LECTURES ON BRONCHIECTASIS.

Delivered at the Hospital for Consumption and Diseases of the Chest, Brompton.

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LECTURE I.

BRONCHIECTASIS, or dilatation of the bronchi, can hardly be considered a disease, inasmuch as it is present in several and somewhat differing states of the lung. It is rather to be called a result of disease, the chief characteristic being a loss of the natural elasticity of the bronchial tubes, and, therefore, an inability to resist the normal air-pressure, and to expel the bronchial secretion.

There are three principal causes of bronchiectasis, and the part which each plays in the course of dilatation determines, to a great extent, the form of the lesion; these are, (1) inflammatory changes in the walls of the bronchi; (2) accumulation of secretion; (3) increased air-pressure. The action of the first cause may be seen in cases of bronchial dilatation following bronchitis; of the second, where a tumour obstructs the main bronchus, and causes accumulated secretion behind the obstacle. In this case a whole lung, or at any rate a whole lobe, is converted into what appears, at first sight, to be a series of abscesses extending to the pleural surface, but which are really bronchi dilated from accumulated secretion. The action of the third cause is well seen in long-standing cases of pleuritic effusion, and in fibroid phthisis, where, owing to the air-pressure, which would naturally be distributed over the whole pulmonary surface, being concentrated on the limited area of the bronchial tubes, dilatation of the walls takes place.

Bronchiectasis varies considerably in extent; in many cases of bronchitis and phthisis, it is limited to a few of the tubes, and, beyond causing delay in expectoration, is of little significance. Where, however, it involves the greater part of the bronchial tree, and even if only that of one lung, it becomes a serious and very dangerous condition, forming an insurmountable mechanical difficulty in the way of expectoration. In these cases, the secretions are retained, putrefy, and give rise to ulcerative changes in the bronchial walls. Cavities, differing from tubercular caverns by their labyrinthine shape and their foetid contents, form, and still further intensify the difficulties of the patient. The violent expiratory efforts are followed by deep inspirations, and by inhalation of the putrid matter into the hitherto healthy portions of lung-tissue; septic pneumonia supervenes, and the patient dies, poisoned by his own secretions. The cases I shall in the next lecture relate will prove to you that this mode of termination is by no means uncommon.

Before proceeding to the clinical examples of this disease, let us briefly consider its morbid anatomy and pathology.

Morbid Anatomy.—Two principal forms of bronchial dilatation, the cylindrical and the globular, are found in the *post mortem* room; in each case the tertiary bronchi are dilated, so as to measure as much, and even more, in circumference, than the primary. In the cylindrical form, the dilatation is more general, the tubes being stretched like the fingers of a glove; the walls are thickened, the submucous and mucous layers are hypertrophied, and stand out in folds. The generally tubular form of the bronchi is owing, according to Hamilton, to the traction on their ends from an adherent pleura. In the globular form, the tubes are dilated at or near their ends into pouches or ampullæ of large size, the adjacent dilated tubes often freely communicating with each other, the mucous membrane appearing to form folds or wrinkles in their interior. The lung beyond the ampullæ is generally non-vesicular from collapse and from pneumonic infiltration of the air-vesicles. The globular dilatation is sometimes fusiform and sometimes saccular, presenting a surface of mucous membrane which secretes largely, and thus leads to greater accumulation of mucus than in the cylindrical form. The walls of the bronchi are so thin, that the grey tissue of the lung can be seen through them.

In the specimens before you, there will be noticed the smooth, shining character of the lining-membrane of these cavities, which generally distinguishes them from the vomicae of phthisis; for, though it is not rare to find a phthisical cavity lined with a membrane, the

membrane in this case is pus-secreting and thin, and seldom of the shining appearance of a bronchiectatic one. The cause of this is the presence in these dilatations of the basement-membrane of the bronchus, which appears to be remarkably tough and elastic, and therefore dilates with the bronchus without cracking. In some cases, however, a later stage is arrived at, when ulceration of the wall of the bronchus takes place, and the fresh excavation, which is often accompanied by gangrene of the lung, is not lined by any membrane. In the cylindrical form, we find the lung-tissue generally compressed and airless, or else in a state of interstitial pneumonia, the pleura adherent, and the interlobular fibrous septa, which pass from the fibrous coat of the bronchi to the pleural surface, enormously thickened, the walls of the bronchi themselves being apparently indurated. Another feature is the labyrinthine character of the cavity; one dilatation opens into another, and, besides the recesses which are seen within the original cavity, several bronchi are to be found opening into the same bronchiectasis.

The globular form is found in connection with emphysema, and in lungs collapsed by pressure of fluid; the extent of dilatation of the tubes varies in different bronchi, being as a rule most extensive in tertiary tubes where the amount of cartilage present is very small.

The condition of the intervening alveolar tissue, as you may have gleaned from what I said, varies greatly. In cases of slight bronchiectasis, there need be no great variation from the normal standard. In many instances, large-lunged emphysema is present, though, curiously, Biermer denies its possibility, and argues that the resiliency and support which such tissue would give to the bronchial tubes would prevent their becoming dilated. He, however, quite forgets that the great inspiratory efforts which produced the emphysema might also produce bronchiectasis. Dilated bronchi often accompany the small-lunged emphysema of the aged. In many cases, however, where the dilating force is great, and, specially, where some obstruction exists in the main bronchus, the alveolar tissue appears to be almost entirely absorbed, and the lung has a honeycombed appearance, being composed entirely of dilated bronchi, full of yellow secretion retained through obstruction. I recorded a case of this kind in the *Pathological Transactions* (vol. xxiv, p. 23), where the obstruction was a cancerous growth invading the right bronchus, but growths of which, in the form of villous excrescences, blocked the lumen. Sir George Burrows *Medico-Chirurgical Transactions*, vol. xxvii) and Biermer have also recorded cases.

The pleura, in most of these cases, shows adhesions. Barth found that, out of thirty instances, only two had the pleura unaffected; in two, there was liquid effusion, and in the rest adhesions, which as a rule overlay the seats of bronchiectasis.

In one of our specimens, you will see the distension from accumulation of secretion well shown. It is a specimen taken from a patient of Dr. Tatham's, who died of tubercular meningitis. The lungs contained old tubercle, but also a curious condition of the bronchial tubes. The left bronchus is slightly contracted at its commencement; the secondary bronchi are slightly dilated, but empty; the terminal ones in several instances are largely dilated, *i.e.*, to the size of a walnut or filbert, and contain masses of opalescent mucus. The lung-tissue is partly pneumonic and partly in a state of fibrosis. Here it is probable that the contraction of the bronchus, by opposing free expectoration, caused accumulation of secretion, and dilatation of the terminal bronchi.

Histology.—If we examine sections of a dilated bronchus, we soon discover the cause of its diminished elasticity. In cases of bronchiectasis originating in bronchitis, the course of events appears to be as follows: (1) congestion of the bronchial capillaries; (2) proliferation of the transitional epithelium, the ciliated being shed, and the new cells partaking of the ovoid form; (3) infiltration of the submucous and inner fibrous coats by serum and migrated leucocytes; (4) the proliferation of the connective-tissue corpuscles of the peribronchial fibrous tissue. This infiltration of the inner and middle coats becomes in time organised into a species of fibroid tissue, which, pressing on the glands and non-striated muscle of the submucous layer, soon accomplishes their destruction. We find the muscular fibres undergoing granular degeneration, and replaced entirely by this fibroid tissue. The openings to the mucous glands become the seats of small abscesses, which are often the starting-points of ulceration of the epithelial surface, their acini being abolished by the infiltration. Bronchial cartilages disappear under the same pressure. The hyaline matrix is absorbed, leaving larger spaces in which at first the cartilage-cells remain as wandering cells; but these speedily undergo degeneration, the nuclei being only traced by globules of oil. Thus the bronchus becomes a fibroid tube, incapable of contraction, and only capable of gradually yielding to the air-pressure. The columnar epithelium being shed, it has lost the help which ciliary movement gives to expectoration. In this state of things, the increased bronchial secretion accumulating blocks up some of the

smaller bronchi; and consequently, the remainder being subjected to a larger proportion of inspiratory pressure, further dilatation takes place.

For the various theories which have been put forward to explain bronchiectasis by Laennec, Andral, Renand, C. J. B. Williams, Corrigan, Gairdner, Stokes, Grainger Stewart, and Lebert, I must refer you to Dr. Grainger Stewart's excellent article (*Edinburgh Medical Journal*, vol. xiii). But, in attempting to explain bronchiectasis, we must take into account the three following factors: (1) loss of elasticity of the bronchial walls through inflammatory changes; (2) the results of accumulating secretions; (3) an increase of inspiratory pressure.

As a rule, the first and third causes are generally those in operation; for, we must bear in mind, pneumonia precedes bronchiectasis in a large number of cases. Barth found it in twelve out of forty. Kapp states that twenty-one out of twenty-three of his cases died of pneumonia; but he probably refers to the secondary or septic pneumonia induced by the inhalation of the foetid matters, which is not what we are considering at present.

With regard to the second cause, it is doubtful, provided the bronchial wall be healthy, if accumulation of secretion alone can cause bronchiectasis. There must exist a narrowing of the bronchus above, whether the obstruction be caused by the pressure of an enlarged bronchial gland, or from a lymphoid or cancerous growth pressing on or invading the channel, or, again, from shrinking of the lung-tissue around the tube, as in fibrosis or cirrhosis of the lung. The inspiratory pressure is a most powerful element; but, in severe coughing, the expiratory act, owing to partial closure of the glottis, may assist in the process of dilatation. The effect of the inspiratory pressure may be well seen in cases of chronic effusion into the pleura and of pleuropneumonia. Here the inspiratory force, which ought to be distributed over the whole aërial pulmonary surface, is concentrated on a few bronchi; and it is not wonderful that in time their walls should yield.

Clinical History.—In the history of this complaint, it is sometimes marvellous to note how such great changes can take place in the air-tubes without giving rise to more symptoms. In most cases, all that can be obtained from the patient is a history of winter-cough for some years, with perhaps a slight attack of hæmoptysis. The breath has become short lately, and the expectoration more profuse. The patient lays great stress on his not losing flesh; and often states that, were it not for the cough, he would be quite well. Let us take a few of the symptoms and consider them closely. The cough is very characteristic; it is a convulsive, frame-shaking cough, often followed by deep inspirations; it is by no means continuous, the patient generally complaining from four or five paroxysms in the day, which sometimes end in vomiting. The greater part of the expectoration is frothy mucus, mostly brought up from the smaller bronchi and from the alveoli, largely intermingled with air. In time, we get the more marked nummular sputa, the inspissated mucin, and the fibrinous casts. Besides these, lung-tissue is soon detected, showing that the basement-membrane is broken, and that ulceration has begun. The odour of the expectoration is very offensive, and has been assigned to the presence of various chemical combinations; to sulphuretted hydrogen; and to lactic and butyric acids, crystals of the latter having been detected in the expectoration. Various fungi have also been found.

The fœtor is of the sickly rotten-egg kind, and contrasts strongly with that of gangrene of the lung, which is much more pungent and overwhelming. The taste of the expectoration, according to the evidence of a sufferer, is very sweet; and this patient stated that it annoyed her more than the fœtor, which was objectionable to her friends rather than to herself.

The aspect of these patients differs entirely from that of phthisical ones. They are generally middle-aged people, with large chests of the emphysematous type, though often with some collapsed portions in the lower mammary regions. They are ruddy, and occasionally livid, seldom pale; in fact, in their appearance, they perfectly show forth the local nature of their disorder; their trouble being, in truth, of a mechanical nature—to wit, difficulty of expectoration. The temperature and pulse vary little from the normal, unless septic pneumonia sets in; and they then follow the pneumonic type. The breathing, on the other hand, is quickened. The appetite is good, and gain of weight more common than loss.

Physical Signs.—It must always be remembered that dilatation of the bronchi is not a disease of itself, but an affection secondary to other diseases; and therefore we must expect it to be marked by the physical signs of its accompaniments, such as emphysema and pneumonia, fibrosis of the lung, and occasionally pleuritic effusion. The signs are, of course, those of a limited excavation, modified by those of the surrounding tissues. Where much emphysema is present, it is impossible to diagnose dilatation of the bronchi by physical signs, as a careful examination of the whole chest may fail to detect one point where

even tubular breathing is audible. This is sometimes the case when the expectoration gives undoubted proof of the existence of ulceration of the bronchi, and where, from the quantity discharged at certain intervals, it is obvious that certain portions of the bronchi must be dilated.

As regards the actual character of the sound heard, there is no definite distinction to be laid down between the sounds of bronchiectasis and those of a limited cavity; but much may be learnt by noting the position of these sounds. In many of the text-books, it is laid down that dilatation of the bronchi shows itself by cavernous sounds in the lower portions of the chest, and especially in the mammary and interscapular regions, and not at the apices. This, however, is an erroneous statement, as dilated bronchi are not limited to special regions of the chest. The specimens I have just shown you prove decidedly that dilatation may take place in any bronchus and in any part of the lung; and it is as possible to have the signs of a dilated bronchus in the first interspace as at the posterior base, or in the scapular region as in the interscapular. Far more importance should be laid on—(1) the extent of surface over which the cavernous sounds are audible; (2) on their jarring nature; and on their (3) combination with the sounds of emphysema. When there is a dilated bronchus near the apex of the lung, the cavernous sounds are heard in the first interspace; not midway between the sternum and the shoulder, but nearer to the sternum than the sounds of a tuberculous cavity. There is resonance too; and you are surprised that, with such loud cavernous sounds, amphoric breathing is not audible. Where the ectasia is tolerably superficial, and has no emphysematous lung in front of it, or if it has only a layer of fluid separating it from the chest wall, the tracheal or amphoric note is often to be detected; and this generally occurs in the upper regions of the anterior chest.

The termination of bronchiectasis varies; but, if extensive and involving a large number of tubes, it is generally fatal. The contents of the dilatation, which sometimes are so extensive as to reach the pleural surface, set up further ulcerative processes, and may open into the pleural cavity and cause pneumothorax. Two of Dr. Grainger Stewart's cases and four of Biermer's proved fatal in this way. When this is not the case, the inflammatory changes round the tubes often assume the gangrenous form, and either gangrene of the lung or foetid abscess is the result, from which the patient may sink exhausted.

A third ending, and one which has been the commonest in my experience, is death by septicæmia. Some of the foetid material is inhaled into the healthy lung, where it induces septic pneumonia, which soon proves fatal.

SYRUP OF BUCKTHORN.—At Salisbury, an inquest was held lately on the body of a child, the son of a woman named Crocett, who had died from a dose of buckthorn, administered by the mother to alleviate the irritation caused by the child having been vaccinated. Mr. Gowing stated that buckthorn was never prescribed by the profession, and was generally used for dogs. Diarrhœa and contraction of the arms and legs resulted, and the child died in a few hours from exhaustion. A verdict of death from natural causes was returned, and the jury were strongly of opinion that the poorer classes, who frequently used buckthorn, should be warned against it.

POISONING BY ATROPIN SUCCESSFULLY TREATED BY HYPODERMIC INJECTION OF PILOCARPIN.—Professor Purjesz relates, in the *Pester Med.-Chir. Presse*, Nos. 15, 17, and 18, 1880, the case of a hospital patient, aged 19, under treatment for aortic insufficiency and keratoiritis, who drank with suicidal intent the contents of a bottle of solution of atropin which was used for dropping in his eye. The quantity of atropin taken was about nine-tenths of a grain. The pupils were dilated to the maximum, the face was pale; respirations frequent, 40 to 50 in the minute; pulse 140 to 150. The patient was perfectly unconscious; sensibility was not lost. Relying on the observation by Luchsinger of the physiological antagonism between atropin and pilocarpin, Dr. Purjesz injected in succession six syringefuls of a one per cent. solution of pilocarpin (about 0.9 grain in each); and repeated the injection at intervals from five to eight minutes. After the tenth injection, the patient began to speak; the pupils were distinctly narrower; and, as the secretion of saliva and sweat had not yet appeared, another injection of nine-tenths of a grain of pilocarpin was made. After this dose, the patient gave rational answers; the pulse fell to 80; the pupils regained their normal size, but reacted rather slowly to light; the respiratory movements became normal, the skin moist, and all the symptoms of poisoning by atropin disappeared.