

CONDENSED REPORT OF

Lectures
ON

TUBERCULOSIS AND TABES MESENTERICA.

*Delivered before the Hon. the Grocers' Company in the
University of London,*

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LECTURE II.

PULMONARY TUBERCULOSIS.

"SIR JAMES PAGET AND GENTLEMEN,—As it will be impossible for me to give more than a mere sketch of the results at which I have arrived in connexion with pulmonary phthisis, and as the bulk of my observations up to the present have been made upon the lungs of children, I shall draw upon the cases already spoken of for most of my facts, but where necessary and possible I shall refer to phthisis as it occurs in older people. It is now generally accepted that all cases of rapid infective phthisis are the result of the action of the tubercle bacillus, although it cannot be denied that certain lesions may be produced as the result of the action of other and non-specific irritants. In these conditions, however, tubercle is very frequently associated with some other lesions, and it is often extremely difficult to say which changes are due to the one and which to the other. For instance, it has long been held that in stonemason's phthisis all the characteristic lesions met with in chronic tubercular phthisis are present, but my experience of stonemasons' lung has been that along with the chronic interstitial and arterial changes, set up by the stone particles, there are structural alterations which can be accounted for only on the assumption that they are of tubercular origin, and in some few cases, in confirmation of this, the presence of tubercle bacilli has been demonstrated in certain of the new growths. It may be assumed for the present that in all the cases of phthisis the pulmonary lesions to be described are of specific infective origin."

The structures affected by the action of the tubercle bacillus were then briefly described, and it was pointed out that in the first instance we should expect under given conditions the mucous surface of the bronchus to be attacked. Such an assumption might most fairly be made after a consideration of Julius Arnold's observations on the course taken by dust when inhaled into the respiratory passages, Arnold pointing out the very important part played by the walls of the small bronchi and their terminal passages in the disposal of inhaled dust particles. Secondly, there is the alveolar epithelium, which under the influence of any irritant material undergoes proliferation more or less rapid, this in certain cases terminating in what we know as catarrhal pneumonia. Here, again, in proof of the statement, take what may be seen under the microscope, when particles of coloured dust have been inhaled into the lung. The cells lining the alveoli are seen to be in an active state of division; some are still adherent to the wall of the air vesicle, and in these small particles of the pigment may be seen embedded in the protoplasm. The epithelium in this position is a structure which may be attacked by tubercle bacilli, just as in it coal or other particles may be found. Passing still further, and following the course taken by the pigment granules, the lymph spaces around the air vesicles are reached, then the lymphatics in the interstitial and interlobular tissue, the peribronchial and perivascular lymphatics, and lastly the glands at the root of the lung, either directly or by the deep layer of the pleura, over the surface of the lung, and so to the root. As may be seen on reference to a section of coalminer's lung projected on to a screen, the pigment (in this instance a material which gives rise to little irritation) is carried to every part of the lymphatic system, and is seen to have accumulated in very considerable quantities along the lines of the septa, around the bronchi and bloodvessels, and in the deep layer of the

pleura. On microscopic examination, the pigment acting as an irritant, and so giving rise to the formation of a slight excess of fibrous tissue in all these various positions, may also be seen. Lastly, the small points of lymphatic tissue which occur at intervals along the lymph channels, first described by Burdon Sanderson, then by Klein, Arnold, and others, are the seats of pigmentation. Tubercle formation also may be met with in any of these positions. It would seem at first sight to be an easy matter to determine at once in what tissue the tubercle has originated in any special case. In the lung, however, where the tissues are so delicate and so complicated, and where in consequence the changes are so rapid, this is not the case; and it is only in exceptionally favourable cases that the mode of origin and spread can be at all satisfactorily demonstrated. Further, the variety in the life histories of individual tubercular growths at one time rendered it a matter of considerable difficulty to arrive at any definite understanding of tuberculous processes, especially of those associated with pulmonary phthisis. The anatomical structure in the various forms being so absolutely defined in the earlier stages of the growth, it was difficult to bring into a common group forms which differed so widely from one another, not only in naked-eye but in microscopic appearances.

There is now, however, sufficient evidence to justify pathologists in stating that many of those forms which different clinical observers have from time to time described as tuberculous are undoubtedly tubercular in character, from the small grey, gelatinous, or fibroid nodule, to the large caseous masses, leading to cavity formation; and the presence of the specific bacillus has time after time been demonstrated in all these forms, both by staining and by inoculation. There can be little doubt that these forms are essentially the same, and that the differences observed are due firstly to the resisting power of the tissue attacked, and secondly to the numbers and activity of the attacking bacilli. If the behaviour of other tissues under the action of mechanical or micro-organismal irritants be borne in mind, there will be little cause for wonder that there should be these numerous varieties of manifestation of the action of the specific irritant in tuberculous lungs. In connexion with this statement, a matter was insisted upon which has certainly been mentioned, but to which, as a rule, far too little importance is assigned—viz., the intercurrent of suppurative changes, which are evidently set up by the activity of a different micro-organism.

The lecturer observed that Mr. Hare and he were much struck by the fact "that after one micro-organism has completed its task, another may step in and continue the process of breaking down. How frequently a pyæmic condition supervenes on a tubercular. How often has a patient suffering from a tubercular abscess of the kidney or of the lungs succumbed at last (if not carried off by acute tubercular disease) to pyæmia, and pyæmia in which the symptoms are extremely well defined."¹ How frequently localised suppuration steps in to aid in the breaking-down process, more frequently in the lungs and on the intestines than in other positions, because of the greater ease with which organisms giving rise to the irritant material can arrive at and remain on the tubercular surfaces in these organs. Writing on this subject, Coats, in his Lectures to Post-graduates, points out that tubercle is essentially a disease of surfaces and channels, and this is so far true that bacilli can reach the tissues only by such surfaces and channels, and that in these channels there are irritant secretions often containing numerous micro-organisms and other products which assist in completing and hastening the breaking-down process commenced and partially continued by the tubercle bacilli. The actions and interactions in these cases are extremely complicated, and but for the occurrence of more simple cases now and again the observer would be completely lost amongst it all. These facts were mentioned simply to assist in following and accounting for the different changes which occur in the lung during the course of many cases of tubercular phthisis. Of the 100 cases spoken of in the last lecture in which there was tubercle of the mesenteric glands, the mediastinal glands, or the glands at the root of the lung, were also distinctly affected in 69 cases, whilst in 62 cases the lungs themselves were affected. Of these 62, 59 were included under the 69 in which the glands at the root were tubercular, the other three

¹ Pathological Mycology, p. 13.

having developed first simple catarrhal pneumonia, which had later become tubercular in character (in two of these bacilli were found). There were also, as the figures show, 7 cases in which, although the glands at the root were affected, there was no tuberculous process in the lungs. This is important, for when the lung cases as a whole are considered the figures are slightly different. Of 110 cases of tubercle of the lung, the glands at the root were not affected in every case; this was especially noticeable in cases of miliary tubercle and in several cases of catarrhal pneumonia. There were distinct tubercle nodules, and caseation in the glands, except in those cases where there had been adhesion of the lung at any point, or where there had been collapse of the lung. Where these conditions were present—i.e., adhesion, collapse, &c.—the tubercle in the glands at the root was, in a much larger proportion of cases, of more recent date. In the 110 cases there were cavities in 32, caseous masses but no large cavities in 39, racemose tubercle with a tendency to fibroid change in 25, and tubercular catarrhal pneumonia in 15, in each case the character of the predominant lesion only being recorded. The cavities and caseous masses were not, as in the adult, usually confined to the apex, but were, as in monkeys and other animals, scattered throughout the lung—a fact which might be explained on the supposition that in such cases the general predisposing causes of tuberculosis are acting quite apart from those of local origin. Where these local predisposing causes do come into play, the seat of election in children is not at the apex, but at the root of the lung, very frequently posteriorly, and at the base. (Large sections were exhibited.)

The general tuberculous affection of the lung in children appears almost invariably to have associated with it one of three antecedent conditions:—

(a) Simple capillary bronchitis and catarrhal pneumonia, such as very frequently follow or are associated with measles, diphtheria, scarlatina, whooping-cough, and other similar conditions, in which there is a weakened condition and impaired power of resistance of the epithelial cells lining the capillary bronchi, the alveolar passages, and the air vesicles; further, there are those interstitial changes in connexion with the lymphatic network mentioned in the former lecture; and, lastly, there is the irritable (speaking now of the proliferating tissues of the gland) and weakened condition of the glands, both the small collections of glandular tissue along the lines of the lymphatic channels and the larger glands at the root of the lung. Not only is the resisting power less in these conditions, but owing to the large amount of work they have been called upon to do in connexion with the absorption of the catarrhal products, the glands, though enlarged and swollen and evidently acting most vigorously, are unable to respond to any increased demand for work, either in taking up or destroying fresh *materiæ morbi*. A case of tubercular catarrhal pneumonia, following typhoid fever, was then exhibited, in which the greater part of the lung, especially the lower lobe and the lower part of the upper lobe, were consolidated; it was grey in colour throughout, was not firmly consolidated as in croupous pneumonia, but was slightly more spongy in texture. From the cut surface a large quantity of purulent fluid could be squeezed, in which were present very large numbers of tubercle bacilli. On section the air vesicles throughout, but especially around the terminal bronchi, were filled with large proliferating epithelial and catarrhal cells, some in an advanced stage of degeneration, but others containing well-marked bacilli in and around them. Although in this case the interstitial changes were not very prominent, there were nevertheless numerous bacilli in the lymphatics, some free, but many contained in the small round corpuscles found there; and, again, a few bacilli were to be distinguished in the lymph sinuses in the cortex of the glands. In the medullary portion of the gland there was evidently rapid cell proliferation, but little or no caseation. In several other cases of catarrhal pneumonia following diphtheria, scarlet fever, and measles, an earlier stage still could be observed. In these conditions the lobular catarrhal pneumonia is frequently preceded by collapse in certain small areas, the result probably of closure of the bronchus with catarrhal products of the bronchus itself. In these plugs, and in such cases in these only, large numbers of tubercle bacilli were found; they were so numerous that the idea of their having all come originally from some other definite tubercular focus was put out of court entirely, and their presence in such large numbers could only be accounted for on the supposi-

tion that a few had first been introduced into the catarrhal mass, which had formed such an excellent cultivation medium that an almost pure culture of the bacilli in the small bronchi had resulted. In the air vesicles in the immediate neighbourhood, a few bacilli may also occasionally be seen, but in the larger proportion of these cases the bacilli are confined to the lumen of the bronchus. It may be readily understood how in such cases, had the patients lived long enough, the bacilli and their products would have made their way into the lymphatics, and so to the glands; but the process is here extremely rapid, and caseation very speedily supervenes, this apparently being in great measure due to the lowered vitality of the tissues.

(b) Another form of this general tuberculosis of the lung is where there is inhalation of the specific virus from some localised nodule or mass. In adults the most common termination of a case of apical phthisis is brought about by a form of acute tubercular pneumonia in the dependent parts of the lung. The extreme consolidation and rapid caseation in such cases are here also well-marked features of the process. In like manner it is found that in children the general dispersion of the virus often takes place from a small cavity which eventually opens into a bronchus. In three cases the lecturer had observed a second form of primary focus, one not very commonly recognised, but one of which a case is recorded by Dr. Coats in his *Lectures to Practitioners*, p. 170. In two of the cases under consideration the glands at the bifurcation of the trachea were enormously enlarged and distinctly caseous, and at one point they were much softened; just above this softening there had been ulceration into the left bronchus near the bifurcation, and the contents of the softened glands had been practically emptied into the bronchus. As a result of this there were well-marked recent tubercular catarrhal pneumonic patches, swarming with bacilli, throughout the lung, but accompanied by interstitial tubercular changes, although bacilli could be demonstrated as present in the lymphatics. In these cases the catarrhal condition is evidently tuberculous from the first, and the process goes on with extreme rapidity. In the third case, one of the smaller glands at the root of the lung was the infective focus, the ulceration having taken place in one of the larger branches of the left bronchus. In the specimens exhibited the tubercular pneumonia was pretty regularly distributed in connexion with the branches of the bronchus beyond the point of ulceration. A third form of ulceration met with and giving rise to a disseminated catarrhal tubercular process, is that where the wall of the bronchus itself is tubercular, and where in consequence there is ulceration and setting free of the infective material in smaller or larger quantities. This form is very frequently met with, and is a cause of tubercular catarrhal pneumonia in a very large number of cases.

(c) The third of the antecedent conditions of general catarrhal tubercular pneumonia is one which has already been touched on—i.e., that condition in which the lymphatic glands at the root of the lung and the mediastinal glands are practically inactive. This, of course, occurs specially when the tubercular glands are caseous, but it may also occur in other conditions, where the glands are either altered in structure or are choked (if such a term could be used) by the presence of foreign particles. In these cases, where the nutrition of the tissues is greatly impaired, general catarrhal tubercle is very frequently met with, but it is then almost invariably associated with chronic or more recent interstitial tubercle.

In localised tubercle the lymphatics seem to play a much more prominent part than they do in the general form, though here, again, the two forms, lymphatic and catarrhal, are so closely associated that it is difficult to say where the one ends and the other begins. The seat of election of tubercle in children is not at the apex, but near the root of the lung, posteriorly, at the free margins (in patches) or at the base. In those cases of tubercle at the root, the predisposing cause is, as has been pointed out, the tuberculous condition of the gland. The lymph stream in the lung cannot be looked upon as going constantly in any one direction, on account of the very free lymphatic anastomoses there are in the various parts of the lung, and through this free anastomosis an area may be drained by another set of lymphatic vessels, even though its own proper vessels are occluded. Thus when a gland at the root of the lung becomes functionally inactive from its conversion into caseous tubercle, the lymphatics going directly to it from

an area in the immediate neighbourhood are obstructed, and there are both impaired nutrition in and diminished excretion from this part. Although, however, the tissues in this area immediately around the gland have their lymph supply altered, the tissues outside the localised area are drained into the lymphatics (1) of the glands corresponding to these areas, and (2) into those which eventually find their way into the deep layer of the pleura, and so to the root of the lung; and it appears probable that in consequence of this "backward flow" the affected area may in certain cases give rise to a further extension outwards. Tubercle at the posterior part of the lung and at the free margins is to be associated with obstruction and collapse, conditions so frequently met with in children and so common a cause of simple catarrhal pneumonia. The essential conditions necessary for tubercular catarrh and impaired lymph discharge are present, and the bacilli, left at rest, develop very rapidly in the air vesicles, or more slowly in the lymph spaces in the interstitial tissue. Tubercle at the base of the lung is found especially in those cases where there is pleurisy, or in cases of peritonitis, tubercular or not. Of the cases analysed (127), the liver or spleen, or both, were adherent to the under surface of the diaphragm in 65 cases, most frequently as the result of old peritonitis, now without a trace of tuberculous structure remaining; but in 17 recent tubercular peritonitis was well marked. In a very considerable number of cases where old pleurisy was present, the base of the lung was markedly tubercular. In 4 instances the lower part of the lower lobe was transformed into a solid caseous mass, whilst in the fibrous tissue of which the adhesion was composed there were well-marked tubercular nodules, some quite young, but others in an advanced stage of caseation. In these cases the extension is, in the first instance, by the lymphatics, and then perhaps to the epithelium; but the predisposing cause appears to be the state of rest induced by the adhesions of the organs of the abdomen and thorax to the opposite surfaces of the diaphragm, leading to imperfect lymphatic circulation and exchange.

In the case of tubercular peritonitis, in which caseation occurs at an early stage, it might reasonably be expected that tubercular pleurisy and mediastinal tubercle would be rapidly developed. Professor Klein and Dr. Wm. Hunter have both laid great stress upon the fact that foreign materials which find their way into the abdomen are soon passed on to the under surface of the diaphragm; and Dr. Hunter, in his experiments on the absorption of injected blood from the abdomen, demonstrated most clearly the presence of blood-corpuscles, comparatively little altered, first between the liver and the diaphragm, and secondly in the lymphatics of the diaphragm. The bearing of this on the adhesions of the liver to the diaphragm are exceedingly important, as are also Klein's observations on the paths taken by foreign particles after passing from the abdomen. Bearing these facts in mind, it may be easily understood how it is that tuberculosis of the mediastinal glands and tubercular pleurisy of the costal pleura are both so frequently associated with tubercular peritonitis. The glands, too, at the root of the lung frequently (as seen in so many of the specimens) become caseous before the lung itself is affected; in some cases, no doubt, this is due to a process similar to that described as occurring in the mesenteric glands, where it is accompanied by no permanent lesion in the intestine; but in other cases it seems to be equally beyond question that the specific virus has passed (*a*) from the mesenteric and retro-peritoneal glands, and (*b*) from the peritoneal cavity through the central part of the diaphragm and the broad ligament of the lung, or (*c*) by a more or less circuitous route along the lymphatics of the parietal pleura to the root of the lung. It is worthy of note that until pleurisy is set up in these cases there is no transmission from pleura to pleura, but that as soon as the slightest adhesion takes place there may be continuation of the process on the two surfaces. It should be observed, however, that in addition to this affection of the costal pleura the visceral pleura may also be the seat of tubercular nodules, the virus in this case having come probably from the abdominal cavity by the diaphragm and the broad ligament. In all these forms there is abundant evidence of the transmission from point to point of the virus by way of the lymphatic channels, especially where the tissues generally are highly resistant, and where the epithelial cells, though they do not arrest the passage of the bacilli into the lymph spaces have

still sufficient vitality to continue to grow in a more or less regular manner. In such cases the connective tissue resistance is also great, and though the bacilli may still continue to grow and to attack the cells in their immediate neighbourhood, those cells outside the immediate sphere of action of the irritant are stimulated into proliferation and fibrous tissue formation, so that a fibroid capsule is formed around the cellular or caseating centre. When the epithelium itself is attacked caseation rapidly ensues, and absorption from this point may take place for some distance along the lymphatics. In consequence of this method of spreading by the lymphatics of the lung, nodules may be sought in those positions in which coal pigment is found to accumulate, the only difference observable being that in tubercle the nodules are usually somewhat limited in their area of distribution, the pigment, on the other hand, being disseminated over the whole lymphatic area. The changes around the vessels and in the bronchi are marked by no special features; the tubercles are formed in connexion with the peribronchial and peri-vascular lymphatics, and in some cases, as has been observed, they appear to be formed in the small lymphoid nodules which may be seen in the walls of the lymphatic vessels.

In the intima of the vessels, as Cornil and Ranvier, Hübner, Greenfield, Hamilton, and others have insisted, the changes are extremely well marked, and quite recently attention has been called to the fact that even some of the systemic arteries may be deeply affected with arteritis obliterans in cases of chronic phthisis. How far the changes in the intima are associated with those in the adventitia is as yet not fully decided, but there seems every reason to hold, with Arnold, that wherever the lymphatic circulation in the adventitia is disturbed—especially where there is irritation and proliferation of the endothelial cells of the lymph spaces—corresponding changes are met with in the intima, particularly where the process is chronic in character. In the specimens of lungs exhibited, this change in the intima is a very marked feature.

It was then mentioned that acute miliary tuberculosis must be looked upon as the result of spreading of the infective material directly by the blood channels. The demonstration of this fact was first accomplished by Weigert, who, in a series of several cases of acute miliary tuberculosis, was able to determine the presence of ulceration of the pulmonary vein. The process being similar to that in or near the wall of a bronchus in the cases mentioned, Ponfick had first supposed that the bacilli might pass from a tubercular thoracic duct into the venous trunks, and thus to the general circulation. It is probable that both observers were correct, and that both forms may occur. Coats further points out that a limited distribution of tubercle by the blood may be due to the passage of bacilli into the minute venous radicles in the glands in which tuberculous changes are occurring. That bacilli are found in the blood has been now frequently demonstrated, and quite recently several cases have been recorded in which general tuberculosis has come on after hæmorrhages in patients suffering from apical phthisis. This is a matter of all the greater interest when it is borne in mind that all these cases of acute tuberculosis were developed in from seventeen to twenty-five days, just the period given by Koch as that required for the development of tuberculosis when produced experimentally. The importance of this can scarcely be over-estimated from a surgical point of view, indicating as it does the methods of procedure to be adopted in operating on any tuberculous part. The bacilli, though found in the blood in such cases, do not become active until they come to some part of the circulation at which they can make their way into the surrounding tissues. In some cases bacilli are present in the emboli, or they may be actually distributed in the embolic area, in many cases appearing to make their way from the capillary vessels into the lymph spaces, and only then giving rise to the characteristic series of changes.

It was long unfortunate (but natural), for the sake of prognosis, that only the worst cases were seen in the post-mortem room, but in the present day tubercle has come to be looked upon as a comparatively curable disease. After some experience in the post-mortem theatres of a large general hospital and a children's hospital, the lecturer felt convinced that the recent change which has come over medical opinion as regards the curability of phthisis in the early stage is thoroughly justified by facts. In proof of this

there are the numerous localised fibroid and deeply pigmented bands of tissue seen in the lungs of old people, or people who have died during middle life, sometimes without caseous or calcareous nodules in the centre, but perhaps more frequently with one or other of these marking the centre of the cicatrix, the puckered pleura near the apex of the lung also marking a considerable loss of substance at some period during life. In children this is not found in nearly such a large proportion of cases, but well-marked examples may be met with even in very early life, and pretty frequently before the eighth year.

This is so far encouraging, and entitles us to hope that, as more facts concerning the life history of the bacillus tuberculosis and the conditions under which it may flourish in the body are gathered, the death-rate from tuberculosis may be materially diminished. Milk, air, and food can one and all convey the bacillus from cattle or swine to patient, or from patient to patient; and if the bacillus or the disease can be successfully attacked in any one of these, a possible source of infection to others is done away with. From clinical experience it must now be concluded that the general health of the patient has in all cases much to do with the resisting powers of the tissues, so it is imperative on every medical man to try to improve the general health of those of his patients having a tendency to scrofula or a tubercular family history. Children of low vitality are scrofulous because the introduction of a comparatively small number of bacilli brings about complete degeneration of the lymphatic glands, and there are no giant cells and few bacilli found in a scrofulous gland, not because of any change in the nature of the bacilli, but because of the difference (non-resistance) of the tissues in which they grow. The number of bacilli attacking a healthy gland would be rapidly disposed of; but in the delicate child, with its weakly tissues and imperfect nutritive and excretory power, the gland tissue gives way on the slightest stimulation, and the cold abscess is the result. The lecturer was convinced from the experiments he had performed that this was the case. Bacilli differ in number, but not in character; and if once a cultivation can be obtained from a cold abscess (a somewhat difficult matter), well-marked tuberculosis may be produced by it by inoculation.

The question of the structure and importance of giant cells was then touched on. Each authority, it was pointed out, had his own theory about the nature of these structures. It was now becoming evident that each of many of these observers, though describing different conditions, might still claim right on his side. Those who advocated that the giant cell was a lymph space with proliferating endothelial cells around are apparently justified. Then, again, Weigert has proved that a giant cell is nothing more in some cases than a collection of cells in which bacilli are causing proliferation at the margin, fusion and degeneration in the centre, a mass of caseous material in the centre and proliferating cells with bacilli between them at the periphery resulting. Klein saw the giant cells being formed by the fusion of epithelial cells of the air vesicles. Small bloodvessels in transverse section have, like the lymphatics, been described as giving rise to giant cells. Dr. Barrett finds them in the seminiferous tubules in tubercle of the testicle, and the lecturer had seen them developed in connexion with minute bile ducts in the liver, and in the milk ducts and acini in the mammary gland of the cow. In all cases the process Weigert describes occurs, but at different rates and with slightly varying results. The presence of these giant cells affords evidence that the cells are making a determined resistance against the advances of the bacilli, are giving way slowly, and so limiting the area of caseation. In many cases where the giant cells with their rings of nuclei are best marked, very few bacilli are to be found, as they have been destroyed by the phagocytes at the margin—i.e., the active cells with deeply stained nuclei. In other cases, however, the bacilli have taken the place of the nuclei at the margin of the giant cell, the boundary line in such cases being determined for a time by the basement membrane of the tube in which the mass is formed.

PRESENTATION.—Dr. Philip Addis of Iver has been presented with a testimonial by his friends and others, as a token of respect and gratitude for his fourteen years' gratuitous services to the Iver, Langley, and Denham Cottage Hospital, with a handsome silver tea set, a silver salver, and the sum of £93, which was left after the purchase of the above articles.

ABSTRACT OF A

Post-Graduate Lecture

ON THE

PATHOLOGY AND TREATMENT OF
THE ENLARGED PROSTATE.

BY REGINALD HARRISON, F.R.C.S.,

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GENTLEMEN,—In bringing this subject before you, I would ask you to observe that I purposely avoid speaking of the prostate as a gland, as I consider such a term inappropriate to a part where, so far as function is concerned, the secreting element is subservient to the muscular. As I have recently discussed this subject at considerable length in my Lettsomian Lectures, I shall confine myself as closely as possible to those points in pathology which it is necessary to make prominent for clinical purposes. If we sum up our experience as practitioners relative to enlargement of the prostate as observed in advancing years, I do not think we shall find much difficulty in recognising that this physical change exists under two conditions which are sufficiently well marked. Whatever may be the proportion of males over sixty years of age who experience some degree of enlargement of the prostate, the evidence appears tolerably conclusive that it is only the minority of this number who develop symptoms which can be regarded as evidence of disease. Hence we may divide persons who have large prostates into two classes: (1) those who do not suffer from them, and (2) those who do.

Taking the former first, I have for a number of years carefully watched persons who had large prostates, but were not aware of it themselves from any circumstances which might tend to suggest it. In many instances the discovery was made, as it were, quite accidentally. In addition to evidence of this kind, I have met with numerous instances where post-mortem examination revealed the presence of a considerable prostate, though no symptoms previously existed. Facts such as these seemed to suggest that the enlarged prostate had come in for much uncalled-for abuse, and that, like other hypertrophies in the body, it might be serving a useful but hitherto unrecognised purpose. Passing to the second class of cases, it was equally evident that there existed a considerable proportion of instances of prostatic enlargement which were attended with most distressing symptoms of vesical obstruction and irritation. The contrast between these two classes of cases, which did not appear to be necessarily transitional, was so marked as to almost suggest in itself some physical alteration in the part to account for the difference. Without going further into detail, my examinations during life and after death led me to the conclusion that so long as the prostate retained its natural structure, it did not seem to matter much, so far as its function was concerned, what size it attained. On the other hand, when it underwent degenerative changes which reduced it to little else than a mass of fibrous tissue in the form of lobulated, nipple-like, or interstitial tumours, it was pretty certain to excite varying degrees of irritation.

The next points that naturally arise are: First, how is it in some instances that the prostate, though increased in bulk, still remains throughout life histologically and functionally normal? And, secondly, under what circumstances does it pass into the condition of a fibroma, and produce symptoms of obstruction and cystitis?

In reference to the first point, I would remark that the human body furnishes us with undoubted instances of hypertrophies, proving themselves to be not only necessary, but precisely compensatory relative to what is required. If, as I have urged, the chief function of the prostate consists in providing a retentive as well as a supporting apparatus for the contents of the bladder, there is no reason, when the time comes for substituting quantity for quality, why the provision should not prove to be permanently compensatory. The conditions under which muscular hypertrophy exists, as