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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.

MR. PRESIDENT AND GENTLEMEN,—To the student of disease, whose own period of observation becomes somewhat prolonged, two questions ever arise afresh: Do we advance in our knowledge? Do we improve in our treatment? To such of us as are actively engaged in practice, these are difficult questions to answer. We are immersed in the details of individual cases, hourly pressing on our notice; we are embarrassed by the multitude of minute facts; and our judgment of the whole is obscured by the very nearness of our contact. For large views, and general deductions, we should like to retire to an elevation; and survey, as from a distance, the collected observations of which our knowledge is composed. From such a height, the relation of events to each other becomes more truly estimated; their value sinks or rises to its proper level; and their very colour and form assume a more or less certain aspect. That which seemed all-important and typical in individual cases, was often found to be but a segment of a larger sphere—a fragment of a greater truth; or, possibly, to belong to quite another category of knowledge.

Again: our private records of cases have possibly, as years pass by, become an accumulation, which, for want of time, of opportunity, or of the master power of organisation and arrangement, has lost its value as a whole, and is as purposeless to the student as a museum without catalogue or classification. Loaded, as we are, by the public with the grave responsibility of life and death, we are taxed to the last hour of our day. We are careful observers; but we are scarcely left the time for a faithful fleeting record of the natural phenomena we witness hourly. We would fain organise the knowledge which is passing under our hand. We would fain deduce general conclusions from our multitude of facts; but to few, indeed, is granted the leisure, or the opportunity, for thus fashioning our knowledge into a form from which others shall learn; or which shall become, even to ourselves, a store on which we may draw.

The mind, so occupied, is not reposing; nor are its powers stationary; nor does its development flag: for judgment improves, vision and appreciation of truth become clearer, and individual cultivation reaches a high level. There are many such men among us, who are practical, cultivated, and whose opinions on individual cases are of the highest value. But their knowledge too often dies with them; and, though they may have advanced the cultivation of the individual mind to the highest point possible, they may leave little or nothing behind to add to the ever progressive accumulation of knowledge. How much is lost here! Who can appreciate the depreciation which medical science undergoes by the silence of such men? Their experience has been the honey on which they have themselves fattened; but it has been lost to those who are to follow them.

I will not stop to inquire whether such men, if placed on the mental elevation which they covet, and with the leisure accorded which seems now ever to elude them, would become revolutionisers of old theories, or light up the old paths with such genius as to entitle them to be called discoverers. Believing, as I do, that knowledge is ever progressing,

anon with great loud-sounding discoveries, but ever with a silent sure advance, to which many forces contribute, I rather class such men as among the invisible builders who, by precept and contact with others, spread and increase the seed which is to ripen at a harvest-time where they may not personally assist. As the Coliseum of Rome was built by a hundred thousand captive Jews, yet is said to have been raised by Titus, so science achieves colossal products; but the units who contribute are lost in the great name which one man only may be permitted to inscribe on the summit.

But, I will remark of this class of practical men, that, while they are scrupulously minute workers, and the most careful of observers, they are slow to change the names which their teachers gave to phenomena, or diseases; they cling somewhat tenaciously to ancient doctrines, and respect, to an extreme degree, that which they believe to be a *form of sound words*. Thus, though diseases and facts remain the same, while nomenclature has altered, your entirely practical man is often puzzled by the latter; and thinks that a new fact is announced if the language used for its description be changed.

Again: there are plenty of youthful students, hard-workers with the scalpel and microscope—and, above all, diligent students of modern German writings—who, instinct with the zeal of discoverers, hail every novelty, and have already changed their pathological nomenclature more than once for most structural affections. They represent the onward current—the apex of the advancing wedge, which is to cleave all mysteries, and dissipate all ignorance. Unquestionably, we owe much to them; but much more to a whole army of observers, not wedded to any school, unbiassed by any master, *nullius in verba magistri*, who are the faithful chroniclers of our pathological facts, and the heralds of all our advances.

I have thus introduced you to the statement that there have been, and still are, progressing among us, great changes in our views of disease, founded on our advancing knowledge—sometimes on our ancient wisdom or errors—but always manifested in the current literature of the day, and in our intercourse with the inquiring minds of the best men in our profession; and I propose to occupy these three lectures, commemorative of one of the great fathers of our knowledge, in an inquiry into the changes which modern pathology, and especially our views of treatment, have undergone regarding diseases affecting the lung.

The task which I have assigned to myself is to consider the changes which have in our time occurred in the pathology of chronic chest-diseases, and, if possible, to reconcile these changes with facts of clinical observation.

By an easy advance, we shall be led to consider their treatment. Our question is, Has modern pathology thrown light—more light than we had previously—on living phenomena and vital questions, and have we benefited by it so as to have more than a mere change of nomenclature?

Of course, as we advance in this inquiry, we shall become more practical, and our subject less dry; but it is a matter of very grave inquiry whether we have made real improvement, and have come to know more than we did some years ago of the real nature of structural changes in the lung; and I must ask your indulgent patience while I consider, in the first place, some anatomical and histological points. Of theories, I shall have little to say; of facts, much. Such questions as the definition of tubercle I must leave untouched; and I would remark that it is especially noticeable that our greatest modern authorities decline to pin themselves to a definition.

But I wish to bring modern knowledge to the front, and, irrespectively of theories, inquire if we can so divide our cases clinically, that their vital phenomena shall correspond to certain known changes in the lung, so that our anatomical and histological knowledge shall guide us to a more correct appreciation of the living case, its events, and its future. I shall not, of course, advocate the views of any individual, but, out of the great mass of recent knowledge added by many observers, select those facts on which all have pretty well agreed regarding the changes in diseased structures. We shall then inquire whether it is possible to arrange our living cases into corresponding groups. If we find certain points of agreement among all observers, although there may be differ-

ences in detail, we may, I think, safely conclude that we are near the truth.

I shall first state generally and briefly the leading change which has taken place in our time in the pathology of chronic lung-disease. We may say that, up to the time of Bayle, and Louis, and Laennec (that is, within the present century), there was no definite theory of the morbid condition of lung constituting consumption. It is, perhaps, to Laennec, more than to any other, that we owe definite ideas regarding what he called tubercle. His well-known theory was that, owing to a depraved state of constitution, an exudation took place into the structure of the lung, which, in time, underwent certain changes of caseation and softening, ending in ulcerative destruction of all the lung-structures. In studying Laennec, I fail to find—plainly for want of microscopic knowledge of the minute structures of the lung—any information as to the part primarily invaded. We are not told whether the so-called exudation was within or without the air-vesicles, or in the interlobular or perivascular structures. The miliary tubercle of Bayle, which was supposed subsequently to pass into a yellow caseation, and finally to undergo degenerative changes, is open to the same remark; and I am not here interested to say whether this theory is susceptible of support.

It remained for Addison in this country, and for Niemeyer more recently in Germany, to express their conviction, that the disease which is called consumption is mainly due to inflammatory changes; and that the resulting exudation-product in the lung undergoes degenerative alterations, which ulcerate all its structures. Virchow showed that the yellow caseation so resulting may be the product of the fatty degeneration of various morbid matters, as pus and cancer, and that much of what is called tubercle is of inflammatory origin, chiefly epithelial. The adoption of these views of inflammation in this country has given the stamp to the modern doctrines regarding lung-disease. Following the German view, inflammations of the lung are now divided into two leading forms, croupous and catarrhal; and it requires a very able practical physician to separate clinically the latter form from consumption. We must ask whether phthisis is an inflammation? and is the lung-exudation, or that morbid material which is found in the lung in chronic ulcerative diseases, identical with the product of acute inflammation? Are the structures of the lung so invaded or infiltrated (if I may use the term) identical in both cases? It is plain that I mean entire identity, not a mere histological sameness, the same cells or the same nuclei, but a real conformity of the structure invaded and altered, and of subsequent history; for these products of which we are speaking are short-lived, and undergo certain chemical changes dependent on their diminished vitality. They lose their blood-supply, become cheesy, disintegrate, undergo fatty degeneration, and may be absorbed; or they may become changed into fibrous structures of low vitality but contractile, which alter the whole structure of the lung. Again, is the anatomical portion of the lung primarily affected the same in the acute inflammatory attack and in the so-called tubercular phthisis?

In the answers to these two questions, viz., Is the product in the lung identical histologically and chemically in the two affections, and are the structures first invaded actually the same? I hope to show that the key will be found to many important practical problems in the history of chronic lung-diseases, and also in their treatment; for not only is the nature of the affection involved in this inquiry, but also the progress and ultimate event of these diseases implied.

I proceed, therefore, briefly to consider the phenomena of acute inflammatory disease of the lung, and compare them with those of chronic forms. The divisions of pneumonia are now-a-days into the croupous and catarrhal. In the first, which is the old sthenic acute form, we have high febrile symptoms, ushered in by chills, a temperature rapidly rising to 105-108, and all the signs of intense consolidation of the lung; the vesicular structures of the organ being obliterated, and only the larger bronchi remaining pervious. The result, as is well known, is a fibrinous exudation into the alveolar cells, which, after an interval of eight to twenty days, undergoes rapid degenerative fatty changes, and is absorbed, leaving the walls of the air-cells unimpaired and elastic as before. Recovery is perfect, the breathing being mechanically and physiologically performed as in health. Neither is the interlobular structure altered, and there is no permanent thickening around the ultimate vessels or bronchi in the interalveolar cellular tissue.

When opportunity is given of examining such products in acute pneumonia, they are found to consist mainly of an exudation of fibrin with white and red corpuscles, and the bronchioles are filled with coagula, even up to those of fifth magnitude. As regards their site, they are strictly intra-alveolar, that is, they are confined to the air-cells, and the walls are not engaged, unless indeed by pressure from within. We have all no doubt admired during life the perfect recovery which takes place after pneumonia, and how speedily vesicular breath-sounds

succeed to the blocked dumb condition of lung, which prevailed in the first stage. This is due to the fact that the walls of the air-cells have never been impaired nor disintegrated in the degenerative process of clearing up the cells.

But sometimes these pneumonias, however acute at first, do not clear up and resolve. At the end of about the second month, the patient, who has expected convalescence, but whose leading symptoms have subsided, becomes again somewhat feverish. His temperature rises, say, to 103° in the evening; and he has slight sweatings, and does not recover flesh, and begins to cough. The physical signs tell that the lung, although less solid, has not recovered sonority; and air does not penetrate the remotest vesicles; or there may be patches of dulness and bronchial breath-sounds, showing consolidations, partial, and somewhat insulated by tracts of lung more pervious. If opportunity be given of examining the morbid portion, you will find not only the air-cells blocked, but the alveolar walls themselves thickened, and invaded by a small cell-formation, with some fibrillated structure; so that their elasticity is lost; while the interalveolar structures are thickened, and the terminal vessels, pulmonary capillaries, and minutest bronchioles are beginning to be strangled by a new growth. These changes precede those of softening, in which all these structures are broken down and ulcerated. The case now looks very like phthisis.

I take now the description of catarrhal pneumonia from the latest German authorities, for it is to them we owe its definition. First, we have catarrh of the minute bronchi, with its fine mucous rales, gradually becoming of a character impossible to discriminate from crepitation, except that they are heard on expiration as well as on inspiration; next, signs of consolidation, beginning at the base and spreading rapidly upwards, and usually bilateral. These signs are often difficult of recognition. Above all, there is an indefinite limitation of disease, as judged by its physical signs. The evidence of infiltration is often doubtful. Certain tracts of lung become of diminished volume; or, again, an increase of volume may occur. The pathological conditions represented by these two opposite states are, collapse of a tract of lung, and infiltration of a similar tract, causing induration. A simple deficiency of breath-sounds accompanies the former; the latter will, of course, be indicated by bronchial breathing and voice. With the loss of such respiratory space, there is a proportional dyspnoea and lividity. The fever is of varied type—102.4°, with remissions. It is not typical, because it does not reach the height of croupous pneumonia, nor fall to that of ordinary phthisis; and, on the whole, there is a want of relation between the local and the general symptoms. Often there is fever before physical signs, and fever with waste; for one is the measure of the other. It is held, indeed, that fever is caused by the presence in the blood of septic material—in this instance, probably the detritus of local degenerations. Have not, then, the degenerations preceded the fever?

The pathological results of catarrhal pneumonia are, sero-sanguineous exudation into the alveoli; desquamative epithelial products, which block the air-cells; and, if the attack proceed long enough to cause infiltrations, the walls of the cells are thickened; numerous lymphoid cells appear in the blood-vessels and in the interstitial connective tissues, having escaped from the blood-vessels. Later, degenerative changes occur of the caseous kind. Isolated nodules, from the size of a pea to that of a hazel-nut, are found within the collapsed portions of lung; and these often become confluent, so that an entire pulmonary lobe may be so occupied. These nodules degenerate, and may form cavities. Examined in a later stage than this, the lung is found contracted; its connective tissue increased, so that a section presents thick whitish-gray bands of fibres. There is pigmentation in patches, and dilated bronchi are not uncommon. Albuminuria is a not unfrequent complication. This form of pneumonia has received the names of desquamative, catarrhal-cheesy. It is distinctly held to be a *non-tubercular* disease. Tubercle may be developed in the later stages; but the disorder is held to be inflammatory; and hence the new doctrine and the old stand in opposition. Laennec and his school would have taken such lungs as I have described to be tuberculous. The cheesy masses would have been ascribed to the secondary changes undergone by grey tubercle, which should have certainly preceded the caseation. There seems, however, no doubt whatever that such changes may occur; and such patients may die without the development of a single tubercle. It is true that groups of tubercles are almost always found; but they are secondary, of recent formation, and short-lived, often not sharing in the softening degenerative changes undergone by the other indurations in the lung. "The differential diagnosis between a catarrhal pneumonia," says Jørgensen, "developed from an acute bronchitis, and one developed from a tubercular bronchitis, must depend chiefly on the history and on the course of the disease before the occurrence of consolidation." "Whenever groups of tubercles in the lungs

are associated with catarrh of the finer bronchi, it is very difficult to distinguish them from catarrhal pneumonia." To you, gentlemen, who have kindly followed me, and who have compared these descriptions with your clinical experience, it will be evident that I have been describing to you a phthisis. In such a case, it is not "very difficult", but impossible, to distinguish the two affections.

I have dwelt on this description, however, for a clinical purpose. Names change; but diseases remain the same. If these lung-changes are due to an inflammation, and not to a constitution or innate depraved process of blood-making, may we not be more hopeful about some of their forms, and predicate with some certainty how some may rise from the hopeless into the remediable class of affections?

I desire to contrast with this description of catarrhal pneumonia that which the same German school (and, following in their track, the English) have accepted pretty generally as applicable to chronic phthisis. The affection is characterised by "waste", as its name implies; and it is applicable to several forms of disease different in character, but all possessing, as common features, a constantly increasing emaciation and gradual destruction of life. Phthisis is multiform, but has family features connecting all its varieties, and a widely differing progress, and an infinite variation in duration. Some die of it in weeks or months; others survive its structural changes for years. We shall inquire into the causes of such diversity, and endeavour to select living features which shall enable us to detect them when brought before us.

Tubercle is, then, not an essential element in the disease; but, when found, is a secondary superadded result, arising from infection, or the resorption of inflammatory results in the individuals themselves. Tubercle is a short-lived product, arising from inflammatory residua which have undergone degeneration—caseation—and been conveyed into the system, or to distant parts of the lung or other organs, by the blood-vessels and lymphatics, or even directly by the air-tubes. Tubercle probably lives but some weeks or months; but the changes in the lung formerly ascribed to tubercle may last for years. But how does this self-infection arise? What is the origin of the disease which furnished the infecting source? For there is no such thing, according to this school, as chronic miliary tuberculosis. Phthisis is anatomically a chronic inflammatory disease. It is a specific variety of inflammation, with characteristic caseous secondary changes, which are, indeed, only the death of the product. It undergoes a necrosis. Not being provided with vessels, and its vascular supply being cut off by the thickened and altered perivascular tissue, the vessels are strangled. Of course, pathologists of this school are forced back on a primary cause, as were the preceding generations; and, if lung-changes do not arise spontaneously, it is necessary to account for them. A catarrh of the minute bronchi will not, in a healthy person, originate a phthisis; but a neglected catarrhal bronchitis in a scrofulous person will do so. Scrofula, then, is the key to the whole sequence of changes. In a scrofulous person, inflammatory changes are slow; they cause permanent infiltrations of the cellular connective tissues of organs, which caseate and die. An exudation of large cells occurs, which swell up, and undergo necrosis; and the detritus passes into the lymphatics, and is thus transported into the subepithelial connective tissue. The exudation is hard, dense, grey, and unprovided with blood-vessels; so that necrotic changes soon begin, and in those changes the walls of the air-cells and the surrounding perivascular and peribronchial tissues participate. Such indurations know of no recovery. They may degenerate and be removed, leaving a hole in the lung; but they are never organised and absorbed. The first step, indeed, in the history of a restoration to health in such tissues, must necessarily be a restoration of a healthy circulation; and this does occur around all cavities which may close and cicatrise.

But how does phthisis arise? how does scrofula become tubercle? or how does the lung-affection, with all its changes of structure which we have been considering, and call phthisis, commence from an infection? If it be a result of infection—a self-poisoning—the poison must have originated somewhere else, and this argues an antecedent disease of a part, for it is no longer pretended that the blood or humours elaborate a septic material to be deposited in the meshes of the lung-tissues. The reply is, that a scrofulous disease of mucous membrane, or of joints, or of a lymphatic gland, has furnished the detritus of a caseation which was afterwards carried by vessels or lymphatics into the lung, and originated the destructive changes in question. You see, those holding this doctrine have abandoned the theory of a general disorder of the blood or system reflecting itself in a local disease, and come to the conclusion that a local disease of some distant part has originated another local disease by infection; thus the mind is carried to localisation of disease as a primary factor and an originating cause in an affection hitherto considered constitutional. It is the tendency of the matter-of-fact mind of the day to believe only in influences which we can test and

handle and examine with the scalpel and the microscope! And yet—as a sage observer has remarked—"at bottom, we are all believers in the theory of humours". I see, in this recognition of the vast importance of structural change in all vital disorders, although it may surpass our means of proof, a key to much that is of value in prognosis and treatment. For it does, indeed, depend on the nature, the limit, and the proclivities of diseased products whether structures can be repaired, indurations dispersed, or changes tending to life and organisation, or to death and degeneration shall take place. And, in treatment, if the fever and wasting be a measure of the inflammation in a part, we can hope to moderate *both* by checking it in the affected organ; but if these formidable symptoms be a blood-dyscrasis only, we are lost in the search for blood-remedies which shall meet and modify or reverse them.

[To be continued.]

ABSTRACTS OF LECTURES

ON

FURTHER INVESTIGATIONS ON ANTHRAX AND ALLIED DISEASES IN MAN AND ANIMALS.

Delivered at the University of London, December 1880.

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LECTURE III.

DR. GREENFIELD discussed the various forms of anthrax affecting man. The two external forms—typical malignant pustule and its modification of "anthrax œdema"—have long been known; but the internal forms, unattended by external lesion, have only been investigated of late years; and, until recent investigations on woolsorters' disease, the form of intestinal anthrax was the only one scientifically established. Now, however, he was able to bring conclusive evidence that the disease affected man internally in other ways; and it was to these forms of the disease that he would especially refer in this and subsequent lectures.

Malignant Pustule, long known to affect persons dressing the carcasses, or working in factories with the wool, hair, or hides of animals which have died of splenic fever, was first described, and typical cases related. The principal symptoms are briefly as follows. At the local seat of inoculation, a small pimple forms, which soon passes into a papule, and then changes to a flat vesicle, which gradually enlarges and bursts, discharging a clear or bloody fluid. The base of the ulcer dries, leaving a dark brown or black spot. Around this smaller vesicles appear, which soon undergo the same changes; and in this way the ulcer enlarges to the size of a shilling or more. The surrounding tissue is swollen, red, and inflamed; so that the ulcer is seated on a hard raised red base; and there is a kind of erysipelatous blush or swelling extending for a considerable distance around. The lymphatic glands connected with the part are frequently greatly swollen. If the patient survive, the central black eschar may become detached by sloughing, and the wound granulates; the œdema and lymphangitis gradually subsiding. The constitutional symptoms vary greatly, and do not in all cases at all correspond in severity to the local symptoms, and in some cases are entirely absent. The temperature is usually high when any fever is present; the pulse and respirations accelerated; the latter frequently oppressed. Great prostration, anxiety, delirium, profuse perspirations, and sometimes convulsions, precede death, which may occur in thirty or forty hours, or within five or six days, but rarely later.

Passing next to internal anthrax, Dr. Greenfield reviewed the history of the recorded cases of real or supposed internal anthrax in man, contracted by eating diseased flesh, or in other ways. Most important amongst them was the form described by Wagner, and subsequently by Leydig, Monck, and others, as *mycosis intestinalis*. In this form, which, in the cases described by Wagner, arose in persons employed in working Russian horsehair, the lesions were chiefly confined to the gastro-intestinal tract. The symptoms were, extreme prostration; vomiting; sometimes dysphagia; slight pain and uneasiness in the abdomen, or colic; diarrhoea, usually bloody, and, it might be, accompanied by profuse hæmorrhage. Bleeding also sometimes occurred from the mouth and pharynx. Cyanosis and collapse usually ended the case, and death might occur very rapidly. Very often there was