HARVEIAN LECTURES

ON THE

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

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LECTURE II.—(Concluded.)

Hæmoptysis has a leading place among the events of chronic disease of the lung; and new doctrines have recently been enunciated about its influence, both as a cause and a consequence of such affections.

Hæmoptysis is generally a symptom of congestion, which, in such cases, is the real condition to consider, and to treat. It is only another word for pulmonary apoplexy, of greater or less extent. There is another, and very fatal, form, which is a mere leakage from a broken vessel, and almost always the result of the rupture of a small aneurism of the pulmonary artery.

There are, therefore, two kinds of hæmorrhage from the lung: the congestive and the passive.

To those who hold that chronic changes in the lung are due to inflammation, a hæmorrhage, arising from increased afflux of blood to a highly vascular tissue, is no unexpected event. It is, in fact, a part and a symptom of congestion.

On the other hand, the school who believe in tubercle-formation being the essence of lung-induration, are puzzled to account for it. I would remark that acute tuberculosis—by which I mean an invasion of a large tract of one or both lungs by the grey miliary (millet-seed) tubercle—is not accompanied by hæmoptysis. The acute croupous pneumonia has its coloured sputa (coloured, that is, by exuded blood); but hæmorrhage, as such, is not a feature in the case.

I think we need not discuss the question, whether hæmoptysis is of pulmonary or of bronchial origin. It is almost always pulmonary.

Whether the first step in the lung-induration be an inflammation or tubercular, we may, I think, concede that, excepting in the slowest and most insidious forms, it is accompanied by congestion of lung-tissue; and hence the great prevalence of hæmoptysis. It will be remembered that the earliest changes in phthisical lungs are shedding of alveolar epithelium and block of the air-cells, with consecutive small cell changes in the walls of the cells and in the intercellular tissue, in which lie the blood-vessels and lymphatics of the lung. Engorgement is sure to follow, and impeded return of venous blood; while the tissues become softened and disorganised.

The occurrence of congestive hæmoptysis at the beginning, or in the progress of phthisis, is accompanied by a high temperature, running up to 104° or 105°. Its persistence may also be gauged by the thermometer and by the pulse. Should a more or less sharp hæmoptysis subside, the temperature falls, and the pulse becomes soft.

Should the bleeding initiate a lung attack—that is, occur to a person apparently in good health—we may expect that it will be followed by the signs of consolidation of a portion of lung, and the events of phthisis. There is a form of rapid phthisis, of which I have given an instance, which proceeds with great activity after an initial florid hæmoptysis of some extent; and we must be on the look out for such, and remember that it proceeds by progressively causing patches of consolidation in the lung, of which you will have the usual physical signs.

Should congestive hæmoptysis occur (as it generally does) in the course of chronic phthisis, you may have a long pause, or suspension of active symptoms, following its cessation. I have so often had occasion to observe this event, that it seems well worth bearing in mind when called on to deliver an opinion on the result. How often, also, do we witness repeated attacks of rather profuse hæmoptysis, at long intervals, in the same patient? That a second and third hæmoptysis may succeed, is almost certain; and that an appreciable amount of relief to the lung is produced by the bleeding, I have no doubt. All these events bear strongly on my proposition at starting: that the local congestion of the lung has much to say to the clinical history of phthisis. I shall afterwards speak of its bearing on the treatment.

The structural changes induced, or directly caused, by hæmoptysis in chronic lung-affections, have recently received much attention from the investigations of my colleague Dr. Reginald Thompson.

According to his careful observations, blood poured into the bronchi, during an hæmoptysis, is drawn by insufflation into the deeper parts of the lung; and, in many instances, becomes impacted in the alveolar tissue. In this situation, the clots may remain for months and undergo slow changes, being converted into fibrinous nodules, which become capsuled, and undergo further secondary changes. "The microscopical appearances of these nodules consist of a group of alveoli, firmly packed with a semi-opaque homogeneous fibrinous material; and there is also thickening of the alveolar tissue, and of the interlobular tissue, which forms the investing capsule." In some instances, a copious exudation of blood-corpuscles is found in and around the air-cells. The ultimate changes are, that the nodule, by shrinking, often becomes detached from its capsule, causing occasional hæmorrhage in this process; and, being disintegrated, is poured into the bronchi, and expectorated—leaving a small cavity filled with a glairy fluid like honey. Many such secondary cavities of this kind may be found in a lung. What Dr. R. Thompson calls the traction of the sound part of the lung, initiates the separation of the nodule from its capsule.

The localities in which such blood-clots are found are observed to be chiefly in the "periphery of the lung, *i.e.*, the summit and middle part of the upper lobe, the middle axillary region close to the pleura, the anterior inferior border, and the middle part of the base corresponding to the summit of the arch of the diaphragm." These positions are exactly those in which an inhaling force, directed from the root of the lung, or larger bronchi, to the periphery, would lodge them.

The secondary changes in such clots are not due to putrescence, for blood does not putrefy if air be excluded; and in the positions indicated, they are not reached by air. This is in accordance with Zeimssen's experiment, in which no putrescence nor caseous pneumonia followed the injection of blood into the lung-tissues of a healthy dog. The fibrinous clots become hard, of a white or light red colour, often pigmented with black granules. Dr. Thompson considers that they do not degenerate into tubercle, nor irritate the tissues unless septic matter, probably from a tuberculous cavity, becomes mixed with them. Occasionally they calcify, and, such products being dislodged, a small dry cavity results.

These observations, which possess much interest, show us that hæmoptysis is itself a cause of important changes in the lung in certain cases. While acknowledging their value, I must observe that a large number of cases of lung-bleeding are certainly not followed by any such results. On the other hand, we should always be on the look out for them. Hæmoptysis may, as we are aware, be caused by other than tuberculous or phthisical diseases, and occurs often without any secondary result whatever, as in certain instances of pulmonary congestion, due to cardiac causes. I cannot enter here into the recent controversy imported from Germany, as to whether tuberculous or destructive diseases of the lung are initiated by hæmoptysis. My own belief is, that it is not a primary causative agent in any case, but that some morbid process in the lung has preceded it.

The other form of hæmoptysis—the passive—resulting from the rupture of a pulmonary vessel, or from an aneurism of the pulmonary artery in an ulcerated lung, is very familiar to us. The mechanism appears to be that one side of a pulmonary branch will be unsupported in the walls of a cavity, so as to present softening and aneurismal bulging. The result of such rupture is often immediately fatal. It occurs in males much more frequently than in females, for, out of twenty-two fatal cases, twenty were males.

The prognosis, in cases of large hæmoptysis, should of course be guarded. No one dies of a first hæmoptysis. Large hæmorrhages are apt to be repeated, the patient belonging, probably, to the class of individuals affected by hæmophilia; or the form of disease under which he labours favouring such structural deviations as account for his bleeding. Hæmoptysis is often followed by a pause in all the symptoms, the congestion of the lung being relieved. Many of the longest cases I have seen have been most frequently attacked by moderate hæmoptysis.

The case of a schoolmistress, now aged 43, whom I have watched since she was fifteen, has been often referred to by me. At the age of fifteen, a cavity formed in her left lung. Under my notice, long since, the subclavian walls have fallen in, and a dry cavity of smaller size exists. She has a rather profuse hæmoptysis once in several months. In her ordinary state she is rosy, well nourished, and weighs about twelve stone. Every hæmoptysis relieves her from dyspnoea and cough. She still teaches in a national school.

Having considered the secondary changes which hæmoptysis may produce in the lung-tissues, apart from any tubercular development,

we may examine the secondary local affections which occur in the course of most cases of chronic diseases of the lung.

In old times, these fibrinous or calcified deposits were regarded as tubercle. But modern pathology decides that in certain instances, sufficiently numerous, they are simply due to impacted blood-clots, which ultimately undergo degenerate changes. They are thus analogous to the alterations undergone by inflammatory exudations in other parts of the body. Many of our most chronic cases are doubtless of this class, and their extreme chronicity is an argument in favour of their origin from such cause. It has been said of tubercle that its life is short; and it is certainly most in accordance with modern observation, that a few months, at most, complete the various changes from its development to its death and degeneration.

But those puzzling and very chronic cases which last for years, with signs of consolidation or of cavity in one portion of a lung, may often be accounted for by an altered blood-exudation, which has retained only its fibrinous character, or has become calcified in the lung-tissues. These considerations should be present with us in forming a diagnosis, or in offering a prognosis of very chronic cases. In such we may not be witnessing the final degeneration of a mass of tubercle, but only the result of hæmorrhagic impaction, or of inflammatory exudation.

I turn now to the *secondary local affections* of the lung, and they are of great clinical interest.

A patient, suffering from active disease in a part of one lung, will have the opposite lung at base or apex, or the lower part of the same lung, attacked by congestion: that is, an induration similar in physical conditions to that which first existed, will develop at a distant part. You will have, say, a patch of dulness; breath-sounds more or less tubular, and crackle or moist crepitation. Febrile symptoms accompany, or rather precede, the physical signs, for the patient will have chills, high temperature up to 104° or 105°, pain in the side, rapid pulse, and possibly a congestive hæmoptysis before the physical signs are developed. Is this first attack in a distant part of the lung due to taking cold or exposure? As the event is witnessed daily in our hospitals, where the patient is guarded from every such event, we must reject this explanation.

That it is due to infection seems likely from the following circumstances:

1. From the likeness of the secondary to the primary affection. The congested or indurated portion of lung is found in the same pathological condition; that is, either an inflammatory exudation occurs affecting the alveolar and interalveolar tissues, or a deposit of that which is recognised as milary tubercle takes place. The irritative fever accompanying it is also identical.

2. That it is not a mere extension by continuity of morbid processes is evident from the fact of distance, portions of healthy lung intervening. At the same time, such direct extension does occasionally occur, but forms no argument against the infective theory.

3. Facilities for conveyance of morbid matters are abundant; either directly by mechanical conveyance through the air-tubes, or indirectly by the lymphatics and blood-vessels.

4. Analogies may be found in the extension of cancer to distant parts, or in the secondary abscesses from pyæmia, although the septic element has probably much to do with the latter.

An argument in favour of the infective theory may be found in the period of lung-disease in which these secondary attacks occur. According to my observation, they are most common during the period of softening, when tissue-compounds are disintegrating, and in a condition to enter the circulation or the lymphatics. They are also least common in cases of chronic cavity well defined, and, as we know, lined by a membrane, and surrounded by fibrous formations, which obstruct all the vessels and harden the tissues.

Before leaving this part of the subject, I would remark on the prognostic indications to be gathered from such attacks. They are among the most fatal events in phthisis, and it may indeed be said that they form the terminal period to most of our chronic cases. Viewed as congestions they are never completely recovered, and, regarded as extensions of tubercular deposit, they only indicate a further and lower stage of disease.

In forming a prognosis of any given case, we must then mainly consider the form and limits of disease, the existing symptoms, and the modifying agents found in age and heredity. Of form and limit I have given illustrations in the apex disease, well defined with much induration; and in the diffused form of disease which does not attain to the stage of cavity.

In chronic cavity, large or small, but single, and situated at the apex of one lung, we have generally a very prolonged example of

tolerated disease. The mechanical conditions of localisation and limit have been complied with; the necrotic processes have been completed, and a membrane lines the walls. The pulmonary and nutrient vessels have become obliterated. It is, however, a necessary condition that a healthy circulation should exist in the surrounding parts, and this is a *sine quâ non* for shrinkage and possible cicatrisation. The surrounding cells are generally emphysematous, and two or three are often found thrown into one. Such cavities may enlarge, or, from traction of other parts of the lung, may become united to another neighbouring cavity.

But their undoubted tendency is to contract, and that of the surrounding parts of healthy lung is to expand; and this should be remembered in treatment. Such cavities are large secreting surfaces, and are liable to catarrh, or, as my colleague, Dr. Powell, expresses it, a man may catch cold in his cavity from ordinary causes, and at such time there is increased secretion which may afterwards dry up. Cavities deep in the lung, with imperfect or circuitous connection with a bronchial tube of some size, are very apt to permit the formation of collections of secretion which become fetid, when the patient becomes intolerable to himself and others, and septic fever may arise and prove fatal or shorten his life. In such a state, the condition differs but little from that of chronic abscesses in any part of the body, and the surgical treatment by free emptying and drainage will form part of our considerations when next we meet. The other misadventures to which a cavity is liable are, profuse hæmorrhage from a pulmonary vessel generally lying in its side, and unprotected at one part, permitting bulging and progressive softening, or aneurismal dilatation. A pneumothorax from softening of the walls of the cavity or tuberculous ulceration of the investing pleura may, in rare instances, complicate and precipitate the case.

But on the whole, from its representing a local disorder which has completed its degenerative changes, from its distinct limitation by fibroid alterations of connective tissue around it, and from its isolation in the lung, it must be regarded as a form of disease tending to great chronicity.

In forming a prognosis in any given case, the chief symptoms which must decide a favourable or adverse result, are *fever* and *wasting*.

High temperature means the presence in the blood of febrile materials, the result of caseous degenerative changes in the lung. As is well known, there is a daily evening exacerbation, and the minimum is generally found about 7 A.M. In a chronic quiescent case of phthisis, these changes vary but little from the healthy standard. That which is of most evil import is a high evening maximum followed by a low morning minimum. The acute angled curve in the diagram, with a large diurnal change, is much to be dreaded. The gradually declining curve means diminished febrile material in the blood, and a coinciding diminution of urgent symptoms. A rapid sudden rise often indicates an approaching hæmoptysis, or a local congestion, or a deposit of milary tubercle in a distant part of the lung. A sudden fall to a sub-temperature is indicative of collapse, and often precedes a fatal issue.

From the fact of the minimum temperature occurring in the early morning, we may gather the indication for nourishment to be administered to the patient at that time.

Upon the subject of temperature, we may notice that the absence of high temperature contraindicates phthisis in doubtful cases where the physical signs are of a dubious character; and it may be remarked that, although the thermometer is occasionally at fault in offering positive evidence of existing disease, it is invaluable where giving negative results in doubtful cases.

Finally, we may remark, that the temperature will be found highest in acute florid phthisis, with extensive infiltration of caseous masses, while it is lowest in cases where fibrous alterations are most complete, because in the latter case there remains no broken-down material to be absorbed.

Of wasting of the body, it is to be remembered that progressive waste and high temperature are almost always coincident; and that the third element of phthisis, progressive local disease, is seldom absent in such an evil concurrence.

Progressive improvement in weight is generally, but not always, coincident with a stationary or improving condition of the lung. It is of course a favourable state; but in very numerous instances it will be found that, while a patient is daily adding to his weight, a steady but slow increase of local disease is proceeding.

Hæmoptysis, which is common to every stage of phthisis, is an uncertain guide in prognosis. A rapid uncontrollable form of disease is often begun by hæmorrhage, the tendency of which is to repeat itself at short intervals. I have before considered the conditions of lung which result in such forms; some consisting of a succession of conges-

tions, which unite and break up all the tissues. The more remote events which follow the impaction of clots should also be remembered.

But it is undeniable that some of our most prolonged cases are hemorrhagic. The congestion of lung appears to be relieved by the bleeding, and a long pause often occurs, during which the symptoms are in abeyance, and the patient loses his fever and recovers flesh.

The influence of age on the development and progress of chronic affections of the lung is well marked. The strumous cases—infectious from the break up of scrofulous inflammation—are naturally found in the earlier periods of life, during the years of growth, and in early adult age. Hence the large proportion of cases are found at that time. The more acute and rapid forms of disease are also placed here, and the more urgent symptoms, as high temperature and rapidity of waste, and profuse congestive hæmoptysis. But, about the middle period of life, although phthisis has not ceased, there is a noted lessening of its frequency, and a marked difference in its type. The degenerative changes in the lung are more of the class belonging to chronic induration than to caseation. At Chelsea Hospital, in the bodies of men aged from sixty to eighty, half the cases exhibited chronic diffused grey consolidation, concretions in the lung, excavations, or traces of recent and old cicatrised cavities.

The type of chronic lung-disease after 45 is slow: the febrile action mild, the secondary complications few, and there is precisely what might have been expected from a knowledge of its pathology. After 45 many cases of phthisis occur, although at that age insurance-offices reckon that the liability has been passed. Christison found that 22 per cent. of the deaths in lives selected by the Scottish Widows' Fund, occurred from phthisis between forty and fifty years of age. It is at this time of life that old deposits in the lung, the result of low inflammatory action, often at the base, break up, and may assume the tubercular form.

On the whole, we may assume, in offering a prognosis, that, the more advanced the age, the more likely is it that instances of disease will be latent in development, and chronic in form.

Permanent acceleration of pulse in the aged is often indicative of latent advancing disease. The complications, as profuse hæmoptysis, diarrhoea, and laryngitis are uncommon.

Did time permit, I should have much to say of the influence of heredity on chronic disease of the lung, both as a cause, and as influencing the form and progress of disease. I must confine myself to a few of the leading considerations.

From extended and careful observations, I believe that two-thirds of the cases of phthisis have had the hereditary element. Heredity should include remote ancestry. The common mode is to inquire if parents, brothers, or sisters have had the disease, but this is manifestly only a part of the story of our position in the chain of animal life: our inquiry, to be correct or reliable, should extend back for several generations on both father's and mother's side.

We are familiar with the fact that form and features, organic peculiarities, mental configuration, the shape of hand or foot, habits of movement, moral power or weakness, and especially emotions, are transmissible, not only from parent to child, but often an individual will reflect some of the peculiarities which have been known to characterise an uncle or grandparent in a remarkable degree. And so it is of what we call constitution, which means peculiarities of vital organisation.

It is true that the germ so inherited does not always produce fruit, because the soil may be unfitted for its growth. Thus, one individual in the chain of inherited weakness may escape. On the other hand, it is not to be forgotten that he may be a transmitter of disease, although not personally a sufferer. His children may, under favourable circumstances, develop the ancestral tendency.

Again, forms of disease are transmitted. Many members of the same family often die at the same age, of the same form or variety of phthisis, which one individual after the other appears to develop on arriving at the same period of life, which, in such cases, is generally early. We are all of us familiar with this painful fact in practice. Just as longevity is distinctly transmissible, just as members of the same family reach the same age of advanced life, which is indeed the term for which the machinery is adapted to last—so the inherited germ takes root, and develops a fatal disorder at a given period of growth in earlier life. I have known several members of one family, in repeated instances, exhibit precisely the same variety of phthisis—cavity, or diffused disease, or acute tuberculosis—as others of the same stock who preceded them. The hæmoptysical variety is notably heritable, and runs in families.

The question of marriages of near consanguinity arises here; and I believe they are safe, unless there be in the race some proclivity to disease, which, in such case, is precipitated on the offspring from both

parents. Such are some of the more prominent facts about heredity; and, in forming a prognosis of any given case of disease, they should always be borne in mind.

In concluding this portion of our subject, we may enumerate some of the leading features of the more favourable, and contrast them with the less favourable conditions of chronic lung-disease.

The more favourable local conditions are:

1. Well limited induration. The more a portion of lung is indurated, shown by marked and defined dullness, and the other signs of consolidation, the better the prognosis. The more marked, on the other hand, the signs of caseation and degeneration processes, the worse the prognosis.

2. The single cavity, well limited, and defined: the other parts of the lung unaffected.

3. The diffused form of disease, which I have described as exhibiting crepitant sounds over a large surface of lung, with moderate dullness, and slightly impaired movements in a well-shaped chest.

4. The existence of extensive fibroid alterations, with well-marked characters.

Among the general favourable conditions, the most important by far are the absence of fever and waste. These two symptoms are, as we know, concurrent in almost every case. The fever, being fed by morbid material from the disintegration of caseous products in the lung, is a direct measure of local irritation; and the amount and degree of local irritation may almost be said to be the measure of the patient's danger. Loss of flesh is always an evil symptom, as progressive regain of weight is good.

As regards temperature, high maxima and low minima are of bad import. The table-curve of temperature which is best, is one which, avoiding extreme fluctuation, gradually subsides to low or normal level, and remains there, with the slight diurnal variations of health.

The absence of secondary complications, as diarrhoea or laryngeal ulceration, is of primary importance, if we are to hope for prolongation of our case, or restoration to comparative health. Diarrhoea of the persistent form is especially prejudicial; also anorexia or vomiting, often repeated. With a fairly sound digestion, your patient may weather many lung-difficulties; and I consider this next in importance to the absence of fever and wasting.

Finally, to the above conditions we may add the following:

Absence of hereditary taint;

Age past forty-five;

The chest well shaped (not of the phthisical form).

In making a summary of the symptoms of any case, the physician, carrying his knowledge with him, ever ready of application, will doubtless apply the doctrine of averages to every individual he sees. But he will also gather indications from a variety of minute signs—the face, the hand, the skin of his patient; and also from his general nerve-condition; and, by a multitude of minute symptoms difficult to put in words, he will come to a judgment as to his vital powers of resistance. Especially, he will remember that chronic chest-disease is multiform; that some individuals outlive a vast amount of local disease and systemic waste, while others succumb to comparatively light disorders. He should also not be in haste to prophesy results from any given condition of his patient. Especially should he avoid condemning a case as in the "last stage", a term formerly applied to cavity in the lung, but inapplicable as expressing a local condition only. The patient's powers should be measured, and his vital resistance estimated, and the numberless cases remembered which have outlived evil prognostics, and possibly adorned the short-lived triumph of some quack medicine.

While condemning every thing untruthful in opinion, we are bound to give our patient every encouragement which is truly derived from an intimate study of his disease. Above all, let us never prophesy as to time. You will often be pressed to say how long; but, unless in cases of impending death, we are not called on thus to map out disease. For, indeed, it is not often possible to assign a duration to even our most advanced cases; and to become prophets as to time is to subject our art to almost certain depreciation in the eyes of the vulgar.

SOCIETY FOR RELIEF OF WIDOWS AND ORPHANS OF MEDICAL MEN.—The usual quarterly court of directors of the above society was held on Wednesday, January 12th, at 5 P.M., Mr. John Gregory Forbes, V.P., in the chair. A sum of £1,270 was awarded to 59 widows, 15 orphans, and three recipients of relief from the Copeland Fund. The expenses of the quarter were £63 2s. 6d. There were no fresh applicants for ordinary grants, and one only for extra relief from the Copeland Fund. Two new members were elected, and the death of one was announced.