HARVEIAN LECTURES

ON THE

PROGNOSIS AND TREATMENT OF CHRONIC DISEASES OF THE CHEST IN RELATION TO MODERN PATHOLOGY.

Delivered at the Harveian Society of London.

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LECTURE I.—(Concluded.)

Inflammation and localisation of disease are, then, the two propositions at which we have arrived in the teaching of the modern school. Phthisis is constitutionally a chronic inflammatory disease with intercurrent simple forms of inflammation, which, in some cases, heal by cicatrization. Miliary tubercle is an occasional and secondary element, which never heals, but undergoes only necrotic changes. Scrofulous inflammation consists in an abundant proliferation of cells pressed together in glands, which die, and the contents become caseous and form infecting foci which develop phthisical diseases.

The well-known experiments of Villemin, by which he produced tubercle in certain animals (the rodentia) by the inoculation of tuberculous matters, have raised the question whether a like result cannot be produced in man. But the tubercle so formed is not identical with human tubercle; and septic matters of any foreign origin, so introduced, will produce a similar product. It is believed, then, that scrofulous persons are only self-infecting in the mode I have described.

A remarkable series of experiments, however, bearing on this question have lately been published in Virchow's *Archiv*, and were made by Tappeiner. His object was to ascertain whether the poison of scrofula is identical with that of phthisis. He caused dogs to inhale scrofulous matter taken from a boy in atomised spray daily for a quarter of an hour for about ten days. He also performed a similar experiment on other dogs, substituting for the pus from a scrofulous gland the sputa of a phthisical person. The animals were killed twenty-three days later. In the two dogs treated with phthisical sputa, tubercular nodules were found in both lungs and spleen, their nature being verified by microscopical examination. The dogs treated with gland-pus were found free from tubercle, as were also two other dogs similarly treated, and killed on the 29th day. Other dogs treated with the sputa from simple chronic bronchitis were found to be free from tubercle on the twenty-eighth day. The period of incubation, from the commencement of the inhalation until tubercles were distinct in the lungs, was also investigated. One dog was killed on the thirteenth day after, and no tubercle was found, although he had coughed, and wasted three-quarters of a pound in weight. In another dog, killed on the nineteenth day of the experiment, no tubercle was found. In others so treated, and killed after three months, abundant tubercles were found in lungs and pleura. Tappeiner concludes that the inhalation of phthisical sputa by dogs produces tubercle in the lungs; that the incubation period is more than nineteen, and under twenty-three, days; that the inhalation of scrofulous-caseous pus produces no infection; nor does the sputa of chronic bronchitis. As has been remarked by the *Lancet*, from which I borrow this notice, "these observations constitute experimental confirmation of the infection of phthisis; but the experiments have hitherto been too few to permit of our accepting the doctrines as proved.

Up to the present time, however, we are without any evidence which proves that phthisis is communicable, in the ordinary sense, from one individual to another. My own experience decidedly negatives the idea. I believe that all the cases adduced of communication from husband to wife, or from the sick to their near attendants, are to be included in the category of phthisis arising in the ordinary way. In the course of about twenty-seven years at the Consumption Hospital, we have only had two cases of phthisis among the class of attendants: one, an assistant-physician, who was phthisical before he joined the staff; and a nurse, who was ill when she came to our service at the hospital. Heredity, and the influence of depressing causes, and the occurrence of the usual antecedents of consumption, account for all the cases adduced. One of my colleagues, Dr. R. Thompson, has recently advocated another view, and believes that the phthisis he has seen so produced has characters belonging rather to septic pyæmia than to consumption. It is right that such inquiries should be made, and the subject minutely

investigated; but as yet I cannot accept the doctrine. It is closely connected with the theory of phthisis which we have been discussing—namely, its origin in infecting material, and the influence of primary scrofulous inflammations as infecting centres from which secondary results in distant parts originate.

If pulmonary phthisis be, then, a compound of scrofulous inflammation and tubercular new growths, in what forms recognisable to our senses shall we find them? 1. A chronic pneumonia of one apex, with much dulness, slight rales, shrinking walls, slight fever, possibly occurring in the rheumatic constitution, and not by heredity. This may run on to cure, or become very chronic. 2. Bronchopneumonia, or caseous pneumonia, which consists of nodular foci in the lungs. Here there has been wasting and fever before any discoverable physical signs. It is hereditary in most instances; and from the proneness to degeneration and death of caseous matters, may rapidly run into cavity. 3. Catarrh of the apex; crackling rales in the subclavian or supraspinous region; dulness slight and diffused; possibly an early hæmoptysis; certainly early fever and wasting. A neglected catarrhal bronchitis has originated this. The anatomy would give a cellular infiltration of the pulmonary connective tissue of catarrhal origin, but with inflammatory products. Tubercles of recent formation are found around these. In the process of degeneration, the albuminoid substances first become soluble, absorb water from the surrounding tissues, and melt; and so a central cavity is formed. The consecutive events are secondary infiltrations of other portions of the lungs, which are properly infections, the morbid matters being carried by vessels and lymphatics, and deposited in other parts of the same or of the opposite lung. This is ordinary progressive advancing phthisis. The fever is high, for the blood is constantly charged with the detritus of inflammatory and tubercular products. Local morbid processes manufacture the fever; for febrile substances, the result of their necrosis, are constantly being added to the blood. Hence the high temperature and the waste of tissue or marasmus; for nutrition is spoilt by the same process, and a blood laden with morbid detritus is unfit for the ordinary nourishment of the body. I ask you to bear this in mind: *how local disease, itself originated by a local infection, becomes the workshop in which is elaborated fresh morbid material for new infections.* Till such centres of manufacture are exhausted, the morbid process continues; but if it be stopped for want of pabulum or material (and it seldom stops from other causes) the disease has no check nor limit. This explains many of the phenomena of chronic lung-disease, and will be found to have an important bearing on treatment.

I must diverge here, as the German pathologists always do diverge, to consider the question of apex-disease. At first sight, it seems trivial to ask why the apex is the part earliest affected in phthisis? but it is worth examining. Catarrh of the apex, pneumonia of the apex, mean phthisis. The same signs located at the base have a different import. It may be said that localised physical signs in any portion of the lung, —I mean limited in extent and pronounced in character—have a more ominous meaning than signs of consolidation or catarrh spread over one or both lungs. But why is the apex first affected? It is replied that the immobility of this part, as compared with other portions of lung favours the retention of secretions, which thus inoculate surrounding tissues. These apex-movements are limited by the position, and by the frequent adhesions of the pleura at this part. Mechanically, also, it is more difficult to empty this part of the lung. Again, it is said, that less blood circulates in the apices; and, further, that as the lung is fixed at its root, and the apices lie at a higher level, the expanding influence of the diaphragm is less felt at those parts. The inference is, that all that retards movement, and impedes expansion, and diminishes vascular movement in a portion of lung, makes it more likely to become the seat of chronic induration. In these investigations, we see again how local changes and the influence of locality have come to be considered important in the study of chronic disease.

Now, it will be very evident to those who have followed me that, in describing the effects of inflammation of the lung, and in dividing pneumonia into the "croupous" and "catarrhal", I have limited the possibility of complete restoration of the lung to the "croupous" form in its well pronounced acute variety; and that, in describing the changes which occur in the "catarrhal" form, I have been painting the events of chronic phthisis. Taking the facts to be so—that there is an acute variety of inflammatory disease, tending, in the vast majority of cases, to recovery; and another variety, commencing also by an attack more or less acute, but which inevitably destroys some textures of the lung, and leaves a permanent mark, as it were, with degenerations tending to the destruction of lung-tissue and secondary congestion in sequence—and as it is evident that their ordinary progress is in different directions, we have inquired, In what consists this most important difference? and what common features do they bear which justify us in including them under the same name of inflammation? They have diverging patho-

logical results; the one tends to degenerations which remove the new product most rapidly from the system, the other to degenerations which ulcerate and destroy the cell-walls and surrounding tissues, or harden them and convert them into fibrous structures of low vascularity.

Acknowledging that inflammation—that is, that the inflammatory products of congestion—play a large part in the history of chronic diseases of the lung, and influence the result more than any other agents, I still feel that the name “catarrhal pneumonia” has been unfortunate, as tending to mislead those indifferently acquainted with the minute pathology of the day, and to induce the practitioner to predicate results for his patient which may never occur. To the word “catarrhal” I do not object; for it fairly expresses the product of the attack in the earlier stages, when it is limited to the interior of the air-cells, the walls remaining intact; but the term “pneumonia”, I conceive, had better be confined to that sthenic form of the croupous variety, to which alone it was applied before the introduction of the modern German pathology.

It appears to me that the terms croupous and catarrhal have arisen to supply the place of the word “tubercle”. It has long been known that there may be phthisis without any tubercle, such as Laennec or Bayle described, whether miliary and caseous, infiltrated or scattered, whether acute or chronic. The acute tuberculosis, indeed, is still acknowledged to be tubercular; and it is seen and known that tubercles may be developed in the course of chronic phthisis, but are then a mere incident in the story of the disease. But while tubercles—small scattered bodies, miliary or aggregate—are found not only in the lung, but in other organs and in the membranes of the body, and are daily seen without any microscope, and such appearances are associated with a train of symptoms indicating destructive degenerations, it is impossible to deny them a place in our pathological category. It is histology, chiefly, which has displaced the word “tubercle”, and the impossibility of defining it under the microscope, or recognising it by invariable and peculiar characters, which has led to the necessity of new terms to express the product of disease which leads to consumption. One thing is clear: that, whereas in the early stages of an attack the products of inflammation and the part of the lung-structure affected are nearly identical in the two forms of pneumonia, in the more advanced stages, even long before softening occurs—indeed, we may say in all its subsequent pathological events, catarrhal pneumonia differs from croupous as much as any two disorders can do affecting the same organ.

Perhaps the most important structural change in all chronic diseases of the lung, is that which results in fibroid alteration; for it influences the course, the symptoms, the duration, and the result of every such affection. It would appear that this growth is not dependent on vascularity, but is almost extravascular. The very minute capillary circulation is not necessary to all vital tissues. This may be seen in fibro-cartilage. The subsistence of fibrous tissue is placed low in the vital scale; and it appears to originate when the flush of inflammation is over and rapid degenerations have settled down and removed the debris of inflammatory products.

In the lung, it would appear that, when the growth which we have described, in the alveolar walls and in the interlobular tissue, has not degenerated so as to break down into purely ulcerative and necrotic processes, the result of necrosis from obliterated vessels, a lymphatic increase of interlobular connective tissue occurs, along with a cellular thickening of the old walls. This tissue, in which blood-vessels are not found, extends between the air-cells and between the lobules, and spreads its connections till it reaches the pleura, to which it becomes attached. The changes which the lung undergoes in this process of fibrous conversion are very characteristic. The lung itself is contracted; not only in parts, but as the new growth extends, the whole lung occupies less space; and, from its connection with the pleura, the chest-walls are found also contracted—so that a considerable diminution in the cubic contents of the pleural sac occurs, and a very manifest contraction of the side. This contraction is so characteristic, that it is evident at a glance. The effects of this extension of fibrous tissue through the lung is to cause obliteration of vessels and extinction of air-cells in its tract, by contraction of their walls. Very little blood circulates through a lung in which this process has much extended, and but little aeration takes place; for respiratory cubic space has greatly diminished. The bronchial tubes often undergo dilatation, and afford physical signs mimicking those of ordinary cavities in the lung. Their dilatation may, perhaps, be accounted for by a loss of elasticity in their walls, and of support from the surrounding tissues. It is conceivable, also, that the inspiratory force is more expended on them, as the alveoli lose their capacity for expansion. The pleura has always shared in these fibroid changes in old cases, and has become closely attached to the lung. From such physical condition results that extreme fixity of the chest-walls characteristic of chronic phthisis. The vital results

on the lung are most important. That organ is deprived of a large part of its circulation; and, in fact, its capillary circulation has almost ceased. Hæmoptysis is a rare event, for the larger vessels are strangled. Respiration scarcely proceeds, for many air-cells are annihilated; and, there being little expansion, a proportionately small quantity of air enters the lung. The few remaining parts of the lung may expand, and become emphysematous, perhaps, for the same reason that the bronchi have dilated, namely, impaired elasticity of the walls, and the expanding forces more felt. The physiological vital blood-changes depending on the lung diminish as breath-space is lessened. Deficient aëration of blood leads to lividity of the terminal vascular parts, and new growth of low fibroid tissues is seen in the fingers and toes, where clubbing occurs from new growth. Following the mechanical contraction of the lung, the heart is drawn up or down; the diaphragm is raised; the intercostal spaces are drawn in; and the opposite lung crosses the middle line. I am picturing a rather extreme case; but all chronic cases possess, more or less, the same features, and they are dependent on the growth of fibrous tissue in the interlobular structures of the lung. I would ask you to remember that the results are vascular stagnation, from compression and extinction of vessels; and contraction, with comparative immobility of the lung and of the side. Every case of phthisis which becomes chronic presents such alteration of structures more or less marked. It is the inevitable condition of chronicity; that is, of every case which has outlived the first destructive degenerative changes which take place in the early history of lung-disease. There is no such thing as fibrous phthisis; that is, a phthisis which originates in the formation of a new fibroid product in a lung hitherto healthy; which is in itself a *materies morbi*, and in its changes destroys the lung. For what is, and where is found, this fibroid formation? It is a change always secondary to that many-celled invasion of the alveolar walls and interalveolar tissue, which I have been describing. It is, in fact, a conversion of that tissue, resulting from its previous invasion by this nucleated growth; and it is found, not as a product of inflammation, but as a formation of new and extending tissue, replacing and originating in the proliferations resulting from inflammatory action. It differs from them, in that it is slow to degenerate; it amalgamates and alters other surrounding tissues into its own growth, which is ever spreading and involving new portions of lung in firm elastic, contractile extensions; while inflammatory products present rapid degenerations, fatty and caseous. This fibrous element gives chronicity to all old cases; and is, in fact, the conservative element of chronic phthisis. It is a barrier against hæmorrhages, and against ulcerations, and is the safeguard of the patient. The local condition of a lung, so bound down in its circumference to the pleura, with thick bands of tissue passing through it in all directions, is that of a comparatively inert organ. Such a patient neither dies of hæmorrhage, nor is very liable to pneumothorax; nor does he waste from the hectic of an irritated lung, so long as his affection be one-sided. The secondary infections are also delayed, and that ulceration of the ileum, leading to diarrhoea, so common in advanced phthisis, and which may or may not be of septic origin, is of rare occurrence. The blood has, in fact, ceased to be loaded with febrile material; for the degenerative processes have stopped, and the temperature remains normal.

I have thus far sketched for you, gentlemen, the leading pathologic changes in acute inflammation in the lung, and in the chronic variety which lead to destructive changes or to fibroid alterations. Some of the other events in chronic lung-affections will next occupy us, when I shall have less to say of anatomical and histological changes, and more of the clinical events of disease; but, for to-night, I would close by summarising the propositions which I have endeavoured to lay down.

1. Inflammatory products form a large part of that which is found in chronic lung-affections, excluding cancer, syphilis, and pure cirrhosis.
2. Acute inflammatory croupous pneumonia, which is the result of an extreme vascular congestion, has its seat mainly within the alveoli or air-cells of the lung. The alveolar walls are intact, as is also the interalveolar tissue in which the vessels of the lung are contained.
3. Inflammatory croupous pneumonia become chronic means more than this; the alveolar walls have become invaded, and the interalveolar tissues, with more or less strangulation of vessels.
4. The integrity of the alveolar walls is a chief element in all lung-affections, acute and chronic. Where such integrity is maintained, recovery is sure to take place; but where the walls have become invaded by small-celled growth, disintegration occurs, and permanent loss of elasticity and resulting collapse of the air-cells takes place.
5. The phenomena called degenerative result from the death of the parts, their vascular supply having been strangled. The formation of cavity, small or great, is, therefore, due to the necrosis or death of the tissues, deprived of their blood-supply.

6. In so-called catarrhal pneumonia, the phenomena ultimately involve a new growth in the alveolar walls and interlobular spaces, affecting the vascular supply of those tissues.

7. A further necrosis of the alveolar walls, and of the new growth surrounding the vessels and involving the neighbouring parts, may lead to cavity in the lung.

8. A cretification may occur, the products being imbedded in the lung.

9. Or a new development of fibrous tissue may take place, deficient in vascularity, and of low vitality, but contractile and capable of extension, involving all tissues of the lung and also the pleura.

10. A resulting contraction of the lung and of the whole side may take place, followed by displacement of viscera.

11. These fibrous alterations in the lung-tissue are an inevitable feature of all chronic lung-affections, and influence their progress and termination more than any other factors in such diseases. As a leading feature in any case, their influence is conservative, and tends to chronicity and prolongation, retarding and counteracting the more serious events of hæmorrhage and secondary septic infections.

There are one or two points which suggest themselves from the considerations I have brought before you.

If chronic phthisis arise out of inflammatory products in the lung, and not from a special and peculiar deposit—the result of a diseased constitution, or a previously existing condition, antecedent in point of time, to all local disorder—is it to be considered a local or a constitutional affection? If local, it belongs to the class of inflammations; or, at least, it is a specific inflammation.

Another and most practical question is, whether catarrh in its ordinary form leads to phthisis? As it has been ascertained that catarrhal pneumonia, which seems, indeed, undistinguishable from phthisis, originates in a catarrh of the minutest bronchi, which has become localised. If this question be answered in the affirmative, we should find innumerable cases of ulcerative disease of the lung arising out of that capillary bronchitis which affects the minutest bronchioles. But I believe it may be clinically asserted that such sequence is a very rare event—so rare, indeed, as to be exceptional. While most cases of phthisis arise out of a gradual, almost unperceived, disorder of health, in which the patient can scarcely say whether the weakness or the cough was the earlier symptom. It is quite certain, as Louis has observed, and every other authority has confirmed, that it is most rare that any patient can refer the first beginnings of his cough to a cold-catching or exposure well within his notice.

Again, such catarrhal colds affect large tracts of both lungs when they become bronchitis; and the essential character of phthisis is localisation—one apex—one limited tract of lung.

This opens the way to much deeper considerations, which I shall take leave to bring before you at my next lecture.

WOLVERHAMPTON.—The report for this borough is for the twelve months, ended September last. This is to be regretted, as the statistics of Wolverhampton cannot be properly compared with those of other large towns. The death-rate during the period reported on, was 21 per 1,000, or, deducting deaths not belonging to the borough, 20 per 1,000. Of the 1,640 deaths, no less than 726 were of children under five years of age, and 491 of infants under one year. Mr. Love seems to think that the sanitary authority can do nothing to reduce this terrible proportion, though the uninhabitable nature of the dwellings in some parts of the borough, would seem to point to the adoption of the Artizan's Dwellings Act, as a partial remedy for the evil. He, Mr. Love, whilst admitting that errors of diet and delicacy of infants, have their place in causing infantile mortality, feels "constrained to add, that it may in the main be traced to the scourge of this country, drunkenness; for where do we find premature birth, marasmus, inanition, atrophy, diarrhoea, and convulsions most prevalent, but in the wretched squalid hovels of the drunkard, the dissipated, and the indolent?" The number of deaths from "preventable" disease is given as 46, though we find 196 deaths referred to the zymotic class, including 101 from diarrhoea. Scarlatina was fatal in 31 cases; the agency of schools in the propagation of the disease having been especially noticed. The mortality from measles has largely diminished, and typhoid fever was fatal in only two cases. Phthisis and chest affections again contributed the largest quota of deaths, 491, and were especially fatal to young children and old persons. The sanitary work of the borough seems to be pushed rapidly forward, and if we may judge from this prefatory report, there is an unusually enlightened sanitary committee in the town.

ON CERTAIN POINTS IN THE PATHOLOGY AND TREATMENT OF INDIGESTION.*

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THE subject of indigestion is so very wide, that I cannot take up more than one or two points connected with it in the present paper. There are few subjects in medicine of greater practical importance; for there are few that come more frequently under the notice of the medical man, that cause greater annoyance to the patient, or are more satisfactory to treat—at all events, in the earlier stages. I have no doubt that many things which I may say in this paper will be quite familiar to you all; and what I hope to do is not so much to give you new observations on the symptoms of indigestion, or new methods of treatment, as to discuss the causes of the symptoms and the results of the treatment in such a way as, perhaps, to render the treatment more systematic.

By digestion we mean the solution and absorption of the food, and the rejection of the excreta, in such a way as properly to nourish the body; and by indigestion we understand the non-solution or mal-assimilation of the food, and the imperfect removal of its waste products. Digestion begins in the mouth; and, if we retain farinaceous food in the mouth for a sufficiently long time, the starch will be converted into soluble sugar, and will be dissolved. But it is rare for us to allow the food to remain in our mouths long enough for any solution to take place, excepting in the cases of such very soluble substances as salt or sugar. The saliva, which is secreted by the mouth, performs most of its solvent action in the stomach, after it has been swallowed. We might, therefore, look upon the stomach, and not the mouth, as the first part of the intestinal canal where digestion takes place. But, if digestion begins in the stomach, indigestion very often begins in the mouth; for it is in the latter that one of the most important parts of the solvent process takes place—namely, that of mastication. The digestive juices act but very slowly upon large masses either of starch or of albuminous food, though they will attack those substances readily if they are completely broken up. Most of us, I dare say, remember from our boyish days how long a bull's eye of sugar takes to dissolve in the mouth, so long as it remains whole; and how quickly it disappeared after we had once yielded to the temptation to crush it between our teeth. And what is true of sugar is also true of bread and of meat.

Why should bread fresh from the oven be indigestible, and the same bread, after it has become stale, perfectly wholesome? I think you will readily see the reason by crushing a piece of each between your fingers. You will find that the new bread simply forms a toughish mass, while the stale crumbles into a fine powder. Dip a piece of each into water, and the new bread will only take up a little, while the stale sucks it up like a sponge. These two properties of the stale bread—its ready pulverisation and its sponge-like absorptive power—render it easily broken up and easily saturated by the digestive juices, so that it is quickly and conveniently digested; while the new bread is liable to form, in the stomach and intestines, pieces which are not readily permeated by these juices, and are, therefore, dissolved with comparative slowness. The same thing is the case with meat. When it is taken into the stomach in large lumps, the gastric juice can attack it only on the outside; but, when it is thoroughly broken up by the process of mastication, it is dissolved with great facility.

We see the advantage of a fine subdivision of food in the case of milk, which in the stomach is curdled into small flakes, which are digested with great rapidity. These fine flakes are seen in healthy infants, who regurgitate some of their food after having taken too much. But, in children whose stomachs are out of order, the milk curdles in a different way, is thrown down in large indigestible lumps, which are either vomited up, or pass into the bowels, causing diarrhoea. In such cases, we mix the milk with lime-water, which causes the casein to be again thrown down in small flakes, and thus prevents a repetition of the mischief.

Now, imperfect mastication is a very common condition, and is the source of a great deal of the dyspepsia with which we meet. It may have several causes. Decayed teeth are one cause. Even when, on cursory examination, the teeth appear to be fairly sound, a small spot of caries on one of them will make it tender, and its possessor uses the other side of the mouth almost entirely for mastication. If there be tender teeth on both sides, he is tempted to bolt his food after masticating it very imperfectly. Want of teeth is another cause. We sometimes see patients whose teeth, so far as they possess them, are either

* Read before the Lancashire and Cheshire Branch of the British Medical Association.