caseous matter, when swallowed, will also induce the formation of tubercle. Are we, therefore, to say with Cohnheim, that the caseous matter and the tubercle are identical—that all is tubercle which, by inoculation into properly constituted animals, is capable of inducing tuberculosis? I most distinctly beg to oppose this opinion. If I apply solution of cantharides to my skin, and I thereby produce a vesication, am I, by any logical process, to conclude that the cantharides and the vesication are necessarily the same. The two cases are parallel in their conditions. The caseous matter is, I hold, an irritant, and the thing which we call tubercle is merely the fibrous hyperplasia resulting from the application of that irritant to an endothelium. The whole of the histological elements entering into the constitution of a tubercle, if sufficient time is given, and after the stimulating effects of the irritation have passed off, resolve themselves into fibrous tissue. The giant cell is simply, as Virchow long ago pointed out, an enlarged connective tissue corpuscle. Apply any irritant of the same power to an endothelium and giant cells will be formed in abundance.

A great deal has of late been written about the transmissibility of tubercle from person to person, or from the lower animals to Man. The enlargement of the cervical glands in strumous subjects has been accounted for by the consumption of milk derived from cows which were supposed, but not proved, to be the subjects of *Perlsucht*, or bovine tuberculosis. The occurrence of tubercular meningitis has been attempted to be traced in certain cases to the action of a subtle tubercular poison which, when inhaled, finds its way from the nares through the ethmoid plate to the cerebral meninges. Such speculation, for we cannot call it otherwise, is idle, and ill calculated to throw anything like a scientific light upon the subject of the assumed transmissibility of the tubercular poison or irritant from the lower animals to man.

No one can deny that if the milk of a tubercular cow were to produce an epidemic of tuberculosis in the consumers, there would be a strong *prima facie* case in favour of the theory of its transmission from beast to man by this means. But that the milk of tubercular cows actually contains this poison, seems to be very doubtful, and has never been proved beyond dispute. It seems to be a particulate element, and the manner in which it spreads throughout the body strongly impresses one with the idea of its being conveyed embolically.

If, then, it acts embolically, I see some difficulty in understanding how it can escape through the udder of the cow. The fact that tubercular mothers do not give birth to tubercular children is also strongly in favour of the theory of its embolic action. In syphilis the case is quite different. Here we have evidently to do with something which is soluble in the blood, and which is capable of transmission through the boundary between fœtal and maternal circulations.

Taking all circumstances into consideration, I think that the danger of tuberculosis being transmitted from the cow to man has been a good deal exaggerated, and that the subject requires further careful observation and analysis.

Finally:—Is pulmonary phthisis hereditary? I would say that the tendency to it certainly is; but, that it is due to a special poison transmitted from parent to child, I hold we have not any evidence to prove.

That which I hold is transmissible is the peculiarly sensitive character of the pulmonary epithelium, by which it is more easily influenced by outward irritation than that of a normal individual. It is very much the same condition, I presume, as that of the epidermis of the face in certain persons in whom the exposure to cold air would induce desquamation to an inordinate extent, while in another individual the effect would hardly be appreciable. A fineness of skin and a profuse growth of hair are two of the characteristics of persons liable to phthisis.

These conditions of the epidermis are apparently coincident with a similar "delicate" or impressionable state of the alveolar epithelium. This is evidently engendered by bad hygienic surroundings, and when once set up appears to be capable of transmission from parent to child. Such a person is said to have a delicate chest, and the slightest undue exposure is sufficient to induce a broncho- or catarrhal-pneumonia. If the catarrhal products should accumulate and dry, as they have a great tendency to do in such individuals, then caseation occurs, and, as an effect of this, disintegrative softening, or, as I have called it, pulmonary phthisis follows.

Dr. Gairdner said that, having been requested to take part in this discussion, he gladly did so, although fully aware that on the pathological side he could not pretend to either the information or the opportunities of personal investigation which would entitle him to rank with the preceding speakers. In this respect he followed Dr. Hamilton at a great disadvantage; for the Society had just listened to a most able,

luminous, and thoroughgoing exposition of the histological and pathological relations of tubercle, from one who, by his studies abroad and at home, might be considered as perhaps better fitted than any other to represent the great impulse of pathological doctrine commonly associated with the name of Virchow; whereas he (Dr. G.) had acquired most of his experience in pathological histology during the time when these ideas were as yet inchoate—*i. e.*, before the publication of the famous *Cellularpathologie* in 1858, and had since that time only kept up his interest in the subject as a physician might do, without too much diverging from clinical work. Still, in a Society like this, and in a discussion like the present, there might be something appropriate in these remarks from one who, beginning as a pathologist, had ended as a physician. Now, from this point of view he was inclined, in the first place (though not a remark of the first importance), to take exception to Dr. Hamilton's view of *phthisis pulmonalis* as implying merely ulceration of the lungs, just as in the so-called renal phthisis ulceration of the kidney and ureters gave the name to the disease. Beyond all question the term phthisis, interpreted from the historical and clinical point of view, does not mean ulceration, nor yet destruction of any kind, of the lung or any other organ, but wasting, or, as it is still popularly called, *decline*, of the whole body; and it is only in modern times, and for the most part since the time of Louis, that the inverted application of the word here referred to arose. Phthisis does not at all, in its essential meaning, represent tubercle or any other pathological state of the organs; even phthisis pulmonalis does not mean wasting of the lungs, but wasting of the whole body accompanied by predominating pulmonary symptoms, which the pathologist now well knows to be in general significant of what used to be called tubercular disease in the lungs and elsewhere. Passing from this matter of definitions, however, to the more essential questions involved in this discussion. It is not difficult to indicate several epochs, each marked by a special character or tendency, both of observation and of opinion, in regard to the diseases commonly considered as having affinity with tubercular phthisis. We need scarcely, however, for the purpose at present in view, go back beyond the time of Laennec, or from 1819-25—the interval between Laennec's first edition, and the great work of Louis on *Phthisis*, a time which all will admit, notwithstanding the previous foundation laid by Bayle in 1810, to have been the great period of initiation, for the medical profession at large, into the idea of what

was afterwards called tuberculosis. We may call this the period of the recognition of tubercle as a distinct and probably specific morphological type, related in many ways to a great variety of previously well known diseases, and especially to phthisis pulmonalis. By thus defining and specifying tubercle on the basis of anatomical facts observed in connection with clinical, and especially physical, diagnosis, Laennec unquestionably gave an importance and a definiteness to the idea of tuberculosis, which were entirely novel, and which became the starting point of a host of new observations and researches. Inheriting, as we do, the results of this movement as transmitted and carried on by Andral, Cruveilhier, Carswell, and, above all, by Louis, we are perhaps apt to ascribe to Laennec opinions about tubercle which he would probably not have stated without reservations; and errors which were the errors of others more than his. For example, although Laennec undoubtedly laid great stress upon both miliary and crude tubercle as distinct anatomical forms, we are scarcely authorised in affirming that he regarded either of them as being essential to the idea of a tuberculous structure.* On the contrary, in the description he has given us of what he called "tuberculous infiltration," we can easily observe him to be grappling with the same difficulties that we now experience as to the connection of tubercular with inflammatory processes. [Dr. G. here showed a portion of lung which he had preserved for more than thirty years, as being a typical specimen of Laennec's infiltrated tubercle, but which now would probably be designated as caseating pneumonia.] It is by no means to be too readily assumed that Laennec believed a tubercular condensation of the lung, or even what he would have regarded as a tubercular excavation, to be impossible without those definitely rounded forms, called more distinctively tubercles, occurring as a first stage in the process. All that his researches necessarily imply is the frequent presence of the miliary or of the crude form of tubercle, as a note or sign of the specific constitutional taint which leads, in so

* "La matière tuberculeuse peut se développer dans le poumon et dans les autres organes sous deux formes principales, celles de *corps isolés* et d'*infiltrations*. . . . Quelle que soit la forme sous laquelle se développe la matière tuberculeuse, elle présente dans l'origine l'aspect d'une matière grise et demi-transparente, qui peu à peu devient jaune opaque et très-dense. Elle se ramollit ensuite, acquiert peu à peu une liquidité presque égal à celle du pus; et, expulsée par les bronches, laisse à sa place des cavités connues vulgairement sous le nom d'*ulcères du poumon*, et que nous désignerons sous le nom d'*excavations tuberculeuses*."—*Ausc. Méd.*, 2ième edition, 1826. T. I, p. 534.

many cases, to destructive excavation of the lungs, as well as to a number of other local lesions similarly characterised by a tendency to ulcerate. Laennec unquestionably believed that the tubercular processes, taken as a whole, were specific, and distinct from, though they might be associated with, inflammation.* He was, however, familiar with the "secondary eruptions," as he called them, of miliary tubercles occurring as the result of localised caseating deposits of older date, only he regards these older deposits also as a part of the "general disposition" which presides over all the local manifestations alike.† So with regard to hæmoptysis, Laennec does not maintain that the ancient doctrine of *phthisis ab hæmoptoe* is absolutely and in every case wrong, but only that there is no positive fact which proves that hæmoptysis can, *per se*, originate tubercles, while the presumptions are, on the whole, the other way, and the majority of cases of hæmoptysis occur in the course of tubercular disease already in progress.‡ It will thus be seen that Laennec, even when his opinions are not in accordance with more modern pathological ideas, has stated them in such a way as not to be open to the censures that have sometimes been passed upon him.

The second epoch which requires notice is that of the first impetus of pathological histology in relation to tubercles, by the application of the microscope to the analysis of tubercular and scrofulous structures, in the hands of Lebert, whose work on the subject was published in 1849. The successors of Laennec had so insisted on the specificity of tuberculosis, that it was almost inevitable that the microscope, in the first enthusiasm of its application, should be expected to disclose a specific form corresponding with the assumed specific nature of the deposit, or exudation (as it was then commonly called). This Lebert assumed to have done by the discovery of the "tubercle-corpuscle;" and for a time *morphological specificity* was in the ascendant, and not only tubercle, but inflammation, cancer, and almost all kinds of tumours, were supposed to be demarcated absolutely in nature by the cell-forms contained in them. This pathology, however, did not hold its ground

* He maintains this at great length in opposition to Broussais, in a special article "Les tubercules sont ils un produit de l'inflammation." *Ausc. Méd.*, p. 562. "Une multitude de faits prouvent," he concludes, "que le développement des tubercles est le résultat d'une disposition générale, qu' il se fait sans inflammation préalable, et que, lorsque cette dernière coïncide avec l'affection tuberculeuse, elle lui est le plus souvent postérieure en date." P. 578.

† *Ausc. Méd.*, 2ième edition, 1826. T. I, pp. 553 and 579.

‡ *Ibid.*, p 645.

very long. Scarcely had it been promulgated, before Reinhardt was at work on the so-called "inflammation globule," or compound granular corpuscle found in the lungs and elsewhere; and by a magnificent series of generalisations, published mostly in *Virchow's Archiv*, it became apparent that the assumed specificity of cell-forms was devoid of foundation in fact; and that cells, however arising, *e.g.*, in physiological tissues, tubercle, inflammation, cancer, underwent similar processes of evolution and decay, so as at certain stages of their existence to be undistinguishable from each other. Thus, the way was paved for Virchow's famous *Cellularpathologie*, published in 1858, and his larger, if not more important, work on *Morbid Growths*, in 1862-63. The doctrine of these works was that all pathological cell-forms were but evolutions and outgrowths from normal structures; in fact, that every single element of a so-called new formation, instead of arising *de novo* in a plastic medium or exudation, was based upon a pre-existing cell or nucleus; so that *omnis cellula e cellula* became the general law in the light of which all pathology as well as physiology was to be studied; and as regards the tubercle-corpuscle in particular, its specificity was entirely denied, and its morphological characters affirmed to be simply those of any shrunken, withered, organism of feeble vitality, incapable of further development, and yielding readily to disintegration. Thus arose the idea of caseating structures of indifferent origin, sometimes inflammatory, often glandular, the result of previous inflammatory irritations, which, it was held, at certain stages of their retrograde metamorphosis, or *necro-biosis*, gave rise to new combinations of organic debris which, by their influence on the neighbouring tissues, or even sometimes on distant parts, inoculated them as with a virus or ferment, and produced secondary crops of miliary tubercles. Under the influence of this system of doctrine morphological specificities altogether disappeared, and the position of tubercle became somewhat like that of the secondary abscesses in pyæmia; most of the changes in organs which, since the time of Laennec, had been regarded as tubercular, were now said to be simply inflammatory, and especially almost the whole of the yellow or crude tubercles, together with all the scrofulous cheesy deposits in glands, were declared to be non-tubercular, and the name of genuine tubercle was reserved to be applied, if at all, only to the miliary granulations of late origin, or what Laennec had already called the "secondary eruptions." But while this doctrine was becoming largely accepted in Germany, a new impulse was given in France to the doctrine of specificity of tubercle

from the experimental side, by the large number of artificial inoculations in animals, practised by Villemin between 1865 and 1868, the results being published in a volume in the latter year. This was an absolutely new starting point; it was plausibly maintained that tubercle, whatever its morphological characters, is demonstrably as specific, and under certain given circumstances as specifically infectious or inoculable, as syphilis or small-pox; a view obviously opposed to, and, indeed, in a great degree destructive of, the hypothesis of the origin of tubercle indifferently from almost all caseating inflammatory deposits. From that time to the present, the successive researches of Wilson Fox, Sanderson, Klebs, Buhl, Cohnheim, and others, have been directed towards the clearing up of the obscurity left by the apparent conflict of the morphological pathology current in Germany, and the experimental results first formulated in France by Villemin. There cannot be a doubt that the absolute validity of the conclusions of this observer has been justly called in question, inasmuch as it has been shewn that in the animals susceptible of infection, other than tubercular substances will sometimes lead to results not dissimilar from tubercular inoculations. On the other hand, it is not without significance, that a distinct movement of reaction has taken place in Germany, as regards the morphology of tubercle; some of the best authorities, as Rindfleisch, being now disposed to affirm, on morphological grounds, that caseating scrofulous glands, and also in many instances other yellow caseous deposits, must be admitted to be tubercular after all, and not merely inflammatory. We have heard Dr. Hamilton's opinions on this subject, and it is evident that they are not altogether the same as those of Dr. Coats and Dr. Foulis. In particular, the significance of the giant cell, and the claim of miliary tubercle to be the sole representative, morphologically, of tuberculosis, are very differently handled by these experts. But the most remarkable fact in this lengthened story is that Cohnheim, certainly one of the most advanced and original minds at work in Germany, now tells us that neither in giant cells nor in any histological character whatever, can the true pathological diagnosis of tubercle be found, but only in the results of inoculation into the aqueous humour of an animal; and that the sequelæ of such inoculation are specific, in much the same sense as in the case of syphilis or small-pox. It is impossible to reconcile these varying opinions; but in presence of them it may be permitted to a physician to reserve his judgment, and to suppose that the last word in this great question

has not been spoken yet. Dr. Gairdner concluded by putting briefly some questions from a practical and clinical point of view, which appear to remain undecided after all that pathology has told us. F. von Niemeyer, in his *Clinical Lectures on Phthisis*, which are a most thorough-going application of Virchow's doctrine, affirms repeatedly that in order to account for the phenomena of inflammation in different subjects, especially as leading or not leading to caseation, and therefore to secondary tuberculosis, you must assume a "vulnerability" on the part of certain persons to irritations which leave little, or at least far less permanent, impressions on others. The so-called scrofulous child is a vulnerable subject in one direction; his eyes, his skin, his bones and joints succumb to influences that do not disturb the health of other children, and caseating deposits are the result. The adult who ultimately falls a victim to pulmonary phthisis is vulnerable in another direction; attacks of catarrhal pneumonia follow each other, and lead to caseation, and thence to miliary tuberculosis. Dr. Hamilton's expression for the same fact is, that these subjects have an undue susceptibility to proliferation of epithelium, which in its turn leads to accumulation and stagnation, then to obliteration of groups of air vesicles and other changes which he has so clearly described, and so to caseation. But I want to know more intimately (said Dr. Gairdner) what is this "vulnerability" or morbid susceptibility? There is not a man in this room who is not in a certain sense vulnerable. Most of us have had catarrhs at one time or other; many of us, perhaps, have had them often and severely. Now I notice in practice that there are catarrhs and catarrhs. Some men have them rarely and mildly. Others have them severely, but they never extend beyond the larynx. None of these, it may be, are in danger of pulmonary tuberculosis, but they are all, more or less, vulnerable. But there are patients in whom almost every catarrh settles down instantly upon the chest; and here again I notice a new distinction. There are a considerable number who are thus vulnerable, who go on from boyhood to manhood, and from this to old age, wheezing and expectorating at intervals, and at times suffering pretty severe dyspnœa; some of them are rarely quite free from catarrhal symptoms for years together, and we call them asthmatics; but, after a time, if not at first, we get to know that they are not likely to fall into tuberculosis, but will certainly, if they live, become the subjects of pulmonary emphysema, and probably dilated heart. The others I referred to who are also vulnerable, though in a different sense, can scarcely suffer

one or two attacks of pulmonary catarrh without its becoming evident that the apices are unduly involved, and sooner or later dulness on percussion and other signs disclose themselves, and the case goes the way of a more or less chronic phthisis. The following is a case which I am confident every practitioner will recognise as one within his experience, although he may not always be able to ascertain all the facts with rigid accuracy. A child or a young person may have no complaint of the chest at all; and the most careful scrutiny may fail to detect any liability to catarrh—any vulnerability, in short. But from quite another side (say the brain or meninges) the case is suggestive of tubercle, and you make an examination. There, at the very apex of one lung, possibly of both, you find the inevitable dulness on percussion, the harsh or hollow respiration, and perhaps just a minute click or two of râle confined to that single spot; all the rest of the lungs being apparently sound. Why should that spot in this child's lung caseate, with a catarrh absolutely insignificant? And why, on the other hand, should a certain number of persons, eminently vulnerable to catarrh, escape caseation? Suppose that the child belongs to a family of which many have been cut off by phthisis, while the emphysematous and asthmatic subjects have had ancestors who died chiefly of cardiac disease, apoplexy, aneurism. Should I not be justified in assuming that a tuberculous predisposition existed in the one class of cases and not in the other? Again, let us revert for a moment to the case of "phthisis ab hæmoptoe." I produced that case to you because it was, more than any recent one within my experience, capable of being accommodated to the theory of miliary tuberculosis occurring as the result of caseation and ulcerative cirrhosis, which *may* (for aught I know) have sprung directly from blood detained in the pulmonary alveoli, or aspirated back into them from the bronchi. But in how many instances do we all know of blood being so impacted in the lung, and yet no caseation? Why do the hæmorrhagic condensations of mitral stenosis, and of pulmonary embolism, almost never caseate or give rise to tubercle? Why did the blood in this lung (showing a drawing), pumped into the bronchi and then aspirated from an aneurismal sac, so impacted that it led to dense lobular condensations, many of which were undergoing a peculiar grey degeneration, obviously requiring much time—why did this blood and this lung escape caseation and tubercular disease? If hæmorrhage in the lung is as apt to lead to inflammation and caseation of the surrounding tissue as Niemeyer supposes, why does it in cases of heart disease or

embolism so frequently undergo degenerative changes—fatty, suppurative, gangrenous, and other, but never, so far as I have observed, caseation or anything resembling it? And why does an old apoplectic cyst in the brain never caseate or lead to miliary tubercle either of the meninges or elsewhere? Blood is extravasated every day in every region and tissue of the body, as the result of injury, for example, or of purpura, or of scurvy, and we very rarely hear of its being followed by inflammation in any such sense as to produce either suppuration or caseation; and yet, when a young man, perhaps with a bad family history, has hæmoptysis as a first symptom and phthisis follows, we are told to believe it is because of the strong tendency that blood drawn into the lung has to excite inflammation there, and thus to lead to caseation, and in due course miliary tubercle. I incline with Laennec to think that this view of the sequence of events is rather more difficult than the opposite, and that it is easier to suppose, not perhaps that actual tubercle is necessarily there beforehand, but that when caseation or tubercle follows a hæmoptysis, there has been usually some previous constitutional infirmity, which I will continue to call, provisionally, a tubercular predisposition, one effect of which has been to weaken or disturb the pulmonary circulation, and so lead to hæmoptysis, and then to phthisis. But this is only a clinical and practical view, till the pathologist comes and speaks the last word, and tells us finally what tubercles are, and what a tubercular predisposition really signifies.

Adjourned Discussion—*8th March.*

Dr. Finlayson said that, in common with the other members, he had listened with much pleasure to Dr. Hamilton's exposition of his views, and he admired the clearness with which they had been put before the Society. This clearness was no doubt partly due to the fact that Dr. Hamilton was expounding the views which he had formulated after much personal investigation, and also no doubt to his training and practice as an expert teacher. But there was reason to fear that the clearness was also due in part to the very arbitrary distinctions and definitions which he laid down at the beginning of his remarks, and to his ignoring some very important facts bearing on the question at issue.

His definition of what he proposed to term tubercle appeared very arbitrary; and his definition of phthisis pulmonalis seemed

even more unsatisfactory, excluding, as he stated it did, something like one half of the cases regarded as phthisis by hospital physicians during life, and probably also so regarded after the inspection by many of them at least. These points, however, Dr. Finlayson would prefer to leave to professed pathologists, and they would no doubt receive attention that night. He must, however, protest, as a physician, against the definition given of phthisis pulmonalis, not merely on the etymological and historical grounds urged by Dr. Gairdner, but even on the narrower ground of its forming a distinction between local lesions which were thus unwarrantably separated from each other. [Dr. Finlayson here showed the lungs of a woman who had recently died with enormous irregular cavities in both, and with localised pneumo-thorax, as an illustration of what was deliberately excluded by Dr. Hamilton's definition of phthisis.]

It might be asked why he ventured to take part in this discussion if he were not a professed pathologist. He did so with some diffidence, but he felt that the issues raised involved something more, something much greater, than mere questions of histology. Moreover, his attention had been somewhat specially directed to the subject soon after he entered on practice, and ever since 1868 he had been anxiously considering the relationship between phthisis and tuberculosis. About that time his attention was first called to the allegation that in tuberculosis, with the well known grey granulations, there was always some pre-existing cheesy deposit or similar condition; and at the same period he had begun to try to discover whether careful thermometric observations might help the discrimination of cases of tubercular and non-tubercular phthisis. Since then, both by observation and reading, these objects of inquiry had been kept pretty steadily in view. In the earlier part of his experience in Manchester, his cases were almost exclusively those of children, in whom the evidence of miliary tuberculosis, when present in a pronounced form at least, was usually plain enough to the naked eye; in the latter part of his experience he had had the benefit, almost invariably, of Dr. Coats's presence at the *post-mortem* examinations. The conclusion arrived at was this, that although in the great proportion of cases of miliary tuberculosis some cheesy mass or similar lesion could be found, there was a residuum of cases where no such thing was discovered even after a careful search; and indeed in some of the cases where it was discovered the mass was so small and apparently insignificant that it seemed hard to blame it for the tuberculosis, as many such lesions

were often found without any tuberculosis being present. With regard to the discrimination of cases of phthisis, by means of the thermometer, into tubercular and non-tubercular, he had failed completely, as others had also failed, to obtain any reliable distinction.

But the real point which had urged him to open this adjourned debate was the promulgation by Dr. Hamilton of the extraordinary doctrine that neither tuberculosis nor phthisis pulmonalis should be regarded as hereditary.* Now, Dr. Hamilton was much too skilful a supporter of his views to have dragged this opinion before them unless it had a vital bearing on his doctrine. To him phthisis pulmonalis is but a catarrhal inflammation, such as any one may take at any time, going on to the destruction of the upper portions of the lungs, for mechanical reasons which he expounded with much ingenuity; and the lungs are only saved from destruction in a multitude of other cases by the accidental coincidence of mitral disease or renal disorder, which, by keeping them moist, prevent caseation. If all this were so, of course there is but little room for hereditary influence. But the facts were too strong even for Dr. Hamilton; for he no doubt knows as well as any one that the hereditary tendency to phthisis is one of the facts of clinical experience most firmly established. He resorts, therefore, to a mystification of words; phthisis being the destruction resulting from the caseation of the proliferated epithelium of a catarrhal pneumonia, he alleges that the patient has no hereditary susceptibility to phthisis as such, but that there is an inherited susceptibility to catarrhal inflammation and to an excessive proliferation of epithelium; and this leads, in weak or predisposed subjects, to caseation and phthisis! Such distinctions are surely out of date at this time of day, both in science and philosophy.

This might be said to be the weakest part of Dr. Hamilton's speech, but there was, if possible, one part weaker still, and that was the reason he gave for phthisis and tuberculosis being non-hereditary, for, he said, you do not get them in a new born child. It is probable that he meant that they were *never* found in such; for it appears that he does not mean to exclude infantile syphilis from hereditary diseases, although the manifestations of this disorder are usually delayed for some time after birth.

But is it true that phthisis and tuberculosis are never congenital? The earliest age at which Dr. Finlayson had

* Dr. Hamilton, in revising his notes for publication, has somewhat modified the language here criticised.—Ed. *G. M. J.*

verified the existence of phthisis pulmonalis by *post-mortem* examination was in a baby six months old; but this child was said to have had a cough since birth; the lung disease had not apparently been advancing very rapidly; it could just be recognised at the apex of the left lung during life, and death was occasioned, not by the advancing pulmonary mischief, but by an attack of basilar meningitis. In the upper part of the left lung, cheesy deposits were discovered; some of these had softened and formed a distinct cavity. In this case the pulmonary mischief must surely have begun at a very early age. He had made no search into the literature of this subject; but in Gerhardt's *Handbuch der Kinderkrankheiten* (Bd. 3, Hft. 2. S. 787, Tüb. 1878) there are cases given at much younger ages by several observers; and one case in particular proving fatal at the twelfth day of life, with cheesy masses and cavities, varying from the size of a pea to that of a peach stone, is there given. As the author says, it may well be reckoned that these lesions were at least, in part, of intra-uterine formation; so that Dr. Hamilton's extraordinary test breaks down completely. With regard to tubercle, we find in the same book (Bd. 3, Hft. 1. S. 170) that it is stated by Fränkel, of Berlin, on the authority of an oral communication, that congenital tuberculosis was seen on one occasion by Virchow himself.

But even if these cases had not been recorded, or had been overlooked here, what is the value of the reason given by Dr. Hamilton for denying the hereditary nature of phthisis? Is nothing hereditary unless congenital? Surely we may fairly believe that tallness and obesity are often hereditary, although, to use his own words, they are "practically unknown" in new born children. Or, if exception be taken to these as not being pathological conditions, what of gout? Surely we have here a disease notoriously hereditary, and also notoriously late in developing the signs of its presence; so much so that it is seldom found till many years after the full adult age.

In conclusion, Dr. Finlayson said that, in his opinion, both phthisis and tuberculosis were closely allied to that constitutional state in which scrofulous disease in its various forms was found. Phthisis, tuberculosis, and scrofula were so mixed up, both in the personal and the family history of our patients, that the tendency to these had to be regarded as practically identical. The tendency to such diseases was often strong, and bound to manifest itself in some way, but in other cases it was no doubt much less pronounced; and just as mechanical accidents to such persons might determine serious disease in

the joints and bones, although they would be trivial in a sound subject, so a slight catarrhal pneumonia, or the overstimulation of the youthful brain, might lead, in such predisposed subjects, to destructive disease of the lung, or to fatal meningitis.

Dr. Scott Orr said—I regret that I had not the privilege of being present on the first night of this discussion, and so did not hear Dr. Gairdner's or Dr. Coats's papers read. I had, however, the advantage of listening to the speeches delivered at the last meeting, and certainly I heard with great interest that of Dr. Hamilton. It was clear and lucid, and apparently most convincing; founded on carefully observed facts, which, to himself at least, appeared to be incontrovertible. But if I listened with so much pleasure to his remarks, I heard with no less pleasure and interest those of Dr. Gairdner, which, to my mind, contained a full and complete reply. Dr. Gairdner professed to speak with diffidence, because of late years he had not been so much engaged in pathological investigations as formerly; but I think he spoke from a standpoint of pathological and clinical experience which few of us can pretend to.

If he so spoke, it also becomes me to speak with reserve, seeing I have neither the minute pathological nor microscopical knowledge which those gentlemen who originated this discussion have attained to.

I desire, therefore, to speak from the physician's point of view. I have nothing new to offer, but rather wish to revive old doctrines which, though old, are not behind much of the teaching of the present day.

Dr. Hamilton stated that, invariably, previous to the formation of tubercle, a deposition of caseous matter takes place in some part or organ of the body, and that the system becomes infected by this matter, and as a result, we have tubercle.

Now, I would ask, Are caseous matter and tubercle the same? I believe it is generally admitted that they are not; and yet without the one, the other is not, according to Dr. Hamilton. This does not appear to me to be a very scientific theory, unless the caseous matter be regarded as the first stage of tubercle. Both Drs. Finlayson and Foulis have successfully replied to this doctrine. The latter says:—"It might be said that the caseous material was the original starting point of the disease, but it was not enough to say so; for how many cases were there not in which caseous masses failed to infect the system; while, on the other hand, it could hardly be denied that there occurred instances of acute tuberculosis

where a minute inspection of the body failed to reveal the existence of caseous masses."

A much more rational theory to my mind seems to be, that a low state of the system, with hereditary predisposition, producing deterioration of the blood, determines the production of tubercle. This low state is essentially present where caseation has taken place, and it is this state probably, (for it must be remembered that we are dealing entirely with probabilities), and not infection, which produces tubercle. Any disease which lowers the vitality, and deteriorates the blood, will, if there is predisposition, produce tubercle, and in this view I entirely agree with Dr. Gairdner.

But even inflammation will produce this state. This was the doctrine of my late venerable teacher, Dr. Alison, and it is chiefly to refresh the memories of those who have read his papers in the *Edin. Med. Chirurg. Trans.*, vols. 1 and 3, and direct the attention of those who have not, to them, that I have ventured to speak to-night. He dwelt specially on the difference between healthy and tubercular inflammation. In the former, occurring in healthy people, there resulted pneumonia and the products of healthy inflammation; in the latter, requiring the hereditary taint, which Dr. Hamilton altogether ignores, there is tubercle. This taint is an essential principle in Alison's theory, with it we have tubercle, without it healthy inflammation and its results. What clinical physician is there of any experience who will give up the doctrine of hereditary predisposition in such cases? Alison used to illustrate his views by relating the case of a boy who received a severe injury in the *lower* part of the chest, and ever afterwards was affected with cough and dyspnœa. He was suddenly cut off by confluent small-pox, and on inspection a mass of tubercular deposit was found at the seat of the injury, while the apices of the lungs, the usual site of tubercle, were free from the deposit.

Then, again, we were told by Dr. Hamilton that gravity and dryness of tissue had to do with the deposition of caseous matter, and therefore the apex of the lung was its favourite site, tubercle being subsequently developed in the lower parts of the pulmonary tissue, and if I mistake not, that they did not intermix. This appears to me to be reversing the sequence of events, my belief being that the disease begins by the deposit of tubercles which coalesce, then by the continuance of the low, slow, interstitial inflammation already described, caseation, breaking up, and destruction of lung tissue follows.

But it is known that irritation of any kind, particles of dust, glass, coal, steel, &c., will produce tubercle, caseation, and

phthisis. How does it do so? I answer, by producing this low form of inflammation.

The *giant cell* has been much dwelt upon, and by some has been thought to be present in tubercle always; in short, has been considered pathognomonic of it. Are there giant cells in these last instances? Alison tells us that Cruveilhier injected mercury into the femoral artery of a dog, and Dr. Kay of Edinburgh into the tracheæ of rabbits, and they became phthisical with thousands of miliary tubercles in their lungs, pronounced to be so by the most competent observers of the day, who were not aware how they had been produced. In each tubercle there was a *giant cell!* but it was a minute globule of mercury! In explanation, I beg to quote the following from Dr. Alison's papers:—

"It may be said that if this kind of irritation, acting on the lungs of healthy rabbits, is supposed to produce a deposition of tubercles, resembling those which we distinguish in the human body as scrofulous, we depart from the doctrine generally received among physicians, and illustrated in the former part of this paper, that a peculiar general scrofulous diathesis is much concerned in the production of tubercles in the human body.

"I would answer, 1st. That if it be true, as matter of fact, that mechanical irritation of the lungs will produce deposits in the lungs, not differing in appearance from scrofulous tubercles in their early stages, we must not set aside that fact because it does not accord with our preconceived notions of the pathology of the diseases in which similar deposits take place in the living body.

"But, secondly, it was stated already, as the result of the observations of Andral, that the conditions which appear most requisite, in order that inflammation may generate tubercles in the living body, are the *long duration* and *slight intensity* of that inflammation. It is highly probable that the scrofulous diathesis disposes inflammation to terminate by tubercular deposition, simply by giving it these characters—keeping it up long, and not permitting it to rise high."

Dr. M'Call Anderson remarked that, in discussing the pathology of phthisis there was a tendency to take rather too narrow a view of the subject. We must remember that pathology is the knowledge of disease, and that as Wagner has remarked, the materials of it are chiefly derived from four sources:—1st. Observation at the bedside; 2nd. Experimentation; 3rd. Pathological anatomy; and 4th. Pathological chem-

istry. At the previous meetings he thought pathological anatomy and experimentation had been fully dwelt upon, whilst the others, and especially observation at the bedside, had not had sufficient prominence given to them.

In speaking of tubercle, he wished it to be understood to-night that he meant grey miliary tubercle; not that he wished thereby to imply any theory on the subject. Indeed, he held that there is a very close connection between caseous deposit and grey miliary deposition. He believed that four kinds of phthisis may be admitted:—1st. Acute tuberculosis, where the lungs are more or less thickly studded with grey tubercles; 2nd. Pneumonic phthisis, where the disease commences with inflammation, generally a low form of catarrhal pneumonia, and goes on to caseation, and too often to excavation; 3rd. Pneumonic phthisis becoming secondarily complicated with tubercles; and 4th. Fibroid phthisis. An important question now arises—Can we say during life which of these forms we have to deal with? In some cases it is absolutely impossible, in some we can form a strong suspicion, and in some we can say definitely which variety is present. Let us take them seriatim. 1st. *Fibroid Phthisis.* Taken overhead, this is the most chronic of all the forms. It is associated with far less marked general symptoms; indeed, in some they are almost entirely absent. In this form, too, although on making an examination of the chest the physical signs of dilatation of the bronchial tubes may be mistaken for excavation, still there is not the same tendency for these bronchiectatic cavities to occur at the apex, and there is contraction, with falling in of the chest wall, and perhaps displacement of other organs, especially the heart, which is not observed in other forms. In phthisis with cavities there often is falling in of the chest wall no doubt, but this is due to fibroid change in the lung tissue between the cavity and the chest wall. There is, therefore, little difficulty in saying that a patient has fibroid phthisis, or at least that there is a fibroid element in the case. 2nd. *Pneumonic Phthisis.* The usual history of such a case is this:—The patient, after exhibiting for a variable time dyspeptic symptoms, has a short dry cough, and with this he gradually loses flesh and strength, sweats a little at night, and is perhaps a little feverish. On examination of the chest we find gradually developed consolidation of the lungs, usually at one or both apices, ending too often in the formation of cavities. This is the ordinary pneumonic phthisis; the one which is much the most frequently met with in practice. 3rd. *Pneumonic*

Phthisis Complicated Secondarily with the Development of Tubercles. Can we know in such a case when the tubercles make their appearance? In a great many cases they give rise to no special symptom; but in many they do, and if all of a sudden the patient rapidly loses flesh and strength, has profuse perspirations, high fever, perhaps lividity of the lips, great rapidity of respiration, and if, on examining the lungs, no physical signs evidencing increase in the disease are found, then we have reason to suspect that the development of tubercle complicates the case. We can affirm this in a chronic case; but can we tell if the case is one originally of acute pneumonic phthisis, one of the forms of galloping consumption, and probably the worst form of all, because it produces such widespread and rapid destruction of the lung tissue? It is virtually impossible for any one to say during life that tubercles have become developed, for the symptoms of the tuberculosis are overwhelmed, so to speak, by those of the acute pneumonic phthisis. 4th. *Acute Tuberculosis.* In many cases there are no symptoms at all during life. Pathologists know that in making *post-mortem* examinations true tubercles are often discovered in various organs and tissues, which were not suspected, and which gave not the slightest evidence of their presence during life. But the tendency, when tubercles are present in any numbers in the lungs, is for the disease to give rise to the development of acute symptoms. Can we during life suspect that we have to deal with acute miliary tuberculosis? Not positively, but we can form a shrewd suspicion. If a patient becomes very ill with high fever, rapid loss of flesh and strength, profuse perspirations, lividity of the lips, and very rapid breathing, and on making an examination of the chest the physical signs are very slight, then we have reason to suspect it is a case of acute miliary tuberculosis and not pneumonic phthisis. Dr. M'Call Anderson concluded by saying that he thought there was a tendency now for physicians to be dominated too much by pure pathologists, and if this was submitted to, serious errors might creep in; and one of these had been alluded to by Dr. Finlayson, that phthisis is not hereditary, a statement which no clinical physician could make. The physician must at times assert himself, and not allow himself to be overridden by the pathologist. Indeed, there seems fully as much reason now for the warning, which was given by Trousseau a good many years ago, who said, "For heaven's sake, gentlemen, let us have a little less science and a little more art."

Dr. Alex. Robertson said:—In view of the thoroughness and ability with which this subject has been discussed at our successive meetings, and of the fact that, although my field of observation of phthisis in all its forms is a wide one, I have not made it a special study, I rise to occupy your time with considerable hesitation. Probably, I should not have done so at all, had it not appeared to me that, upon the whole, the weight of opinion, so far as the discussion has gone, was in favour of the doctrines of Laennec and the French school generally; and these doctrines, I am convinced, have in the past exerted an unfortunate influence on medical practice. But while that is my conviction, I do not wish to speak dogmatically, as I doubt if our knowledge of phthisis is so far advanced as to warrant a positive conclusion respecting its pathology.

Putting it concisely, the question before us I conceive is this: Is tubercle, and especially phthisis pulmonalis, usually the product of a special morbid state of the blood, or is it a development from a mere local inflammatory effusion?

I will at once say that my opinion is, that in *most* cases tubercle is of local origin, and arises from the products of inflammation which have caseated and softened, and been taken up by the lymphatics or blood-vessels, in the former case giving rise to a local, in the latter to a general tuberculosis. In supporting this opinion, I shall avoid, as far as possible, reiterating statements and arguments which were so well put before us by Dr. Hamilton—the views, in short, of the German school, in which, however, they were distinctly anticipated by our own Dr. Addison. I shall confine my observations to the objections which have been urged against these views, and particularly those which occur to the experienced physician.

Dr. Gairdner stated as a difficulty that occurred to him, the fact that bronchitis continues year after year, and advances to its fatal close without the development of tubercle or phthisis—a case than which there is none more common in medical practice. I understand the inference from this statement to be, If phthisis is of inflammatory origin, why do not such cases of bronchitis terminate as phthisis? Dr. Hamilton attempted to meet this difficulty by stating that œdema of the lungs resulted, and prevented the development of caseation, which was the antecedent of tubercle. This explanation is only partially satisfactory; for in very many cases of bronchitis, even of long standing, there is no œdema of the lungs. How then are these cases to be accounted for? I believe by

the very simple explanation that the inflammatory action does not generally extend to the minutest bronchi and air cells. Should it implicate them the patient is in danger of becoming phthisical, even though the constitution has previously been good, and free from hereditary bias. Of course, if the patient be of weakly constitution, and particularly if disposed to low chronic forms of inflammation, such as we see in the skin in scrofulous people, besides inheriting the consumptive tendency, the danger is increased many fold. Further, inflammatory products in the alveoli and minute tubes block them up, whereas those formed in larger tubes are got rid of by expectoration. So long as the alveolar wall and interstitial structures remain comparatively healthy, these products may be absorbed, but if these parts are materially involved by the inflammatory process, there is no absorption, and caseation is the ordinary result.

Another difficulty is—patients frequently come before us without history of preceding bronchial inflammation, but simply of failing general health, with perhaps slight cough, on examining whom, distinct evidences of phthisis are found at the top of one or both lungs. How are we to account for such cases on the inflammatory hypothesis? As a rule, in these patients hereditary influence is well marked. But what does heredity imply in cases of that kind? Simply that the lung tissue, *more especially*, is under the proper standard of vital power, this being accompanied by other evidences of low vitality of the system generally.* And it is held that the apices of the lungs are more fixed, and do not expand so freely as lower parts, also less readily get rid of inflammatory products, and that their nutrient supply is not so good as that of the remaining portion of the lungs. From some cause, such as cold and damp to the skin, there is an afflux of blood to the internal organs. This quickly passes away except from the constitutionally weak parts. There it remains and originates

* An example from another department of pathology will illustrate what I consider to be the action of heredity in phthisis pulmonalis:—Insanity is a highly hereditary disease. The inheritance is a local weakness of tissue—namely, of the hemispherical ganglia: there is no morbid condition of the blood in this case. And it is to be observed that other parts of the same tissue, directly continuous with these ganglia—basal ganglia, nutritive centres, and spinal cord, are in a very large proportion of cases quite free from disease. So in hereditary phthisis pulmonalis there is congenital weakness, particularly of the extreme parts of the pulmonary tissue, and a consequent disposition to take on at the weakest point (the tops of the lungs) an insidious form of sub-inflammatory action. Along with this, as I have said, there is usually a general low vitality of the system.

a sub-acute low form of inflammation, the products of which, along with the lung tissue caseate, soften, and give rise to tubercle. This, I think, is probably the course of events in many cases of hereditary phthisis. The difficulty we have been considering seems to me less easily explained by the theory of the French school, that there is a special blood dyscrasia in all cases, or, as Dr. Coats puts it, that there is a special virus in the system; for, seeing that the blood circulates everywhere, Why should the tops of the lungs be the only parts to suffer in the first instance? Does that not point to a local weakness of tissue?

Another objection relates to pulmonary hæmorrhage. It is urged that, for example, in cardiac disease blood may be effused into the lung, condensing its tissue, and yet does not caseate nor give rise to tubercle. There seems, however, no good reason to anticipate that result in such cases. For blood is one of the blandest and least irritating of fluids, so long as it is not in contact with an inflamed surface or mixed with air. In portions of lung condensed by it the air is usually thoroughly expelled, and comes in contact with the blood only in the bronchi leading to the condensed part, and there probably a protecting film is formed. Should the air get mixed with the blood decomposition is apt to set in, giving rise to severe inflammation, and even suppuration or gangrene. In these cardiac cases the walls of the air cells and the surrounding tissues being free from inflammation, and the lung not being constitutionally weak, no irritation is produced, and the sanguineous effusion may be gradually absorbed. On the other hand, in patients with weak lung tissue and a hereditary bias to consumption, low inflammatory action precedes the hæmoptysis, and occasions it through the attending congestion, and it follows that the blood that condenses the lung at the part where the disease begins lies in contact with an inflamed surface, and both participates in and accelerates the morbid changes that are progressing there—changes that end in caseation. Should there be no preceding inflammatory action, it is easy to see that blood, though not irritating to strong lung tissue, may be so in one that is constitutionally weak, and initiate disease in such.

The greatest difficulty that I have is to explain certain cases of acute general tuberculosis. Most cases of that kind are readily enough accounted for when we find caseated glands or caseated matter in walls of cavities or anywhere, as its absorption by the blood-vessels shows how the system as a whole becomes involved. But there are cases met with where no

caseated matter can be found; how do they arise? It may be in persons with strongly pronounced heredity, from the absorption of tuberculous matter in the atmosphere, when near some phthisical person while coughing, or that the blood in such persons spontaneously acquires the condition which is generated by the introduction into it of caseous matter in ordinary cases.

I stated at the outset my belief that Laennec's theory had exerted an evil influence on practice. Being of opinion that the state of the blood is the primary and chief morbid condition, and holding the disease in the lungs to be a mere local expression of that condition, practitioners, it is to be feared, have been led in many instances to give almost all their care to the former, and little heed to the latter. But when the physician considers phthisis to be a consequent on inflammation, the local morbid process receives his assiduous attention, and unquestionably in many cases a great deal of good can be effected by local treatment. At the same time, being well aware that heredity and a feeble constitution are present in most cases, he neglects no measure which is likely to strengthen the system, and obviate, as far as possible, the influence of the morbid disposition which has been inherited.

Dr. Barr said—Assuming that acute miliary tuberculosis might be due to the absorption of inflammatory products which have undergone caseous metamorphosis, he would suggest that, in many cases, the source of this infective material might be caseous accumulation in the osseous cavities of the ear, known as the mastoid cells. In chronic inflammations of the middle ear, we have these cells filled with purulent secretion and epithelial *débris*. These purulent diseases go on for years, and in time this secretion and *débris* become dry, and undergo, undoubtedly, a caseous change, and often after the otorrhœa has stopped for some time, on examining these spaces this caseous material is found. Now, the veins may convey the effete matters from the mucous membrane lining these cells, and as they pass into the sinuses of the skull, a connection is thus formed with the pulmonary tissue through the jugular vein; so that, if it is made out that tubercle may arise from caseous material, this is a probable source of it. Von Tröltsch related three cases of acute miliary tuberculosis, and in all three he found, on examining these cells, that they were filled with this caseous accumulation, all being due to long continued chronic inflammation of the middle ear.

Dr. Joseph Coats closed the discussion with the following remarks:—With several of those who have already spoken, I regret that Dr. Hamilton is not present to-night, as it will be expected that I should more particularly refer to his views. I confess that Dr. Hamilton spoke with a clearness and a confidence concerning the morbid processes in phthisis which I am very far from using; nor do I think would any other pathologist in Europe have spoken with the same dogmatic certainty. In phthisis the problem is an exceedingly complicated one, the disease being found in a great variety of stages in each case—at one part only beginning, at another more advanced, and at another more advanced still; so that you are able to get, from a phthisical lung, almost anything. When I began pathological study, my reading led me to a position almost identical with that which Dr. Hamilton has taken up, and I thought that, to a certain extent, I justified that position by observation on the dead body; but I have been driven from it by the evidence of facts which have come under my observation.

There is one important point in which I agree with Dr. Hamilton. He states that tuberculosis results from a virus; he states it most definitely, and further says that it is a very irritating one. That is a most important step in advance, and it is a position which most modern pathologists are prepared to accept. What are the effects of this virus? I would refer to this lung which Dr. Finlayson has shown already. In it we have the cirrhotic form of phthisis, with no caseous material. The cavities, if you examine the more recent of them, are obviously forming by dilatation of the bronchial tubes. I found, in making a section of one of these lungs, that I cut into a small cavity not larger than a pea, which formed a bulbous expansion of a small bronchial tube. A cavity, we may say, just formed, or in process of formation. After hardening in spirit, I made a section, so that I could trace the wall of the bronchial tube into the cavity, and also observe the lung tissue around. I found in the wall of the cavity signs of extreme irritation, masses of inflammatory cells, and of proliferating epithelium. Outside that I found a great inflammatory new-formation in the lung tissue; but I found more, undoubted tubercles with all their characteristics, giant cells, &c., here and there, in this inflammatory tissue. In this case an irritant had been obviously acting on the wall of the bronchial tube and on the lung tissue, and had produced the inflammatory tissue and the formation of the tubercles, and was thus the cause of all these processes.

I think Dr. Hamilton would admit that, and would say these tubercles were due to the virus, but he would deny that this is a case of phthisis, and for no reason that I can see, except that there is a tubercular virus present. Here, then, we have a virus causing processes which lead directly to the formation of cavities.

Let us now take the case of an advanced caseous phthisis, where there has been abundant breaking down, and where there has been so much caseation that abundant virus must have been produced. In such a case, if you examine the peripheral parts, catarrhal products are found, such as Dr. Hamilton refers to, and you find these products caseating. Do you find that tubercles are met with only at a distance from and secondary to the caseating products? This is what Dr. Hamilton would lead us to believe. But I must state, in the most definite manner, that along with the caseating products you find tubercles; side by side with, and in the midst of, caseating catarrhal products, there are caseating tubercles. I deny that tubercles only occur in the third or softening stage of phthisis; they are abundantly present as part and parcel of the process of caseating catarrh. It is surely natural that a virus produced in such abundance should cause, at one and the same time, catarrhal inflammation and tubercles. Dr. Hamilton states that, in these lungs, the epithelium is peculiarly susceptible. Does this virus not produce the inflammatory products as well as the tubercles? My belief is that the virus, acting on the lung tissue, produces the catarrhal products, induces their caseation, and produces the tubercles. And, further, it is not only along with *caseating* catarrhal products that we meet with tubercles; we meet with them along with catarrhal products before caseation. Many facts bear out this. And now it may be asked—Is it not likely that this virus (still referring to the case where abundant virus is produced) may sometimes cause catarrh without tubercles? Is it not likely that a virus acting less vigorously may produce what is a simple catarrh to our eyes, and alongside of it no tubercles? That is exceedingly possible; but I would say that if you have a virus producing catarrhal products, the chances are that, if you search further, you will find at other points a catarrh with tubercles; and this is my universal experience. So that, although the two are not in every case concomitant, yet they are due to the same irritant.

In all this I have spoken of the effects of the virus where there is an undoubted source of it—where there is undoubted

breaking down of caseous material. I should like now to ask, Is the process different at first? When phthisis begins is there something different? I fail to see that there is a particle of evidence to show that. If the process is different to begin with; if the catarrhal pneumonia breaks out without the irritation of the tubercular virus, then what is the irritant which produces this catarrh, and this caseation of the catarrhal products? I regard the caseous change of the catarrhal products, involving as it does the necrosis of the lung tissue, as a most important process, and one not explicable on any doctrine simply of defect of the system, or of any accidental loss of fluid.

We come now to the very important question of the origin of this virus. Supposing it is decided that phthisis is due to a tubercular virus, Where does it come from? Does it come from caseous material in some part of the body, or from the outside? Let us first take the case of other forms of tuberculosis. In general tuberculosis we have undoubted effects of a virus, but as to where it comes from I simply answer that I do not know. One thing is certain, it does not always come from caseous products; in every case of general tuberculosis I have seen, I have examined carefully for caseous products, but I have by no means always found them; and I believe this to be the universal experience of pathologists.

Take again the case of the brain; we meet with a local tuberculosis of the brain, but where the virus comes from I do not know. It certainly does not come from a caseating centre so far as I can discover. Or take the kidney; Dr. Hamilton constructed a theory of its origin in the kidney which I can by no means subscribe to; here again I do not know the origin of the virus. Take again tuberculosis of the joints; this is a most important point. Strumous disease of the joints is undoubtedly tubercular, abundant miliary tubercles being found in the altered structures. In a case I met with recently, a case of Dr. Geo. Buchanan's, there was enormous enlargement of the synovial membrane, so great was it that at first it was doubtful whether it was not a tumour. I found in it tubercles in immense numbers, but not a trace of caseous material. In ordinary cases of strumous disease of the joints you do find caseous material, for the inflammatory processes and the tubercles have produced it, but in this case there was not a trace of caseous material. I do not know where the virus came from in this case either. And now to return to phthisis pulmonalis; the virus, both in the cirrhotic and in the caseous

forms, I think undoubtedly comes from without, and is carried to the lungs mainly by the bronchial tubes.*

In this reference I would venture on some views which may be regarded as too theoretical, but something of this kind seems to be necessary for a proper elucidation of the subject. We cannot accept any theory of phthisis which does not take into account a state of the system as predisposing to it. On my fingers at the present moment are three swellings which I may call pathological spots, due doubtless to the virus acquired at *post-mortem* examinations. The virus has affected the skin, and it or its products have passed up the lymphatics and affected a gland above the elbow, which is enlarged and painful to the touch. The virus or its products have entered the blood, for I am at present generally out of sorts. Every spring I have a recurrence of these pathological spots, and only in spring; and though all winter I make examinations and expose my hands to the virus, it is only in spring that the virus takes effect. There is some state of the system in the spring, perhaps connected with the long work of the session, which renders my skin peculiarly susceptible to the cadaveric virus. And so in phthisis I would say there must be some susceptible state of the system before the virus will take effect. It seems to me that we must acknowledge that this virus is abundantly present in the air, ready to develop itself in a particular state of the system; but I would say this further, neither a state of the system nor a virus alone is enough to account for the effects. That a state of the system is not sufficient to account for tuberculosis is abundantly evident in cases of acute general tuberculosis. I have seen a strong muscular man with no lack of adipose tissue die within a few weeks of this disease; evidently a virus of the most active kind. Dr. M'Call Anderson has referred to a warning that should be given of not accepting pathological theories without a sufficient basis; but I would return a warning, and say that physicians are perhaps liable to take too much the appearances during life into account. And in this reference I would say that pathology undoubtedly points to a virus as the cause of phthisis, and clinical facts point to a state of the system as at the bottom of it. We have to accept both, and my belief is that this position will be that of the immediate future.

* The recent very suggestive remarks of Cohnheim on the probable source of the virus in acute general tuberculosis, and the various local forms, are not specially referred to here. These remarks point to the direction in which future observation may be made, but are not to be taken as a dogmatic statement of ascertained facts.

Dr. Gairdner, from his experience as a pathologist, was interested in the remarks of Dr. Coats. In 1851 he was greatly disabled by the pathological virus. He had at that time a succession of boils—150 or 200, at different parts of his body. After going abroad without much benefit, he was led to give up pathology as a special branch of study. While before that period he was only occasionally and temporarily subject to such attacks, it is remarkable that ever since then, if he merely handled pathological products, he was liable to an occurrence of pathological spots. So that here a special susceptibility to the virus had been induced by the virus itself.

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