On dilatation of the bronchi, or bronchiectasis / by T. Grainger Stewart.

With the Author's Compts

ON

DILATATION OF THE BRONCHI,

OR

BRONCHIECTASIS.

BY

T. GRAINGER STEWART, M.D., F.R.S.E.,

PATHOLOGIST AND EXTRA PHYSICIAN IN THE ROYAL INFIRMARY, PHYSICIAN TO THE ROYAL HOSPITAL FOR SICK CHILDREN, LECTURER ON GENERAL PATHOLOGY AT SURGEONS' HALL, EDINBURGH.

EDINBURGH: PRINTED BY OLIVER AND BOYD.

MDCCCLXVII.

REPRINTED FROM THE EDINBURGH MEDICAL JOURNAL FOR JULY 1867.

ON DILATATION OF THE BRONCHI.

DILATATION of the Bronchi has received less attention in this country than its importance seems to warrant. The following paper embodies the result of an inquiry into its pathology and symptoms, carried on at intervals during the past few years, when examples of the disease happened to present themselves in the Infirmary.

The condition was discovered by Laennec, and the latest extended account of it which I have been able to get, is by Professor Biermer, of Zurich, who, while *privat docent* at Wurzburg, wrote on the malady. As between these two, and even since the latter wrote, not a few observations have been published, it may be convenient to subjoin a list of authorities, so far as I am acquainted with them.[1]

[1] Laennec, Traité de l'Auscultation médiate, tom. i. p. 206.
Andral, Clinique médicale. English, by Spillan. P. 298.
Andral, Precis d'Anatomie pathologique, tom. ii. p. 496.
Reynaud sur l'Obliteration des Bronches, Mémoires de l'Académie Royale de Médecine, tom. iv. p. 117.
Stokes on Diseases of the Chest, 1837, p. 148.
Corrigan on Cirrhosis of the Lung, Dublin Medical Journal, 1838, p. 266.
Carswell's Pathological Anatomy, 1838.
Williams (C. J. B.), Pathology and Diagnosis of Diseases of the Chest, 1840, p. 96.
Briquet, sur un Mode de Gangrene du Poumon, Archives générales, tom. xi. ser. iii., 1841, p. 1.
Beau et Maissiat, Archives générales de Médecine, tom. iii. ser. iv., 1843, p. 283.
Fauvel, sur la Bronchite capillaire suffocante, Mémoires de la Société Médicale d'Observation de Paris, tom. ii., 1844, p. 480.
Hasse's Pathological Anatomy, Syd. Soc. Trans., p. 300.
Barlow, Guy's Hospital Reports, 1847, p. 180.
Gairdner on the Pathological Anatomy of Bronchitis, 1850, p. 75.
Cruveilhier, Traité d'Anatomie pathologique, tom. ii., 1852, p. 874.
Barth, Recherches sur la Dilatation des Bronches, Mémoires de la Société d'Observation de Paris, tom. xxx., 1856, p. 469.
Lebert, Traité de l'Anatomie pathologique, 1857.
Bièrmer, zur Theorie und Anatomie der Bronchienerweiterung, Virchow's Archiv, band xix. p. 94.
Rokitansky, Pathologische Anatomie, 1861, band iii., p. 7.
Luys, Etat anatomique du Poumon dans la Dilatation des Bronches, Archives générales, 1862, tom. xx., serie v., p. 735.
Laycock, Notes on Fetid Bronchitis, Edin. Med. Journal, May 1865.
Gamgee, on the Character of Expectoration in Fetid Bronchitis and Gangrene of the Lung, Ed. Med. Journal, March 1865.
Gintrac (Henri), Nouveau Dictionnaire de Médecine et Chirurgie, tom. v. p. 622.

Besides these, I find quoted the following:—
Gelez (Ed.), de la Dilatation des Bronches, etc., Thèse de Doctorat, Paris, 1844, No. 145.
Rapp (G.), Verhandlungen du Physikal. Med. Gesellschaft zu Wurzburg, band i., 1850.

To many of them we shall have occasion to refer, as we proceed. Treading a path which has been so often trod before, it cannot be expected that I should bring forward many novel facts or opinions; but all the statements that I have to make have been verified by myself, and one or two are, I think, new. But, apart from considerations of novelty, the publication of a paper on the subject may supply a want at present existing in English medical literature.

It may be well in starting to clear the ground, by enumerating the various changes in and connected with the bronchi which have been confounded with the lesion under consideration; for, while this lesion, the true bronchiectasis, which is an important disease, arises, as I shall endeavour to show, quite independently of all other pulmonary affections, there are several minor varieties which result from other changes in the lungs; and much of the confusion which at present exists is attributable to the fact, that authors have not been sufficiently careful to mark them off from the disease which we have under consideration. There are,—

1*st*, *A general dilatation of the bronchi from hooping-cough, or from capillary bronchitis.*—Of this M. Fauvel relates, in his valuable paper on Suffocative Capillary Bronchitis,[1] that in a considerable number of his cases he found a general or partial dilatation of the bronchial ramifications. Tolerably uniform in calibre, it was most marked in the finer tubes, where the cartilages are either rare or wanting. It appeared to result from long-continued violent inspiratory efforts, in the course of which the air, being hindered from passing further in its natural direction, expanded the delicate bronchial walls. His observations are amply confirmed by Barthez and Rilliet. This form of dilatation is doubtless often recovered from: the tubes resuming their natural calibre when the dilating cause has been removed.

2*d*, *Dilatations from stricture of bronchi.*—This variety of bronchiectasis was first pointed out by M. Reynaud, in his paper on

Gombault (U. C. A.), Etude sur l'Anatomie pathologique, etc., Thèse de Doctorat, Paris, 1858.

Casalis (E. A.), Considérations sur la Formation des Dilatations bronchiques, Thèse de Doctorat, Paris, 1862.

Besnier, Anatomie pathologique, Causes, et Diagnoses de la Dilatation bronchique, Union Médicale, 1859, tom. iii. p. 469.

Lancereaux, Dilatation moniliforme d'un grande Nombre d'Extrémités bronchiques, Bullet. Soc. Anatom., 1861, p. 92.

Katz, Dilatation des Bronches, Thèse de Strasbourg, 1864.

Empis, Dilatation des Bronches, Gazette des Hôpitaux, 1863.

Van Genus, Over Bronchectasis, Nederlandsche Lancet, July and Aug. 1854.

Bamberger, Bemerkungen über die Bronchectasis Sacciformis, Oesterr. Zeitschrift, f. prakt. Heilkunde, 1859.

Cejka, Prager Vierteljahrschrift, 1863.

Holzhausen, Ueber Bronchectasie, Jena, 1865.

Skoda, Wiener Allg. Med. Zeitung, 1864.

Bamberger, Wurzburger Med. Zeitschrift, 1864.

[1] Mémoires de la Société Médicale d'Observation de Paris, 1844.

the Obliteration of Bronchi.[1] It takes place on one or both sides of the stricture, and is manifestly connected with that condition. It probably owes its origin to the increased pressure of the air in inspiration, or in forcible expiration, which naturally tends to expand the walls on either side of the constricted part.

3*d*, *Dilatations from long-standing indurations of lung substance, tubercular or inflammatory.*—These are always slight, local, and unimportant. They appear to result from retraction of the altered pulmonary tissue around them.

4*th*, *Spurious Bronchiectases*, which are in truth not bronchial at all, but the remains of chronic tubercular cavities, or abscesses in the lung tissue, which communicate with bronchial tubes.

Such are the minor varieties which we must bear in mind. It remains for us now to consider the most important form, the elucidation of which is the object of the present paper.

There are two forms of bronchiectasis, viz., the general, or uniform; and the sacular, or ampullary.

1*st*, *The general, or uniform*, is a cylindrical or fusiform dilatation of a tube, or of several tubes, throughout considerable stretches of their extent.

2*d*, *The sacular, or ampullary* is, as the name implies, an abrupt dilatation of a tube at a particular point, or at several points. Many writers distinguish two forms—the *solitary*, when but one sacular dilatation exists in a tube; and the *moniliform, or bead-like*, when a succession of them is met with. This distinction is, however, unnecessary, as solitary saculated dilatations are exceedingly rare, if ever they are met with. Indeed, it is almost doubtful whether it is necessary to distinguish between the cylindrical and the sacular, they so constantly co-exist in the same lung, and even in the same tube—the whole course being dilated, but special sacules projecting from the side here and there.

When dilatations exist in neighbouring bronchi, it not unfrequently happens that communications become established between them, and this may go on to such an extent that several bronchial tubes open into one common cavity. This fact was observed by Cruveilhier, but has hitherto attracted less attention than it deserves.

In considering dilatations more minutely, we shall first describe—

The Condition of the Bronchial Walls.

(1.) *Of the Mucous Membrane.*—In some dilatations this membrane is natural. Its surface may be coated with mucus, but the epithelium presents its ordinary characters, the cilia are distinct, the nuclei not enlarged, and the subjacent vascular layer is neither congested nor thickened. In others the membrane is thickened and opaque, the epithelial elements, though still distinct and

[1] Op. cit., p. 876.

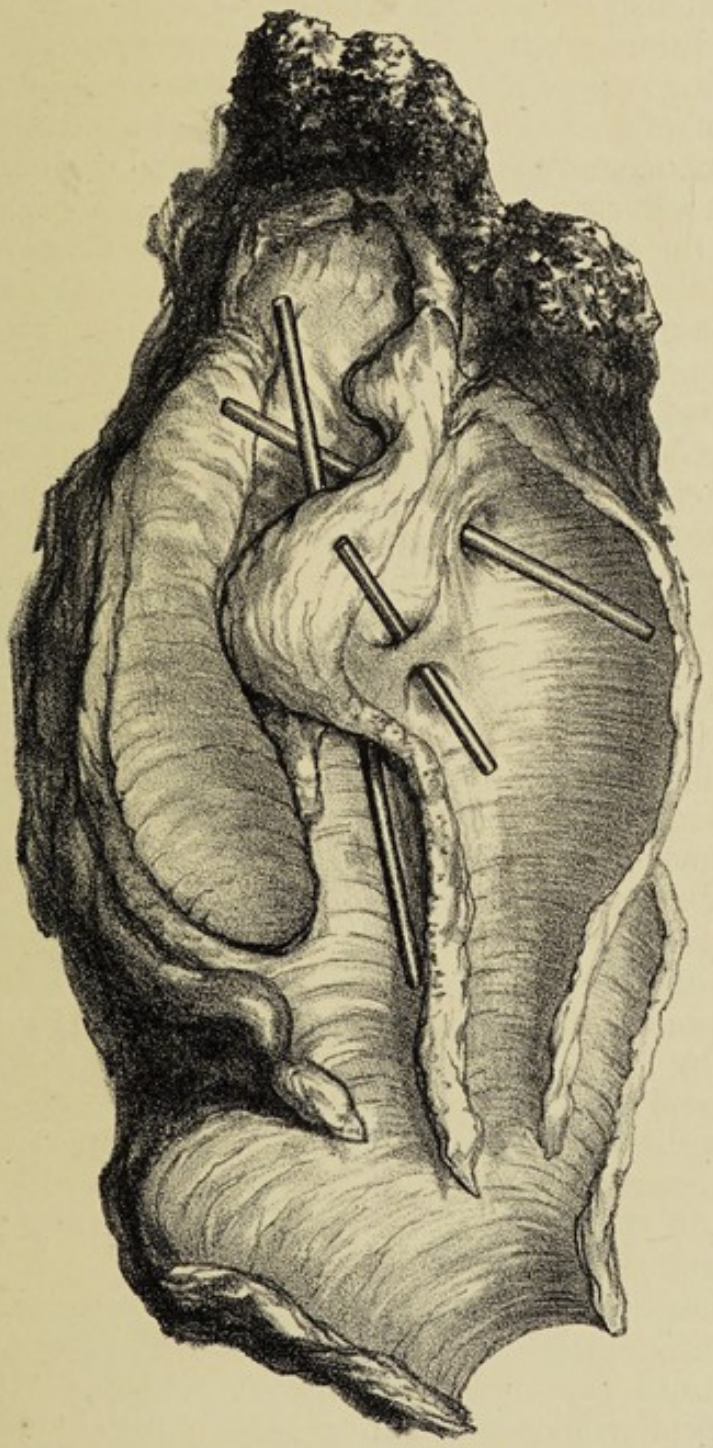
characteristic, are granular and swollen, and the vessels are congested. In yet others, the membrane presents a velvety appearance, numerous villous processes containing congested vessels projecting from the surface. In this condition the epithelium is more altered, though still distinctly characteristic. In yet others, ulcerative or necrotic destruction of the membrane is seen: superficial or deep, limited or extensive abrasions existing.

These conditions are met with in successive stages of the affection, the first being seen only where the dilatation is slight, and the accumulation of mucus inconsiderable; the last only where dilatation is advanced, and decomposition of retained mucus has occurred. It is to the changes in the contents of the tubes, then, that I would refer the changes in this membrane, not to a morbid action originating in itself. In the earlier stages the membrane is unaltered, but, as the secretion accumulates, it becomes granular and opaque; further accumulation and decomposition of the retained material produces, of course, greater irritation, the villous condition, and ulceration.

(2.) *Of the Muscular, Elastic, and Cartilaginous Structures.*—Even in the slighter dilatations, in which the mucous membrane is quite normal, there is distinct wasting of the muscular and elastic coats. Coincidently with this wasting the dilatation of the tubes increases and occurs as well in the cylindrical as in the sacular dilatation. On examination with high powers the tissues appear granular and indistinct, so that the individual elements of the muscular and elastic fibres can scarcely be recognised.

The atrophy sometimes advances in a very remarkable manner, particular portions of the wall wasting, and others retaining their natural volume; the latter parts form bands or ridges elevated above the surrounding mucous membrane. As the atrophy of the neighbouring parts advances, these become more and more prominent, drawing gradually closer till they resemble a mesentery connecting the unatrophied band with the bronchial wall. At length, apparently by gradual absorption, the mesentery-like membrane disappears, and the band is left as a bridge stretching across the lumen of the dilated tube. The process reminds one of the formation of the fenestrated openings which are often met with towards the margins of the valves of the aorta and pulmonary artery, and which are sometimes seen in the mesentery. It appears that the communications between adjoining bronchiectases are formed by a process identical with the above, neighbouring dilatations expanding until two of them come in contact. Through their walls an opening forms, which gradually enlarges without any truly ulcerative process. In the smaller as well as the larger tubes such bands and bridges are met with; the communications to which I have referred are of course most common in the expansions towards the extremity of the former.[1]

[1] See the lithograph, which shows communications between neighbouring bronchi and a bridge within one of the cavities.



W.H.M.ᶜ Farlane, Lithᵖ Edinʳ

Group of dilated Bronchi
shewing communications at different points.

Many tubes present an appearance which, at first sight, might be taken for hypertrophy. The walls certainly are somewhat thickened, but that thickening depends upon changes in the mucous membrane already described, and the presence of ill-formed connective tissue among the denser elements of the walls, not on increased growth of these elements.[1] The irritation which causes the inflammatory thickening of the mucous coat may well also account for the spurious hypertrophy in the other.

In the earlier stages the cartilaginous plates are unaltered, but when the irritative changes occur in the tubes they frequently participate; and Andral[2] and Biermer[3] both describe outgrowths as occasionally met with projecting from them. Such outgrowths must manifestly result from extreme irritation.

The Contents of the Dilated Tubes.

Bronchiectases usually contain much mucus; but in different cases, and in different parts of the same lung, it may be found in very different conditions.

1. There is in some cases a copious yellow mucus quite free from fetor, and presenting the characters of the natural secretion both to the naked eye and on microscopical examination. On agitation with water, casts of smaller bronchi, containing copious epithelium, may be separated, the epithelium presenting a natural or only slightly granular appearance.

2. In other cases we find an inspissated mucus of a grey-yellow and rather opaque colour, and usually free from any disagreeable odour, and frequently partially adhering to the walls. On microscopic examination some altered cells may be recognised among this material, but it is mostly composed of granular and fatty matter.

3. In other cases the casts of the tubes are opaque and greyish in colour, and, on the application of iodine, assume a purplish hue, as was first pointed out by Dr Arthur Gamgee. When such casts are present the mucus is generally very fetid, and, on microscopic examination, innumerable fine needle-shaped crystals of margarin are seen. It appears that these products of decomposition always co-exist with the fetor.

[1] Biermer describes three varieties: — 1. Dilatation with inflammatory thickening of the walls, the mucous membrane often villous. This might be called the simply hypertrophic form, no atrophy being cognizable. The dilatation is always cylindrical or slightly spindle-shaped. 2. Dilatations with pure atrophy of the walls, which occur specially in the sacular form. 3. Dilatations with mixed degeneration of walls,—partly atrophic, partly hypertrophic. In this form atrophy occurs first, and is followed by secondary growth or hypertrophy.

I have not met with any example which I could refer to the first category; of the second and third I have seen many examples, and it appears to me that if the causes leading to the secondary hypertrophy were continued in operation for a considerable time, we should have a condition closely resembling Biermer's first form produced.

[2] Andral, Clinique Médicale, tome i.

[3] Biermer, op. cit., s. 142

4. Sometimes mingled with the strictures just described we meet with fragments of pulmonary tissue, such as are commonly seen in the contents of tubercular vomicæ.

5. The contents of the bronchiectases sometimes become inspissated, hard, and even calcareous. I have met with the two former conditions, but never with the last.

It is obvious that most of these varieties may result from changes in the natural bronchial secretion, and this is, in truth, the case. That material, being retained in the sacules, becomes inspissated and opaque, passes from a yellow to grey colour, gradually undergoes decomposition, in the course of which the peculiar fetor is produced, and the fatty crystals are formed. The fetor is peculiar, quite distinct from that of gangrene, often very nauseous, and, as the disease advances, it becomes worse. It is very possible that the grey membranous-looking casts of the tubes may be formed by fibrin poured out from the inflamed mucous surface. The shreds of pulmonary tissue afford evidence, wherever they are met with, that a destruction of lung tissue has taken place. The only other elements which appear worthy of notice are cholesterine crystals, which are now and then seen, and fungi, which are met with occasionally.

As to the *chemical composition* of the matters described, we have, in the meantime, little information,—the discussion has turned mainly upon the question of the cause of the fetor. Professor Laycock[1] concludes, from experiments and observations made by the late Professor Gregory, Dr Arthur Gamgee, and himself, that the odour must be due to butyric acid. He also states that Dr Gregory detected the odour of methylamine in some of the products of the sputa. Professor Bamberger[2] concludes that the characteristic smell of the sputa in bronchiectasis appears to depend upon a variety of odorous matters, among which the members of the series of acids of the type $C_n H_n O_4$, particularly butyric and formic acids, ammonia, and sulphuretted hydrogen, all of which may proceed from the decomposition of organic substances. He further states that purulent sputa,—*e.g.*, that of tubercular patients,—sometimes undergoes the same decomposition out of the body, and, if long kept, have the same smell as the sputa in question. Dr Arthur Gamgee,[3] from a considerable number of analyses of sputa, concludes that the occurrence of butyric acid cannot at present be proved to have any semeiological value, and that its presence is in no way characteristic of fetid bronchitis, under which term he

[1] Professor Laycock, Medical Times and Gazette, May 1857, p. 479; and Edinburgh Medical Journal, May 1865.

[2] Wurzburger Medizinische Zeitschrift, 1864. Quoted in Medizin Jahrbuch, bd. xii.

[3] Edinburgh Medical Journal, March 1865. Professor Laycock connects fetid sputa with the rheumatic diathesis, and conceives that when it exists there may be an excess of lactic acid in the pulmonary excretion, or of some other acid of the same class, or that the ordinary series of decompositions is interrupted, and other products result in virtue of vital changes, whereby an irritant is brought into contact with the bronchial mucous membrane. If this be

includes bronchiectasis. He found that the distillate passed over acids and alkalies unchanged, and that, whatever the substance is, it is rich in sulphur.

With such discrepant statements as to matters of fact before us, we cannot at present conclude what is truly the nature of the material in question.

The Condition of the Lung Tissue surrounding the Dilatations.

1. In some cases the lung tissue is unaltered, soft, spongy, crepitant; to the naked eye as well as our microscopic examination it appears natural.

2. In others it is collapsed or atrophied. The tissue is not spongy, and does not crepitate; but it is not indurated, and, on microscopic examination, no excess of the fibrous element is to be detected. The atrophy in some cases advances to such an extent that nothing is left in the affected part but dilated bronchi.

3. In others the tissue is consolidated, the consolidation often of a very dense description, constituting what some have called cirrhosis, and others fibroid degeneration of the lung. In this condition it is asserted that, along with occlusion of the air-cells, there is a marked increase of the fibrous tissue of the organ; and this change Sir Dominic Corrigan[1] considers identical in its nature with cirrhosis of the liver. But this whole subject appears to me to be not very well cleared up. The most recent account of it is that given by Dr Sutton in the Medico-Chirurgical Transactions of London.[2] The disease which he there describes, he says, is identical with the cirrhosis of the lung of Corrigan. The principal features, as he describes them, are,—the formation of fibrous tissue in every direction throughout the lungs at the expense of the proper pulmonary

sufficiently powerful, the same consequences will follow as with other powerful irritants (such as the vapour of iodine or chlorine), and thus a bronchitis with a copious muco-purulent discharge be excited. How easily lactic acid may undergo chemical changes is indicated by the fact, that when lactates are left in contact with putrefying matter at a temperature of 86° to 96° Fahr., butyric acid is produced.

Bamberger gives the results of the examination of two cases. In the extremely fetid sputum of one he found sulphuretted hydrogen, butyric, lactic, and, very probably, formic acids, and neither leucin, tyrosin, caprin, nor caprylic acid. But this was not a very fair example of the disease, for the patient, a man of fifty, had for fifteen years been affected with bronchiectasis, which had resulted in gangrenous ulceration and perforation into the right pleura, with the formation of limited pneumo-thorax, and the purulent matter thus formed was mingled with the expectoration. A second case afforded a purer example. A man of forty, who for several years had suffered from bronchiectasis, brought up large quantities of sputum with the characteristic odour. Its reaction was alkaline, and it contained ammonia, butyric and acetic acids, while sulphuretted hydrogen did not appear till after the matters had stood for twenty-four hours. Formic acid could not be detected.

[1] Op. cit.

[2] Medico-Chirurgical Transactions, 1865.

structures: this formation being constitutional, often associated with a similar growth in the liver, kidneys, and spleen, totally different from tubercular deposits, and affecting individuals of a particular constitution, certainly not scrofulous. Now, it seems to me that Dr Sutton gives in outline an account of a form of disease to which the name cirrhosis might very well be applied, but which is essentially different from the cirrhosis of Corrigan, inasmuch as the former is generally distributed throughout the lung, the latter is more localized; the former occurs in nodules, the latter in extensive masses; the former is not specially associated with dilatation of the bronchi, while the latter is. These characters seem sufficient to distinguish the two forms of disease. From my own examinations of cases of undoubted cirrhosis (Corrigan), it appears that the induration results from chronic inflammation, and consists not merely of such an increased growth of fibrous tissue as one sees in a cirrhotic liver, but partly of a granular thickening of the stroma of the lung, certainly with increase of fibrous tissue, and partly of inflammatory products, which are deposited in the air-cells. Such a condition as I have just described is often met with around bronchiectases. In the examples of the disease described by Dr Sutton, which I have seen, there was no special dilatation of the bronchial tubes.

4. In other cases, the surrounding tissue forms an abscess, in the centre of which the thin walls of the dilated bronchus may be seen.

5. In other cases, the walls of the bronchi and the surrounding lung tissue are destroyed by gangrenous inflammation. M. Briquet[1] was the first to draw attention to this variety of pulmonary gangrene, and his observations have since been confirmed by Dietrich[2] and others. The chief conclusions arrived at by Briquet were, that there is a form of dilatation of the bronchi in which the extremities of the tubes are dilated in sacs, which may be with or without dilatation of other parts of the bronchial tree; that these dilated extremities are liable to gangrenous destruction independently of gangrene elsewhere. It appears probable that Briquet reckoned some cases as affording examples of this variety of gangrene, which were really such, but my own experience has distinctly confirmed his general conclusions above stated.

All these conditions of the pulmonary tissue appear to be justly referable to the changes within the bronchi. The induration, the ulceration, and the gangrene, may alike be explained on this theory. The influences which suffice to irritate the bronchial wall must, if continuously or intensely applied, affect the structures lying beyond them, and the changes we have described are exactly such as would naturally follow such irritation.

[1] Briquet, Mémoire sur un Mode de Gangrene du Poumon, dépendant de la Mortification des Extremités dilatées des Bronches, Archives générales de Médecine, 1841.

[2] Dietrich, ueber Lungenbrand, quoted by Gintrac.

The *Pleura* is in many cases adherent and in some is very singularly thickened, but, in a considerable proportion of cases, it presents no such abnormality.

The form of the Chest.—In four examples of the disease which I have observed in the hospital, the chest was more or less deformed; but the number of my cases does not entitle me to form an opinion as to the importance of this complication.

Seats of Bronchiectases.—The lower lobe and the middle lobe of the right appear to be the most common seats of this change. It occurs towards the apices sometimes, but much less frequently; usually it affects many bronchi, and occurs in both lungs.

Theories as to the Nature and Origin of Bronchiectasis.

The opinions expressed as to the origin of the lesion are very various. Many may be passed over without notice, but there are several which deserve special attention.[1] Those to which I think

[1] In the following note the principal theories not given in the text are explained.

Laennec ascribed the dilatation to stagnation of the bronchial secretion and mechanical pressure on the walls. (*Op. cit.*)

Andral recognised three forms of bronchiectasis; refers two of them, in which he says thickening of the bronchial walls exists, to hypertrophy of the walls, but does not explain how such a change could lead to such a result. His third form, the beaded, is, he says, usually accompanied by thinning of the walls, and in it he thinks that the dilatation may be a mechanical result of their distention by mucus at the points where their parietes present least resistance, either by diminution of their elasticity or by their walls really becoming thinner. (*Op. cit.*, p. 298.)

Reynaud conceived the dilatations to be compensatory to the atrophy or collapse of neighbouring parts, the pressure of the inspired air acting with greater force on the diminished respiratory surface. (*Op. cit.*)

Williams lays great stress upon the importance of the influence which is continually exerted between the interior of the bronchial tree and the air,—in inspiration by the air which enters to distend the tubes, in expiration by the tubes contracting to expel the air,—and points out how, if the equality of the pressure be disturbed by more forcible pressure of the air on individual parts or on all parts, by loss of elasticity and contractility of the bronchial walls, dilatation of the tubes must occur. He also directs attention to the connexion of the cirrhosis of the lung with dilatation of bronchi, and claims priority to Sir Dominic Corrigan in this observation. (*Op. cit.*)

Cruveilhier ascribes it to cough, and the accumulation of secretion. (*Op. cit.*, tom. ii. p. 883.)

Barth says that it may arise from changes,—1*st*, in the pleura, pleuritic adhesions drawing the bronchi open; 2*d*, in the lungs, according to Corrigan's view; 3*d*, in the bronchi, according to Stokes' view. These causes, singly or even in conjunction with one another, induce the results. He conceives, further, that an important influence is exerted by the presence of the air, which, being forcibly drawn in through the accumulated mucus, does not pass out with expiration, and so distends them. (Barth, *loc. cit.*)

Other writers adopt either a modification of the theories given in the text, or in this note.

we should specially attend are those of Corrigan, Gairdner, Stokes, and Lebert.

I. *The Theory of Corrigan.*[1]—Having described and figured the condition of the pulmonary tissue, to which he applied the term cirrhosis, he says: "The dilatation of the bronchial tubes is partly owing to the contractile process going on in the tissue of the lung—partly to the expansive action of the parietes of the chest in the act of inspiration." And he explains this contractile influence thus:—"If there were but one bronchial tube with contracting fibro-cellular tissue placed around it, then the contracting tissue would, as in the instance of stricture of the œsophagus or rectum, cause narrowing of the tube; but when there is, as in the lung, a number of bronchial tubes, and the contracting tissue not placed around the tubes, but occupying the intervals between the tubes, then the slow contraction of this tissue will tend to draw the parietes of one tube towards the parietes of another, and necessarily will dilate them." This dilatation is further favoured by each expansion of the chest, which tends to draw open the tubes. Such is the theory of Corrigan.

This opinion has been very generally accepted both in this country and on the Continent, but certain difficulties present themselves which show it to be untenable.

1. That many dilatations are not surrounded by condensed pulmonary tissue, as has been already shown.

2. That retraction by fibrous tissue does not account for some of the anatomical changes met with in the disease, particularly the formation of bridges in the bronchi, and of communications between neighbouring dilatations.

3. That, at least in one of my cases, where consolidation ultimately occurred, it was clinically proved that dilatation preceded the condensation of the pulmonary tissue. (See Case IV.)

These considerations appear to render Corrigan's theory untenable.

II. *The Theory of Prof. Gairdner.*[2]—This author says that almost all the so-called bronchial dilatations, and all of those presenting the abrupt saculated condition, are in fact results of ulcerative excavations of the lungs (tubercular or other) communicating with bronchi, and expanded beyond their original size by the inspiratory force.

I am satisfied, as I indicated in the commencement of this paper, that in some supposed dilatations this explanation is correct, as appeared to be shown in a case exhibited by Dr Sanders to the Medico-Chirurgical Society, and recorded in the Edinburgh Medical Journal for February 1866; but in a great majority of cases the explanation cannot hold good, and that for several reasons.

1. That in many cases of bronchiectasis there is no tubercular disease of the lungs, and no evidence of non-tubercular ulcerations.

2. That many dilatations may be distinctly shown to be in the line

[1] Dublin Medical Journal, vol. xii., 1838, p. 270.

[2] On the Pathological Anatomy of Bronchitis, etc. Edinburgh, 1850, p. 76.

of bronchial tubes, and the bronchi may not only be traced into them at one extremity, but out of them at the other.

3. That the mucous lining of many of them presents the ordinary character of bronchial mucous membrane.

4. That they are not common towards the apices, where tubercular excavations are most frequent.

III. *The Theory of Stokes.*[1]—Dr Stokes conceived that most importance must be attached to loss of elasticity, contractility, and ciliary movement, and that the starting-point is in all cases bronchitis. From inflammation, he said, 1*st*, The non-muscular longitudinal fibres lose their elasticity, and so cough and forced inspirations may dilate the tubes. 2*d*, Paralysis of the circular muscular fibres, whereby the tubes dilate on the same principle as the intestines do in cases of enteritis or ileus. 3*d*, The cilia of the epithelium lose their mobility.

This view appears to be correct, in so far as it assumes certain changes in the bronchial walls, but in the explanation of the origin of these changes, it appears that there are certain difficulties,—1*st*, That if bronchiectasis depended simply on bronchitis, it would necessarily be much more frequent than it is. 2*d*, That this theory affords no satisfactory explanation of the formation of the bridges and communications which have been described.

IV. *The Theory of Lebert.*[2]—This author concurs generally in the opinion of Stokes, but thinks the loss of power dependent upon nervous causes rather than upon inflammation of the bronchi. He thus obviates one of the difficulties which beset the view of Stokes; but it must be borne in mind that the supposed influence of the nervous system is purely hypothetical. Still his view is the only one which is not opposed to known facts.

The *conclusions* which appear legitimately deducible from my own observations are,—

1. That the essential element of bronchiectasis is atrophy of the bronchial wall; that the cause of such atrophy is not yet ascertained, but may perhaps be connected with constitutional peculiarities.

2. That the walls, being so thinned and weakened, readily yield to the pressure of air; it may be in deep and sudden inspirations or during violent muscular exertions, certainly in the sudden expiratory effort made while the glottis is closed in the act of coughing.

3. The enfeebled and dilated condition of the bronchi favour the accumulation of the mucus secreted by the bronchial membrane.

4. That the mucus, accumulating and undergoing decomposition in the dilatations, irritates the mucous membrane, leads to inflammation, and the formation of villous processes from it, to the formation of increased connective tissue in the walls, to irritation of the cartilages, and frequently to consolidation of the surrounding lung

[1] Stokes on the Diagnosis and Treatment of Diseases of the Chest. Dublin, 1837.

[2] Lebert's Anatomie pathologique, tome i. p. 620.

tissue and pleuritic adhesions, sometimes also to abscess or to limited gangrene.

The process, then, would appear to correspond in its early stage to what is met with in other parts of the body; as, for example, in aneurism, where, from weakening of the middle coat of the artery, the pressure of the blood causes the vessel to expand, and that to a constantly increasing extent. It also resembles the process by which staphyloma of the sclerotic is formed, in which the membrane, being weakened at certain points, is unable to withstand the pressure of the fluids of the globe, and, consequently, a bulging ensues.

Clinical History of the Disease.

In many, if not in all cases, the affection of the bronchi comes on insidiously, no symptoms appearing to attract the attention of the patient or his friends, perhaps for long after the dilatations have been formed. But gradually or suddenly inflammation of the bronchial mucous membrane is lighted up, and symptoms of bronchitis become well marked. Those of bronchiectasis, though distinct when looked for, are not such as to force themselves on the attention until decomposition of the secretion has taken place; the breath and sputum then become fetid, and the general health obviously suffers. Decomposition of the secretion appears to be followed by consolidation of the lung, and sometimes by ulceration, abscess, or gangrene. Following upon these, we sometimes have a fatal result from perforation of the pleura, leading to empyema and pneumo-thorax (see Case III.). Sometimes death results from exhaustion from the constant discharge of the sputum. Sometimes from a peculiar febrile disturbance,—a kind of septicæmia, probably induced by absorption into the blood of some of the products of decomposition (see Case IV.).

In a great majority of cases, the disease tends to a fatal result, but now and then recoveries occur. Gintrac relates that recovery has been observed in two ways, each of them sufficiently remarkable: 1*st*, from the cretification of the contents, and the conversion of the walls into a sort of fibrous capsule; 2*d*, from penetration of the pleura and thoracic parietes and discharge of the contents outwards. It seems to me that such cures are scarcely credible, and certainly cannot occur in any except those cases which are limited to a single bronchus, or to few.

The *age* at which it occurs is very various. I have met with it in young people, as well as at and after the prime of life. As to *sex*, it appears that, in the majority of cases, it occurs in the male. It is usually stated to be non-hereditary, and not constitutional in its origin; but, from a consideration of its nature, it appears to me probable that it may ultimately be proved to be both; and I cannot help thinking it more than a coincidence, that of the few cases I have observed, two were in members of the same family.

Symptoms.—The symptoms of bronchiectasis which most attract attention are, the odour of the breath, the characters of the sputum, and the cough.

The *breath* is often peculiarly fetid, the fetor somewhat different from that of the sputum, and often intensely disagreeable to the patient himself. It is difficult to find a smell closely corresponding to it, but it is certainly quite distinct from that of gangrene. This symptom is not present in all cases (see Cases V., VI., and VII.), nor is it present in the earlier stages of any case. It is only as the disease advances, and when decomposition of the bronchial secretion has taken place, that it occurs.

The *sputum* is usually copious—15 to 30 oz. daily—of a greenish-yellow colour and nummular, rarely frothy on the surface; sometimes gelatinous, and with a tendency to adhere to the walls of the vessel. Sometimes it sinks, sometimes floats in water; and when a quantity of it is allowed to stand in a glass jar, it separates into layers or strata; a more mucous and fatty layer, which floats on the surface; a more purulent and granular, which sinks to the bottom; and a greenish watery fluid, in which the other two are embedded, but which forms an intermediate layer. In a majority of cases—at least of cases in which this disease is recognised—it is very fetid. The fetor resembling that of the breath, but in a more concentrated form. It is quite distinct from that of gangrene. On agitating the recent sputum with water, more opaque filaments, of varying thickness, separate themselves, and sink to the bottom. These are casts of bronchial tubes. Their character has been sufficiently described in a previous part of the paper. On microscopic examination, one may find the different elements previously described; but I have not seen, as yet, the needles of Margarin, which are found so abundantly postmortem in the recesses of the bronchi.

Hæmoptysis.—Blood is expectorated along with the sputum, sometimes in small quantities, merely streaking the expectoration, which otherwise presents the characters above described; but sometimes it is brought up in large quantities, much as in phthisis. In such cases (see Case V.), the sputum may be not at all abundant; it is only when the hæmorrhage occurs that almost any discharge takes place. Barth met with two cases in which the hæmoptysis proved fatal.

Cough is frequent, and occurs in paroxysms; but moist, and usually quite painless. After a fit of coughing, large quantities of the sputum are brought up without difficulty.

Respiration is generally easy and quiet while the patient is at rest, but exertion speedily induces dyspnœa.

Physical Signs.—There is, in the advanced stages, dulness on percussion over the parts of the lung which have become consolidated, sometimes relieved by an unusually clear note, if a dilatation be situated near the surface, and empty. In the earlier stages the

percussion note is little altered, and I have seen, even when the fetor was distinct, the percussion note most markedly dull.

On auscultation, cavernous sounds are sometimes heard, and very commonly coarse moist rales, sometimes even gurgling. But the situation of these sounds generally serves to distinguish them from those of phthisis; for while the latter are usually situated towards the apices, the former occur towards the middle and lower parts, the apices being comparatively free. In well-marked cases, these symptoms are sufficiently distinctive, but are slighter. These symptoms, generally, might be mistaken for those of gangrene of the lung; but the history of the patient, and the duration of the malady, soon suffice to correct that opinion.

In the later stages, *the pulse* is generally rapid, even independently of the occurrence of inflammatory complications. There is also some *restlessness at night*, sometimes accompanied by *delirium*, although, as a rule, the patients enjoy considerable immunity from nervous complications.

The *digestive* and *urinary functions* are, in most cases, natural; though sometimes the patients complain of nausea and loss of appetite.

Treatment.—With regard to treatment, it is to be feared that little can be accomplished in the way of cure, and that our efforts must mainly be directed towards the alleviation of urgent symptoms. The following points are insisted upon by some continental writers of experience:—With the view of clearing the tubes, by promoting expectoration, Kermes mineral (ter-sulphuret of antimony), ipecacuanha, and tartar emetic, are recommended; to relieve cough, opiates; to diminish secretion, balsamic remedies, as tolu, tar, turpentine, and copaiba; to diminish bronchial secretion, and avert or arrest hæmoptysis, astringents, as catechu and rhatany; while counter-irritants, of various kinds, applied over the chest, and inhalations of medicated substances, are found eminently useful.

It seems to me that we can do nothing to oppose the formation of the dilatations; that it is only in obviating or diminishing the secondary results that we can expect to be useful. Excessive secretion may be relieved by the administration of astringents and balsamic substances, either by the stomach or by inhalation, and often yield to counter-irritation, especially by blisters.

If the disease has advanced to the stage of decomposition of the secretion, and consequent fetor of the breath and sputum, benefit may result from the inhalation of creasote vapour. This is best effected by pouring a few drops of creasote into an ordinary inhaling apparatus, or simply dropping a little into a cup of hot water, and directing the patient to inhale the vapour as it rises. It is certainly in those forms of fetor of breath, which appear to result from an altered secretion rather than a decomposition of natural mucus, that this treatment is most efficacious; but even in cases of true bronchiectasis it may sometimes be found useful.

Appendix of Illustrative Cases.

Case I.—*Bronchial Symptoms of some years' standing—Consolidation of Lung—Death from Exhaustion—Autopsy—Bronchiectasis—Gangrenous Ulceration and Consolidation of Lung.*

Thomas Murray, æt. 34, was under the care of Professor Maclagan. The patient had for some years suffered from short cough, especially in winter, but had not become emaciated or much enfeebled. His cough became gradually more severe, and he was admitted to the Infirmary, 1st June 1865. At that time there was complete dulness on the left side, posteriorly, with loud mucous râles, especially over certain parts, and harshness, with increased vocal resonance. Over the right side, also, there was dulness on percussion; and on auscultation, the breathing was harsh, and accompanied by mucous râles. His breath and sputum had a feculent odour. While under observation, gurgling sounds became more distinct throughout the chest. His pulse was of good strength. Suddenly, and without obvious cause, he became worse, and sank on 16th June.

Autopsy.—The body was not emaciated. Both lungs were adherent; the left was dense and solid throughout the greater part of its extent; the right was consolidated in its upper half. Both contained numerous cavities throughout their substance, but there was no trace of tubercular deposit, and the cavities were evidently formed by dilated bronchi. A large proportion of the bronchi about the third and fourth division were dilated—some large enough to hold a minié rifle-ball. In some parts, the bronchial mucous membrane was thickened and ulcerated; and in the largest cavity, situated about the middle of the left lung, an ulcerative process had completely destroyed the walls of the dilated bronchi, and the cavity was bounded by consolidated lung tissue. The cavities contained purulent and slough-like masses, which had a peculiar feculent odour, and, on microscopic examination, were found to contain, besides numerous molecules and granules, a few vegetable structures and a large number of fine needle-shaped crystals of margarin. In some corners of the dilated parts, there were firm rounded masses of a dense consistence, but not calcareous. The other viscera were natural.

Commentary.—Symptoms of chest affection, though not of an urgent character, had existed for some years; and the lesions were doubtless well advanced when he was admitted on the 1st of June. The sudden increase of illness and the death were probably due to the gangrenous destruction of lung tissue, which had set in around one of the principal dilatations.

Case II.—*Deformity of Chest—Bronchitis, Hæmoptysis—Fetor of Breath, Consolidation of Lung, etc.—Death from Exhaustion—Autopsy—Bronchiectasis with Consolidation, etc.*

John Storrie, æt. 32, a printer, was under the care of Professor Laycock. The patient had been deformed since he was nine months old; had marked lateral curvature; but was of steady habits, and had enjoyed tolerably good health until January 1865, when he had an attack of what he termed influenza. From this he entirely recovered, after five weeks' illness. In July of that year he lay down in the Meadows, while heated, and at night had cough and sore throat. In August, he went to the country, and then spat a little blood. In the end of September he returned to Edinburgh, and again suffered from slight hæmoptysis. His cough and weakness increasing, he was admitted to the Royal Infirmary on 5th November 1865. At that time his pulse was rapid,—120; his cardiac sounds were natural; his appetite was poor, his tongue foul, and he occasionally passed blood by stool. He had no hæmorrhoids. His genito-urinary system was normal. There was dulness over the lower part of the right lung. The respiratory murmurs were harsh, and the vocal resonance increased. On the left side, there was dulness at the apex. The respiratory murmurs were the same as on the right side. The cough was short, frequent,

and troublesome. The sputum was copious, and smelt of moist mortar, while his breath had a fæcal odour. Some amelioration of the chest symptoms followed the inhalation of iodine, but the weakness gradually increased and he died 18th November.

Autopsy.—The body was deformed, in consequence of marked lateral curvature of the spine. The heart was natural. The right lung was densely adherent at the apex, and posteriorly. The left had only a few adhesions at its apex. The lungs were in some parts emphysematous, in others consolidated. There was no recent tubercle, but some old deposits of limited extent. The trachea was natural. Throughout both lungs many of the bronchi were dilated. It was most marked in those about the fourth division, and existed among both consolidated and emphysematous tissue. In the middle lobe of the right lung there were several gangrenous cavities, the largest of the size of a small walnut, the walls of which were composed not of mucous membrane, but of consolidated lung tissue. In its neighbourhood there were numerous intercommunicating dilated bronchi, and many with bridges in their walls. The bronchi contained much fetid and purulent mucus, among which were many casts of natural and dilated tubes, exhibiting cells altered in various ways, granular and fatty transformation of the casts, and many which exhibited the purple reaction with iodine which was first pointed out by Dr Gamgee. Among the more opaque-looking contents of the tubes, there were many of the peculiar crystals of margarin, here and there a few vegetable structures (whose characters I did not determine) were seen, and in the gangrenous cavities and the bronchi connected with them there were distinct shreds of pulmonary fibrous tissue. The abdominal organs presented no important lesion.

Commentary.—It is to be noticed here that the patient was deformed, and that he had exhibited no distinct pulmonary symptom till four months before his death. That those symptoms came on after exposure to cold, and continued till his death. I cannot doubt that the process of dilatation had been going on for a longer period, and that the inflammatory process merely led to some of the secondary changes. It is to be noticed further, that the dilatation existed alike with emphysema and with consolidation of the neighbouring lung tissue, and was present on both sides, although the adhesions were mostly confined to one.

CASE III.—*Winter Cough for some years—Weakness gradually increasing for some months—Death from Empyema and Pneumo-thorax—Autopsy—Bronchiectasis, Empyema—Limited gangrenous Perforation.*

John Storrie, a tailor, aged 59, admitted under my own care, 15th October 1866. He was a tall, sallow man, with a somewhat greasy skin, and with rounded shoulders. He stated that he had enjoyed good health, though subject to a cough, particularly in winter; that he had had little expectoration, and that his breath never had a disagreeable smell; but in the summer and autumn he had felt weak, and for five weeks before admission had been unable to work. He was uncle to the patient John Storrie, Case II. On admission, the breath was offensive, the sputum copious, yellowish-green in colour, and with more solid grey streaks here and there. These streaks, on being washed out, were found to be casts of bronchial tubes, in the condition of degeneration, and they assumed a violet-blue colour with iodine. The lungs were nowhere markedly resonant on percussion, but nowhere absolutely dull. Tubular breathing and coarse moist râles were heard over many points of both organs, and towards the base of the left lung there was distinct amphoric breathing. The voice was husky, and the digestion was feeble; there was no other morbid condition. There was no difficulty in this case in establishing a diagnosis of dilatation of the bronchi. The patient was ordered good nutritious diet, and to inhale the vapour of creasote. He continued in much the same state until the 20th, when a quantity of thin yellowish pus was expectorated, altering the general appearance, though not the smell of the sputum. After this he became rapidy weaker, and died 25th October.

Autopsy.—The heart was natural, the aorta atheromatous, the two layers of

the pleura were generally adherent, but on the left side at two points there were limited deposits of pus. Many of the bronchi were dilated throughout the whole of both lungs. In some parts, their walls were thin and free from inflammation; in others, thickened and deeply congested; in yet others, it was partially destroyed. The dilatations were filled with a peculiar kind of matter, of a most offensive odour. Among this matter, casts could be traced, similar to those expectorated during life, and fine crystals of margarin. It was evident that these changes of the mucous membrane were secondary to the dilatation. At some of the expanded portions, the ulceration had extended to the lung substance and formed limited gangrenous patches. For the most part, however, around the sacules the lung tissue was indurated and pneumonic, but did not present such an appearance as to deserve the name cirrhosis. Each of the limited empyemata communicated with dilated bronchi; but it appeared from the history, as well as the pathological appearances, that perforation had taken place into the lung. The dilatations were irregular, and everywhere accompanied by thinning of the bronchial walls—many parts presented the trabeculæ bridges and communications between neighbouring bronchi, which I have described in the general part of this paper. The other organs presented no important lesion.

Commentary.—The physical signs, the peculiar appearance, the fetor of the breath, and the character of the sputum, were eminently characteristic. There was no hæmorrhage, and the general symptoms appear to have been quite trifling for long after dilatation had been established. From the general dulness on percussion, I had failed to detect the limited purulent deposits in the left pleural sac, but I doubt not they existed at the time of his admission, and merely opened into the bronchi at the time that expectoration of pus began. The adhesions were so dense that no considerable pneumo-thorax occurred when the openings became established. The lungs afforded admirable illustrations of the disease.

CASE IV.—*Deformed Chest—Bronchitis, slight Hæmoptysis—Form of Cavity—Consolidation of Lung—Death from Septicæmia—Autopsy, Bronchiectasis, etc.*

Neil Mackinnon, æt. 57, a labourer, was admitted to the Royal Infirmary, under my own care, 14th September 1865. Was a Highlander, of dusky complexion and peculiarly oily skin, with greatly rounded shoulders and of short stature; stated that he had been a tall man formerly, but had been creeping down for some time; worked regularly till 20th May, when, after great exertion, he lay down to sleep in a wood, and got drenched with rain. He took a severe cough, spat some blood, was confined to bed for six or seven weeks, and from that time to the date of admission, though able to go about, was unfit for work. On admission his voice was husky, and he brought up, with little effort, large quantities of yellow fetid sputum. There was no marked dulness on percussion over the lungs; coarse crepitation and snoring râles were heard towards the bases; at the apices, the breathing was harsh; there were no moist sounds; there was a distinct systolic blowing at the apex of the heart. He complained much of flatulence; but the appetite was good; bowels regular. On September 24th, the following report of the state of the chest was made:—Percussion note was wanting in resonance, but there was no special dulness. Auscultation on right side elicited coarse moist râles, particularly towards the base, and there were occasional blowing sounds with increase of vocal resonance; on the left side the respiration was harsh at the apex, rather feeble, and accompanied by moist râles at the base. In October the lower part of the left lung was markedly duller than the other parts, and distinct cavernous sounds were heard over the lower part of the right. The character of the sputum was unaltered; early in November the general health appeared improved. On the 11th he had rigors, and the character of the fetor became somewhat altered. The amount of the sputum was 7½ oz. in twenty-four hours; the patient became thereafter more feverish, and died exhausted on 29th November.

Autopsy.—The body was emaciated. The skin was dark. The spinal column was greatly arched, but not deformed Very dense adhesions, at some parts several lines thick, connected the lungs with the thoracic wall and the diaphragm. They were less firm on the left than the right side. On microscopic examination, the lower part of both lungs was much consolidated, but contained numerous anfractuous cavities, mostly lined with smooth mucous membranes, and continuous with bronchi above. Most of them contained a peculiar soft curdy-looking matter composed of granular and fatty material, with numerous fatty crystals. In other sacules there were fœtid pus, and numerous bronchial casts. The mucous membrane was in some parts thickened, red, and velvety; in others thin. The following conditions of the bronchial tubes were observed:—1. Some were simply dilated; the mucous membrane not thickened, but thin and delicate. The walls evidently thinned, many dilatations extending to their finest extremities. Around them no consolidation of lung substance was found. 2. Some were in a condition essentially similar, but the mucous membrane was velvety. 3. In some of the dilatations the mucous membrane was thick and inflamed, and here and there eroded. 4. Some cavities communicated with several bronchi, were livid, with reddened and inflamed mucous membrane. Around these cavities the lung substance was consolidated. On microscopic examination of the consolidated lung tissue, little trace of air-cells could be made out, and it was mostly composed of fibrous tissue. The heart was somewhat hypertrophied and dilated, and the abdominal viscera were natural.

Commentary.—In this case, I cannot doubt that the process of dilatation of the bronchi had preceded the acute and sudden attack, and that it attracted no attention until it was well advanced, and marked bronchitis was superadded. In this case, the peculiar state in which the patient lay for some time before death suggested the idea of septicæmia, and certainly no other explanation occurred to my mind.

CASE V.—*Bronchitis—Hæmoptysis, severe and repeated—Perforation of Pleura—Pneumo-thorax—Death—Autopsy, Bronchiectasis, Pneumo-thorax.*

Annie Wilkie, æt. 19, admitted to Ward XI., under the care of Dr Maclagan, 6th December 1866, a book-folder.

History.[1]—The patient enjoyed good health up to the year 1864, when she caught a heavy cold, which has never left her since. Two years since she first began to spit blood, and for this symptom she was admitted to this ward in November 1865. At that date, the hæmoptysis was severe, but not very frequent, and had recurred for three or four months at the menstrual period in quantities sufficient to excite her alarm. She was not regular, not having menstruated for three months before admission. After the most careful and oft-repeated physical examination of the chest, no trace of pulmonary phthisis or bronchitis being evident, the idea of vicarious menstruation suggested itself, but was soon dispelled by the occurrence of hæmorrhage by the mouth, at intervals of only a few days. Rallying under the rest and quiet of the hospital regime, she left the ward in the end of December 1865, but in the end of January 1866, was re-admitted with very severe hæmoptysis, and continued in the house for two months.

During this time, the only thoracic physical sign that drew attention was remarkable loudness of the cardiac sounds, without any hypertrophy of the organ, and occasionally accompanied by a bruit at the base. The walls of the chest seemed very thin, and showed the heart's impulse much more plainly than usual. There was no comparative dulness on percussion at either apex, nor were the respiratory sounds morbidly altered in character. During the summer months she was under medical care (my own), with occasional severe attacks of hæmoptysis, which came on after exertion. In October, being ordered cod-liver oil, she improved in general health, and the tendency to hæmorrhage

[1] For this report of the history, I am indebted to Dr Wolston, formerly Resident-Physician to the Clinical Wards.

seemed checked till the end of November, when a severe flow occurring she again entered the Infirmary.

On Admission.—Respiratory system.—The whole of the chest is flattened on both sides. On the right side the breathing is weak. Slight increase of vocal resonance and slight crepitation over the middle lobe. No expectoration and no cough, except when hæmoptysis occurs. She cannot lie on right side.

Circulatory system.—Is troubled by palpitation. Heart's impulse increased, otherwise normal. Pulse 80—irregular.

Nervous system.—Complains of pain in the epigastrium and precordial region, also in the lower dorsal region when she stoops.

Digestive system.—Suffers from dyspepsia. Bowels regular.

Genito-urinary system.—During the last six months has menstruated only at intervals of six weeks. Urine clear amber colour; sp. gr., 1029; slight cloud, acid; some urates present.

Dec. 8th.—Was given tannic acid gr. v. every two hours, which, making her sick, was changed on the 11th for gallic acid.

On the 13th, lost 8 oz. of blood in one attack this morning. Given acetate of lead in ten grain doses every six hours, which had also the effect of making her sick, and was discontinued on the 14th. On the morning of that day she had another severe attack of hæmoptysis. The blood expectorated was estimated at 27 oz.

18*th.*—The physical signs were little altered. The sputum was purulent and copious.

On the 21st, she complained of great pain in the precordial region. On the 24th, there was distinct pneumo-thorax of the left side; and on the 27th she sank.

Autospy (28*th Dec.* 1866).—The body was emaciated. The left side of the thorax contained some air and several pints of fetid pus. This was confined by adhesion in the lower and posterior part. The adhesions were not old and dense, but thin, and rather recent. In consequence of the effusion, the lung was pressed forward and upward. Towards the base of the right lung, there was some recent pleuritic effusion, but no fluid; the lung was expanded throughout, but not emphysematous. There was no recent tubercle in either lung; and there were only three or four minute nodules, which might have owed their origin to a tuberculous process. At the apex of the right there were some slight puckerings, and a single emphysematous bulla of the size of a cherry-stone. In the lower lobe of the left (the compressed) lung there were several gangrenous abscesses, each of about the size of an almond,—two of them, both situated towards the posterior part of the diaphragmatic surface, communicated with the pleural cavity. In the centre of these abscesses, dilated bronchi were distinguishable. The larger bronchi in both lungs were natural. Many of them about the third and fourth division, and particularly towards the middle and base posteriorly, were dilated. The dilatations were fusiform, and varied in size. None of them had their mucous membrane inflamed, or contained a fetid secretion; but in most of them there was a quantity of muco-purulent matter, which on microscopic examination was found to be composed of mucous corpuscles, columnar epithelium, natural and granular, along with a considerable amount of granular matter. The tissue around the dilated bronchi was atrophied, and the middle lobe of the right lung consisted of little more than a group of dilatations. The walls of the dilated portions were thin, the mucous membrane not thickened. The epithelium was carefully examined and compared with corresponding structures from healthy bronchi, and was found natural. There was no condensation of tissue around any of the dilated tubes. The heart and the other viscera, excepting the kidneys, were natural; they were waxy.

Commentary.—In this case, marked pulmonary symptoms had existed for between two and three years, and by much the most alarming feature was the hæmoptysis, which occurred several times, and often with much severity. Death was, however, ultimately due to another cause, viz., the formation of abscesses round some of the dilatations, the perforation of the pleura, and consequent pneumo-thorax. In this case there was no fetid sputum, and no consolidation of lung tissue.

CASE VI. — *Long-standing Cough — Typhus Fever — Death — Autopsy — Bronchiectasis, with no Consolidation of Lung.*

History.—The patient, A. W., æt. 16, had suffered from cough for some time before she became affected with typhus fever. Under the latter malady she sank rapidly.

Autopsy.—On post-mortem examination, distinct dilatation of several of the bronchial ramifications was observed. It was most marked about the middle of the lung and towards the lateral region.

CASE VII.—*No History of Chest Symptoms—Typhoid Fever—Death—Autopsy—Ordinary Typhoid Lesions, Bronchiectasis.*

A. L., a woman, aged 24, was admitted to the Royal Infirmary on May 7th, suffering from symptoms of typhoid fever. She had been ill for seven days. The nervous symptoms were strongly marked, and after passing into a state of coma vigil, she died on the 12th.

Autopsy.—The body was well nourished, and exhibited all the features of typhoid fever. But in addition there was observed a distinct dilatation of many of the bronchi, those about the middle part of the lung posteriorly being most markedly involved.

Commentary.—These two cases may be appropriately considered together. Both occurred in patients who died of fever. Both in young women, one of whom was known to have suffered for some months at least from cough and spit, but had exhibited no other pulmonary symptoms. There was no particular accumulation of secretion, no consolidation of lung tissue, no traces of tubercle—no morbid condition, in fact, excepting the bronchiectasis.

CASE VIII.—*Bronchitis—Bronchorrhœa—Fetor of Breath and Sputum—Recovery.*

Francis Hackel, æt. 19, a glass engraver, native of Bohemia, but resident in Edinburgh, was admitted to the Royal Infirmary, under my care, December 12, 1865.

The patient stated that at the age of eleven he suffered from inflammation of the lungs, and had ever afterwards been subject to cough. Eight days before admission he had severe rigors and pains in the left side, with increase of cough and expectoration. On admission, he was suffering from a slight attack of pleurisy, limited to the posterior part of the base of the right lung, but throughout both organs there were signs of bronchitis, and the expectoration was copious, purulent, and yellow. He had a most peculiar fetor,—a fetor so disgusting as to nauseate even those well accustomed to the worst odours of a pathological theatre. He was at the same time feverish, and his face wore an anxious expression. His pleurisy subsided under the use of poultices; the feverish symptoms were relieved by diuretics and diaphoretics; but the most singular therapeutic result observed was the complete disappearance of the fetor of breath and sputum, under the use of inhalations of creasote. From that time he gradually improved, and early in February was able to return to his native country in tolerable health.

Commentary.—This case affords, I think, an example of true fetid bronchitis, in which secretion of fetid mucus takes place from the bronchial mucous membrane. It would be unreasonable to assume the existence of gangrene, and there were no physical signs to warrant the opinion that dilatation of the bronchi existed. The rapidity of the beneficial effects following upon the use of the local medication seem all to point to a morbid secretion from the bronchial mucous membrane.

I cannot conclude this paper without expressing my thanks to my colleagues in the Infirmary for the kindness with which they have allowed me to make use of the reports of cases which had occurred under their care, and to my friend Mr Richard Caton for the accurate drawing which illustrates the pathological appearances.

PRINTED BY OLIVER AND BOYD, EDINBURGH.