On the pathological anatomy of bronchitis, and the diseases of the lung connected with bronchial obstruction / [Sir W.T. Gairdner].

ON THE

PATHOLOGICAL ANATOMY

OF

BRONCHITIS,

AND THE

DISEASES OF THE LUNG

CONNECTED WITH

BRONCHIAL OBSTRUCTION.

BY

W. T. GAIRDNER, M.D.,

FELLOW OF THE ROYAL COLLEGE OF PHYSICIANS, AND PATHOLOGIST TO THE ROYAL INFIRMARY OF EDINBURGH.

EDINBURGH:

SUTHERLAND AND KNOX, GEORGE STREET.

MDCCCL.

[FROM THE MONTHLY JOURNAL OF MEDICAL SCIENCE, 1850.]

MURRAY AND GIBB, PRINTERS, EDINBURGH.

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PATHOLOGICAL ANATOMY OF BRONCHITIS, &c.

NOTWITHSTANDING the great frequency of bronchitis in European latitudes, and the attention which has always been given to this disease by physicians, especially since stethoscopic examination came into use, it has appeared to me that the commonly received doctrines as to its ultimate effects upon the pulmonary texture are not expressive of the importance of the subject, and in many points not consistent with the truth of nature. Having become deeply impressed with the truth of the views to be advocated in the following papers, and having taken much pains to satisfy myself as to their correctness, I venture to lay them before the profession, in the hope that, if corroborated by the further researches of others, they may lead to improvements of some importance, both in pathology and practice.

As to the sources from which my materials are drawn, it is only necessary to state here, that while I have relied chiefly on personal observation, I have endeavoured to combine and harmonise the conclusions thus arrived at with those of prior observers, so far as known to me; nor have I willingly omitted any species of evidence which seemed to bear upon the subject. It would have been, indeed, an unsatisfactory task to have brought forward the results of personal experience on this subject, had they not been found to agree in many, even in most points, with those of some former authors in respect to matters of detail. In placing these pages in the hands of the public, therefore, I profess myself anxious for judgment as to the *truth*, more than as to the absolute *novelty*, of their conclusions; and shall be satisfied if enough of originality be found in them as a whole, to rescue them from the charge of being a superfluous or burdensome addition to our literature.

PART I.

PRIMARY RESULTS OF BRONCHITIS.

In the present part, the direct and primary results of bronchitis will be discussed; comprising under this head those effects which follow, almost constantly, the accumulation of mucus and inflammatory products in the bronchial tubes, when these are sufficient in quantity to cause serious obstruction. A second part will include the secondary and more permanent disorganizations of the pulmonary texture, which result from the former under peculiar circumstances; being induced either by long-continued intensity of the original disease, or by constitutional states unfavourable to the removal of its results.

Obstruction of the Bronchi.

The ordinary effects of bronchitis upon the mucus and other contents of the bronchial tubes, are, for the most part, well understood and clearly described by authors. The vascular engorgement of the mucous membrane, which at a later stage becomes much softened and thickened, and the loading of the tubes with an altered mucous secretion, intermixed to a greater or less degree with purulent matter, are familiar to every one. The connection of these sources of obstruction with the auscultatory and other signs is also so well known as to require but few observations.

The varieties in the character of the secretion from the diseased membrane are very great. In the earlier stages of the affection, the mucus is profuse, thin, watery, and frothy, being mixed with air bubbles of all sizes. At this period it contains but few microscopic elements, a few altered epithelium-cells being only visible, and these in much greater number in the mucus procured from the dead body than in that expectorated by the living patient. At a later period the mucus has become yellowish, more tenacious and viscid, containing numerous pus corpuscles, and more of the altered epithelial elements. Occasionally, but more rarely, the expectoration, or the fluid in the bronchi after death, is composed of pus nearly pure, but always viscid and thick, like that which flows from old abscesses. In these cases the pulmonary tissue is rarely unaffected.

When the disease has been of considerable duration, and has nevertheless been the result of an acute attack, there are usually found, on incising the bronchi, numerous yellowish pellets, or irregular flocculent masses of somewhat curdy consistence, which float in the more recent and thinner mucus, without in any degree becoming amalgamated with it. These masses are found of all sizes, and constitute a well-known form of the expectoration of bronchitis in its advanced stages. They are no doubt formed by the evaporation of the more fluid parts of the mucus, in consequence of the constant passage over it of dry air; the inspissated material clinging to the walls of the air passages, until detached and washed away by renewed exudation from the mucous membrane. Such a mass is not unfrequently seen plugging the whole caliber of one of the larger or smaller bronchi, in such a position as to leave no doubt that in the act of inspiration it must have acted the part of a ball-valve, completely preventing the access of air to the part of the lung involved, by falling back upon the orifices of the smaller bronchi, into which its size would prevent it from entering.

When bronchitis, or bronchial catarrh, has existed for some time in a comparatively slight form, and with the expectoration only of an increased quantity of mucus unmingled with pus, portions of this secretion become inspissated in the form of a thick, glairy, tenacious, semi-transparent material, sometimes resembling raw white of egg. In one or two cases I have pressed out of the smaller bronchi plugs of altered mucus of a still more tenacious character, like cold glue, and nearly, if not quite, transparent. But this amount of inspissation is not common.

The effects of these morbid accumulations in producing obstruction of the bronchial tubes are well known to the auscultator. So long as the mucus is thin and watery, or even more or less purulent, there is no serious or complete impediment to the passage of air; which, as the fine and coarse mucous rales, accompanied by vesicular respiration, indicate, finds its way through the fluid to the ultimate bronchi and pulmonary vesicles. The fluids, too, at this stage, move freely throughout the bronchial tree even to its minutest branches, and when in excessive quantity, are readily expectorated in the act of coughing. This stage of bronchitis, therefore, is comparatively little apt to be accompanied by urgent dyspnœa, or by changes in the condition of the pulmonary texture.

It is otherwise, however, when after a time the secretions within the bronchial tubes have become inspissated; the mucus having either become purulent and formed itself into the tenacious pellets above mentioned, or assumed the stringy, tenacious consistence proper to the more chronic forms of the disease. When, under these circumstances, expectoration is hindered, either by the tenacity of the mucus itself, the weakness of the patient, or any other cause, the tubes become really obstructed, the sound of vesicular respiration is at some points of the lung much diminished, or it may be altogether

lost, and the bronchial râles are correspondingly modified, indicating the passage of a smaller quantity of air with a much greater amount of resistance; the ordinary mucous rattles being supplanted in part by the sharp "clicking" and valve-like sounds so well described by Dr Williams.[1]

It is quite evident to the observer of a patient in this condition, or, indeed, in any stage of bronchitis attended with laborious breathing, that the chief difficulty is invariably in *inspiration*. This act is accomplished only with the aid of all the accessory muscles; and, even then, the contracted state of the chest, especially in children, and the imperfect descent of the diaphragm, show that in proportion to the power employed, the amount of air entering is small. The expiratory act, on the contrary, is always accomplished with comparative ease; and any serious obstruction to the performance of this act is at once got rid of, or dislodged, by the additional impulse given to the expired air in coughing or hawking. These phenomena of laborious breathing, particularly the long-drawn, exhausting, inadequate inspiration, are probably quite peculiar to obstructive bronchitis. They occur, it is true, in diseases of the heart; but in them, I believe, only secondarily, from the accumulation of mucus in the respiratory passages. The dyspnœa of *pure* pneumonia, on the other hand, is something quite different, being a mere *acceleration* of the respiration, without any of the heaving or straining inspiration observed in bronchitis, or in cases where the two diseases are combined. So much is this the case, that I have repeatedly observed patients affected with a great extent of pneumonia in both lungs, and in whom the extreme lividity, and the respirations numbering 50 or 60 in the minute, showed infallibly the amount to which the function of the lung was interfered with: and who nevertheless lay quietly in bed, breathing without any of the violent effort, or the disposition to assume the erect posture, so constantly accompanying the more dangerous forms of bronchitis. If this freedom from orthopnœa and laborious breathing be not uniformly characteristic of true pneumonia, it is because that disease comparatively seldom exists uncomplicated by some degree of bronchial affection.

The cause of the inadequate and laborious inspiration in bronchitis, while the expiration is comparatively easy, is to be found, I believe, not merely in the smaller power of the inspiratory muscles to dilate the chest, nor in the advantage which the expiratory forces derive in the dislodgement of obstructive mucus from the sudden impulse of coughing, but in the mechanical relations of the tenacious inspissated mucus to the caliber of the bronchial tubes. On this subject I have more to say in the next division of my subject.

One fact of considerable importance, in connection with the total obstruction of bronchi by muco-purulent matter, has been adverted to by Andral, and is illustrated by two cases in the "Clinique

[1] Diseases of the Chest, 4th edition, p. 80.

Médicale." This is, that the accidental position of such an obstructive plug may be the cause of a very rapid and unexpected fatal issue, in a case by no means threatening from the violence of inflammatory action. In each of the two instances given by Andral, the respiratory murmur became completely suppressed in the upper part of one lung, the patients having been previously affected with moderate bronchitis, and dyspnœa having become suddenly increased after a fit of coughing. The post-mortem examination showed the signs to be due to an obstructive accumulation in the bronchi leading to the upper lobe of the lung; but the absence of respiratory murmur, combined with clearness on percussion, had led during life to the diagnosis of emphysema. There can be, I think, little doubt that many of the paroxysmal accessions of dyspnœa in persons affected with bronchitis are due to accidental change of position of the pellets and ropy masses of inspissated mucus, which accumulate in the tubes. At least stethoscopic examination frequently reveals the signs of obstruction in particular parts of the lung, supervening rapidly, and disappearing again with equal suddenness, in consequence of the accession of cough.

I have now to advert to a condition of the pulmonary texture which appears to me, from the results of my own dissections, to spring more directly than any other from obstruction of the bronchi by mucus of a certain degree of tenacity. As this condition, the collapse of the air-cells, has been but little noticed by authors in this connection, at least in the adult lung, the subject will perhaps be best introduced by a narrative of my own observations with regard to it, which I shall endeavour afterwards to connect with the facts furnished by others.

Collapse of the Lung as connected with Bronchial Obstruction.

During the epidemic of continued fever which prevailed in Edinburgh in the greater part of the year 1847, it was frequently observed that the lungs, in persons of all ages, were the seat of a form of condensation, characterised by the absence of the friability, and granular appearance on section, of pneumonic consolidation, and also by the peculiarity of its microscopic elements, the large granular cells which form so common an ingredient in ordinary red hepatization being either very sparingly or not at all present. The condensed portions were usually scattered over both lungs, and often very limited in extent, being accurately circumscribed by the margins of the lobules; in most other respects they corresponded with the descriptions to be hereafter given. These appearances occurred very commonly in typhus fever, whether of the ordinary form or the abdominal typhus (now commonly called *typhoid fever*), accompanied by intestinal ulceration; which latter form had at that time a prevalence quite unusual in Edinburgh. Similar lesions were occasionally, though less frequently, seen in the relapsing fever, which was also

epidemic during that year. This state of the pulmonary tissue was the subject of frequent conversation, and repeated microscopic examination, among those chiefly engaged in the pathological theatre of the Infirmary during this period; although my own notes of fever cases at this time are but few, I am fortunately able to compare my recollections with several sufficiently characteristic descriptions of the lesion in question by Dr Waters, whose examinations were recorded with the utmost care and fidelity, with a view to the preparation of a history of the epidemic, unfortunately not yet published.[1]

The nature of this pulmonary affection appeared to me, at the time, very doubtful. By some it was looked upon as corresponding with the "typhoid deposits" in the lung described by German authorities; and the imperfect cell-forms often seen under the microscope seemed to favour this view. On the other hand, careful examination showed that there was frequently little or no real *deposit;* the cell-forms differing very little from those which might be procured from the normal lung, or still better, from one compressed by pleuritic effusion, or any other cause. At this time, my experience in pathological studies was not such as to enable me to form a decided opinion; but towards the close of the epidemic, the result of observations inclined me to believe that the lesion so frequently witnessed in fever was not connected with any specific form of morbid deposit, but was, in its purest form, a condition of imperfect expansion or collapse of the pulmonary tissue, similar to that described in the lungs of infants under the name of *atelectasis*, and differing only in its distribution in patches, and in other accessory circumstances, from the condition of *carnification* described by Laennec as the result of compression of the organ.

That the fever of 1847 was not, in any degree, *specially* characterised by these lesions is fully proved by the researches of other authors, as well as by my own subsequent experience. In fact, in the course of the two succeeding years, I had various opportunities

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[1] From one of Dr Waters' reports I quote the following :—"Inferior lobe of left lung felt condensed, and contained less air than ordinary; its section was smooth, not granular, and its consistence somewhat tough, not breaking down under the finger." On another occasion, "The lower lobe of the left lung was much gorged with blood, and of a dark colour, and smooth section, not granular, void of air, and sinking entirely in water." The reader may compare these with the characters which follow (p. 12) of the bronchitic collapse. The following description from the case of a child dying of fever, corresponds to the characters of lobular collapse, combined with the bronchial abscesses to be hereafter described. There were condensed nodules in the lung which "presented at the surface of the organ, where they were recognised by their bluish-black colour and resistent feel; they were not elevated above the surrounding surface, but rather very slightly depressed." * * "On section, the great majority of these masses presented a reddish-blue colour and smooth surface. They were not friable but of firm consistence. A few of them had degenerated into a reddish-gray somewhat friable substance, and two or three were excavated by central cavities, which were empty and collapsed."

of becoming familiar with collapse, or carnification, of the pulmonary tissue, as a disease distinct from pneumonic consolidation, occurring in various forms, and under circumstances where no external compression of the lung could be conceived to account for it. Many of these lesions occurred in the lungs of children, but those of adults and old persons were scarcely less frequently affected. The disease presented itself, also, frequently in combination with other affections, which gave rise to no small difficulty in forming an opinion on the true nature and mode of origin. Neither was it always very easy to distinguish it from pneumonia, more especially in some of the combined conditions to which I shall hereafter allude; and I am satisfied that the terms *lobular pneumonia*, *red hepatization*, or the more indefinite expression, *condensation*, have often sufficed in my own case, as well as that of others, to cover ignorance or imperfect knowledge of the condition in question.[1]

Notwithstanding these difficulties, however, observations made in the years 1848-9 left me no longer in doubt, that the lungs of adults not unfrequently display portions more or less limited or diffused, which are so perfectly condensed as to sink rapidly in water, and yet differ widely, in appearance and microscopic character, from truly inflamed lung, as well as from all the atrophic conditions of the organ which can be clearly traced to inflammation or structural disease as their cause. In some instances, such portions presented exactly the appearance of fœtal lung; and only the manifest absurdity of the proposition could have prevented the observer from ascribing their state to the same cause as the congenital non-expansion of the lung, or atelectasis of Jörg. In the absence, therefore, of any manifest explanation of the compression or collapse of the tissue observed in these cases, I was obliged, provisionally, to rest satisfied with the knowledge of the fact that most of the lobular and many of the more diffused forms of condensation usually ascribed to pneumonia, in the adult as well as the child, were really the result of some other and unknown condition.

Some time after this collapsed condition of the lung had become tolerably familiar to me, the following case occurred, presenting a marked example of the coincidence of the affection with obstructed

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[1] Had opportunities of examining the lungs of very young children frequently occurred to me, I could scarcely have failed to have soon become familiar with all the phases of this lesion; and to have recognised much sooner than I did, the identity of the congenital atelectasis, the "lobular pneumonia," and the carnification or collapse of the adult lung. But owing to the exclusion of all children under five years of age from the Infirmary, my observations were confined almost entirely to the lungs of persons above the age of infancy; and it was only at a comparatively recent period that the observation of a few infantile lungs, and the perusal of the work of M. Legendre (Recherches Anatomo-pathologiques et Cliniques sur quelques Maladies de l'Enfance, Paris, 1846), revealed to me clearly the immense importance and frequency, though not, I believe, the true significance, of this state of the pulmonary texture in the early periods of life.

bronchi, *both lesions being limited to a very small and circumscribed space in the lung.*

Case I.—*Lobular collapse of Lung—Obstructed bronchi.*—(P. R. 257.[1])

A man, æt. 18, affected with necrosis of the femur, anasarca, and some degree of abdominal dropsy from disease of the liver, spleen, and kidneys, died in the surgical hospital in the last stage of exhaustion and emaciation. No pulmonary symptoms had attracted notice.

The lungs were generally normal in appearance. At one or two places, however, they crepitated imperfectly over spaces not larger than an inch in diameter; these portions were quite circumscribed by abrupt margins; the bronchi leading to them yielded on pressure a very tough gelatinous mucus (like thick calves'-foot jelly), which contained only ciliated epithelium, and had otherwise the ordinary appearance of mucus under the microscope.

In this case there was little or no room for fallacy in judging of the connection of the pulmonary collapse with bronchial obstruction. The parts affected, with the bronchi leading to them, were quite capable of being isolated; and there was no trace of an inflammatory affection in any part of the pulmonary tissue. The co-existence of the collapse with bronchial obstruction thus accurately limited, could only, in all probability, be explained by the dependence of the one upon the other as its cause. The following case, however, which occurred during the present year, is still more conclusive:—

Case II.—*Extensive Collapse of Lung—Obstruction of Bronchi by a Tubular Membrane.*—(P. R. 301.)

A girl, aged about 21, was attacked, after a surgical operation upon the tongue, with urgent dyspnœa having the character of a laryngeal affection. Tracheotomy was performed, but failed to save her. She died about twenty-four hours afterwards.

Dissection performed Feb. 22, 1850. The appearances in the air passages and lungs were the following:—

The right lip of the glottis was infiltrated with serum, which distended the epiglottidean fold of mucous membrane so much as nearly to close the opening. The larynx and trachea were occupied by a tubular false membrane throughout their whole length; it was about a line in thickness, friable, and of a yellowish white colour. This membrane was continued at the bifurcation into the right bronchus, but the left was free from it. It could be traced throughout the bronchi of the right lung even into the minuter ramifications, in which it assumed the form of a very soft opaque matter, like a thick emulsion. On examination with the microscope, the membrane presented the usual appearance of coagulated fibrin, with some pus or mucus corpuscles, but these not in very large numbers.

Both lungs were more collapsed, and contained less air than natural. The left, however, was much less affected than the right. The latter was at some parts completely flaccid and free from air, while others presented an imperfect crepitation. The upper lobe crepitated more than the others, and its colour was

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[1] These figures affixed to a case, refer to the numbering in the Pathological Register of the Royal Infirmary, vol. xii., where the details which I have omitted, as not bearing on the argument, will be found, usually in a pretty extended form.

mostly natural; but in its lower portions it approached more in colour to the middle lobe. This was perfectly non-crepitant, of a grey colour, having a smooth flesh-like section, and infiltrated with a thin serosity, having a faint reddish tinge, nearly transparent, and presenting under the microscope only epithelium, pigment cells, and a very small amount of pus cells, and granular matter. The lower lobe was not quite so much compressed, and contained a good deal of blood. The bronchial glands were slightly enlarged, and the whole pulmonary mucous membrane highly vascular.

In this case, as in the former, there were no pneumonic appearances; for the small amount of pus cells which existed in the serosity scraped from the lung, were probably derived from the small bronchi, and were, at any rate, quite inadequate to account for so remarkable a consolidation. Neither can it be supposed that there was any older structural lesion of the pulmonary substance, as the girl was undoubtedly in good general health at the period of her admission into the hospital, and the whole stages of the fatal disease were too rapid to admit of any complicated structural change. The collapsed lung presented exactly the appearances so commonly seen in organs compressed by pleuritic effusion, except that the middle lobe had rather more of an œdematous appearance than is common under such circumstances. Here, then, is an instance of collapse of the entire lower lobes, and part of the upper in the right lung, as complete as it could have been from external pressure, and coinciding with a manifest cause of obstruction in the whole of the bronchi of that lung; whereas on the opposite side, where the obstruction had been much less considerable, the lung was comparatively expanded and normal. It is impossible not to see here a relation of cause and effect; and as it will not be maintained that the deposit of false membrane was the result of the collapse, the converse proposition is, I think, scarcely to be avoided.

The form of bronchial obstruction in this case is a sufficiently rare one in the adult, but by no means unexampled. It is not at all clear whether the membrane had never extended to the bronchi of the opposite lung, or had been dislodged by expectoration. In the former case it is not easy to understand the non-symmetrical character of the fibrinous exudation, while the mucous membrane appeared equally inflamed on both sides of the chest; in the latter it is still more incomprehensible, how the complete expectoration of an extensively ramified membrane could take place, without being observed during life. But of the fact of its presence on the right, and its entire absence on the left side, there is no doubt.

The observation of these two cases, and the conclusions which appeared naturally to follow from them, as to the connection between collapse of the air cells and bronchial obstruction, tended to throw light on many of the obscurities of the preceding observations. More particularly, it seemed to me that they furnished the connecting link necessary for the explanation of the peculiar forms of "lobular pneumonia" (as they were generally considered), which had frequently occurred both in the lungs of children and adults. The

peculiar connection of lobular condensation with bronchitis was a matter of common observation with me before this period; the more recent literature on the diseases of the lung in children also seemed to point to the same fact; and it seemed every way probable that many of the more complex phenomena of pulmonary disease, would receive explanation from the more careful consideration of their connection with obstructed bronchial tubes. I therefore renewed my observations with great interest, directing them particularly, and with more care than formerly, to the relative condition of the bronchial tubes and pulmonary tissue, in all cases where either pulmonary collapse or bronchitis existed to any considerable extent. Some of the illustrative cases which have occurred, will hereafter be detailed. The general result to which I have been led is, 1st, that in all cases of collapse of the lung not caused by external pressure, the bronchi have presented unequivocal appearances of obstruction; 2d, that in most, if not all, the instances of severe and fatal bronchitis, especially if the secretions had become ropy or inspissated, more or less collapse of the pulmonary texture has also been present; that under peculiar circumstances, which will be presently adverted to, a much less amount of obstruction may be attended with collapse of the pulmonary texture, the symptoms in such cases probably attracting little attention.

In order to justify these conclusions, it will be necessary to describe a little more fully the forms under which I have observed the lesions referred to.

Bronchitic collapse of the lung occurs under two distinct aspects: the *diffused* form, and the limited or *lobular* form. Of these, the latter variety is the more striking and characteristic, and has been, especially in the lungs of children, the subject of more discussion than the former; but the diffused form is by far the more common, and is in fact of very frequent occurrence, at least in its slighter degrees. Both forms present the same fundamental changes of the pulmonary tissue, which is usually of a dark violet colour externally, as seen beneath the pleura; and internally of a more or less deep brownish red, or mahogany tint. The colour, however, is by no means an invariable criterion, depending almost entirely on the amount of blood in the collapsed tissue. The affected parts are always more or less condensed; this condensation may amount to a mere diminution of the crepitation, or to a total absence of it, in which case portions are usually found to sink readily in water. These latter portions are both more flaccid and much less friable than the pulmonary tissue when in a state of red hepatization; and they differ greatly from this lesion in the aspect of their section and the nature of the fluid it yields to the knife. In every variety of true pneumonic consolidation, in which the lung is completely void of air, the air cells are occupied by a deposit, presenting to the naked eye (and still more distinctly to a power of 20 to 30 diameters) the well-known granular aspect of the hepatized lung. If the deposit is

fluid, or semi-fluid, it is capable of being pressed out, or scraped off, in the form of a thick opaque emulsion-like matter, of yellowish, orange, or grey colour; and in all cases it shows, under the microscope, abundant granular elements and cell-structures, of the kinds usually found in inflammatory exudations in parenchymatous organs. In the collapsed lung, on the contrary, the section is comparatively smooth, having somewhat the appearance, as described by Laennec, of muscular flesh; it presents no trace of granulations, and yields, on pressure, or to the knife, only a semi-transparent bloody serosity, which, under the microscope, is seen to contain little or nothing besides blood-corpuscles, epithelium, and other portions of normal tissue, and possibly a small amount of pus from the interior of the bronchi.

In the *diffused* variety, the collapsed condition may be found affecting a more or less considerable portion of either or both lungs, usually at their posterior part, and passing quite gradually into normal tissue; the supple, dense, tough feeling being exchanged for the normal spongy, elastic crepitation; and the violet, or deep purple colour, shading off into the usual hue of the surface. Even in this form of the lesion, however, a tendency of it at some points to be circumscribed by the interlobular divisions may often be observed; this tendency being, so far as I have observed, quite characteristic of the bronchitic, as opposed to the pneumonic, consolidations.

In the *lobular* forms of bronchitic collapse, which often occupy the anterior edges, as well as all other parts of the lung, the affected portions are everywhere accurately and abruptly marked off by the interlobular septa, the portions so limited being various in size and form, but always manifestly shrivelled, and sunk in below the level of the surrounding parts. This is peculiarly manifest when they occupy the anterior edges. When they are scattered through the lung, they communicate to the fingers, in feeling the organ externally, much the same sensation as clustered tubercles in the midst of crepitant tissue. This is the form which was so often described as "lobular pneumonia" in young children, till the experiments of MM. Legendre and Bailly clearly showed it to be nothing but a collapse of the air cells.[1] These observers were in fact the first to apply to this condition of the lung the same test as Jörg had long before used in respect to the congenital atelectasis, and to show that the lung affected with "lobular pneumonia" could be generally restored nearly to its natural condition by forcible inflation, which occasionally requires, however, to be continued for some time. I have employed inflation in both the diffused and lobular form of collapse, as observed in adults, and as above described, with precisely the same results as those of Bailly and Legendre. I may state, however, that though this test is very useful in demonstrating the nature of the lesion, in a favourable case, to one not familiar

[1] "Archives Generales de Médécine," 1844; and Legendre's work before cited.

with its character, I do not believe it to be applicable to the determination of the presence or absence of pneumonia in those mixed cases in which alone there is any difficulty. For I have observed that on the one hand, the partially pneumonic lung may be inflated when the affection is recent, and combined, as it frequently is, with bronchitic collapse; and, on the other, that in the latter lesion in its purest forms, complete inflation is often very difficult or impossible after the collapsed state has been of some duration. In fact, the lung then begins to undergo a modification in its nutrition and structure, which ultimately leads to permanent atrophy.

With regard to the combined forms of lesion above alluded to, they are, I believe, by no means of rare occurrence. The collapsed lung, especially in the incomplete and diffused form in which it most frequently presents itself in adults, may become the seat of a true inflammatory exudation into the air-cells, giving to the section an obscurely granular aspect. The exudation has in such cases the usual microscopic characters, but the shrivelled state of the lung, and the minuteness of the granulations, together with the state of the bronchial tubes, demonstrate the participation of these in the inflamed condition. Occasionally, also, the collapsed lobules are subject to a œdematous infiltration of fluid, when this state prevails in the other parts.

I think, however, that I have also observed the collapsed parts, particularly in the well-marked lobular forms of this lesion, to escape to a certain extent, the inflammatory or œdematous condition prevailing in the parts around them. At least, I have observed cases where these remained comparatively dry, containing only blood, when other parts were bathed with frothy serum or pus. This subject, however, requires further investigation.

In maintaining (as I have no hesitation in doing) that some degree of collapse of the lung is an almost invariable concomitant of bronchitis of a certain degree of intensity, it must not by any means be supposed that *complete* loss of crepitation is to be looked for in any part of the tissue in the majority of cases. The usual fact is, that the collapse is in the incomplete and diffused form; but I believe, nevertheless, that dulness of percussion during life from this cause, and complete lobular or diffused collapse after death, especially in the posterior parts of the organ, will be found to be much more common in the bronchitis, as well of adults as of children, than is commonly supposed. Of the truth of these opinions, both recorded observations, and unrecorded recollections, appear to me to furnish no inconsiderable amount of evidence.

The following two cases are good examples of collapse from bronchial obstruction,—the former in its diffused, the latter in its limited or lobular, form. The accounts during life are, in both cases, very inadequate; but they have, nevertheless, some points of great interest, apart from the lesion now immediately under consideration, which will render reference to them necessary in the sequel.

CASE III.—*Extensive Collapse of Posterior Portions of Lungs—Emphysema of Anterior Parts—Obstruction of Bronchi by Ropy Mucus—Death by Exhaustion.*—(P. R. 362.)

A woman, æt. about 30, died of exhaustion from the combined effects of Bright's disease and ulcerating cancer of the uterus.

The body, examined July 1st, 1850, was extremely pale, and the limbs, with the depending parts of the thoracic and abdominal parietes, were anasarcous. The abdomen was not remarkably distended, but a little fluid was found in the peritoneal cavity, and the surface of the membrane was studded over with small miliary granules. All the pelvic organs were more or less involved in cancerous disease, and small nodules, slightly larger than those on the serous membrane, were infiltrated into the muscles and fat of the abdomen. The kidneys were very pale, and presented an early stage of the waxy (non-granular) degeneration.

On opening the thorax, which appeared contracted and flattened laterally, the anterior edges of the lungs covered the heart more than usual. Both lungs were evidently emphysematous anteriorly. In the left lung large bullæ existed at the lower part of the upper lobe in its anterior prolongation; the dilated air cells forming a portion connected with the rest of the lung by a narrow process, composed of pulmonary tissue in part emphysematous, and in part completely collapsed and flaccid. The anterior edge of the lower lobe was also emphysematous. The pulmonary tissue at the posterior part of both lobes was perfectly collapsed and flaccid, tough, and of a violet colour; not containing enough of air to give the feeling of crepitation. The crepitant tissue passed very gradually into the collapsed. In the right lung there was diffused emphysema of the anterior edges, with here and there collapsed portions. Fully one-third of the posterior portion of this lung was completely collapsed; the rest more or less crepitant,—the non-crepitant tissue at some points shading off gradually into the crepitant; at others it being pretty sharply divided from it by the interlobular septa. On inflating the lungs artificially, the collapsed portions, for the most part, admitted air pretty freely when considerable force was employed; some of them, however, even after several repetitions of this process, retained their collapsed appearance. On making incisions into the lungs, the dense portions were seen to be smooth on section, and of a deep mahogany colour, and contrasting remarkably with the brighter colour of the crepitating parts. The collapsed tissue yielded nearly pure blood to the point of the knife; the crepitating portions a frothy sanguinolent serum. Some of the denser portions sunk in water; other parts were indifferent; and many floated freely. A large quantity of tough, semi-transparent, stringy mucus existed in the bronchi of both lungs. The mucous membrane was of a purple colour. Heart healthy.

CASE IV.—*Lobular Collapse of Lung from Bronchial Obstruction—Interlobular Emphysema—Death from Tubercular Hydrocephalus.*—(P. R. 360.)

A child, aged about 5 years, much emaciated, died with symptoms of hydrocephalus. The state of the lungs not noted during life.

The body was examined June 30th, 1850. Inflammatory lymph was deposited beneath the arachnoid at the base of the brain, and very minute tubercles existed over some of the convolutions. The ventricles were distended with serum. The root of both lungs was occupied by enlarged bronchial glands, which, on being cut into, were seen to be infiltrated with crude yellow tubercle. The right lung was healthy, with the exception of partial and imperfect collapse of some parts of its tissue, and considerable redness of the mucous membrane of the bronchi. The enlarged bronchial glands in the left lung pressed upon some of the bronchi going to the lower lobe, so as distinctly to diminish their caliber, as ascertained by a probe. The upper and lower lobes were glued together by adhesions, and in the upper margin of the lower lobe was a rounded

portion of condensed lung of the size of a walnut, which, on being cut into, showed a thick cluster of yellow tubercles, mostly confluent, and at some parts breaking down into pus. The anterior extremity of the lower lobe was completely collapsed and violet coloured, evidently sunk below the rest of the lung. The anterior end of the upper lobe showed a very few slightly enlarged air-cells at the extreme margin; the interlobular septa were emphysematous over a considerable extent of this margin, even where the air-cells were normal. On inflating the left lung, it was found that the collapsed tissue could be imperfectly inflated by using considerable pressure, but subsided again when the pressure was removed.

In the last case, besides some general bronchitis, there was a special obstructing cause in the bronchi going to the collapsed lobules; their caliber being diminished by the encroachments of enlarged bronchial glands, and of the isolated tubercular mass in the lung itself, which was situated immediately in contact with the collapsed part, between it and the root of the lung. In both these cases, the inflation of the diseased parts was effected with difficulty, and in some parts imperfectly; showing that their nutrition was already becoming modified, and the state of permanent atrophy was supervening on that of temporary collapse.

I have now to advert to the observations of the authors who have described collapse of the lung as a state distinct from pneumonic consolidation, or pleuritic carnification, with the view of showing in how far their researches have tended to throw light on the question of its origin. Laennec described the diffused form of collapse only in connection with pleuritic effusion and compression; and there can be little doubt, from some expressions in his descriptions of the first and second stages of pneumonia, that he must, to a considerable extent, have confounded the two conditions, especially in their more mingled and less characteristic forms. That he has not altogether overlooked the peculiarities of the lobular collapse, however, is evident from a passage in which he speaks of meeting with *carnified* portions of the size of a filbert or an almond in the midst of very crepitant pulmonary tissue. The occurrence of these he ascribes to "a slight inflammation in the first stage, the resolution of which, hastened perhaps by compression of the lung, has taken place in an irregular and imperfect manner."[1] This expression, while it shows that this great pathological anatomist had been puzzled to account for the lesion in question, will not be accepted as anything more than a hypothetical explanation of it. Very many writers, following Laennec for the most part in their pathology, have thrown this passage out of view altogether.

In 1829, M. Louis described the condition of the lung in a variety of cases of typhoid fever. These descriptions were repeated in his work on fever in 1841, in which the state of the lung in fevers was compared with that found in other diseases. The result of these inquiries was a most accurate description of the collapse of the lung

[1] De l'Auscultation Mediate, vol. i., ch. v., art. 1, sect. 366.

as a state altogether different from pneumonia, and which was found not only in typhoid fever, but in a variety of other diseases; chiefly at the posterior part of the lung, but sometimes *disseminated* (lobular.) M. Louis offers no speculation or remark as to the origin of this affection.[1]

In 1830, Dr Alderson, in a paper on the "Pathology of Hooping-Cough,"[2] pointed out most clearly the distinction between the lobular condensation observed in that disease, and true hepatization of the lung. "In hooping-cough the lung is always dense and contracted, as if the air had been expelled, and from the throwing out of adhesive matter the sides of the air cells had been agglutinated together; while in hepatization the lung is less dense than in hooping-cough, and is rendered more voluminous than in its natural state."[3] The inflation of the lungs would probably, in some cases at least, have clearly disproved this supposed agglutination of the air-cells. The state of the bronchi is carefully described by Dr Alderson. In one case, for instance, "most of the tubes were filled with a light yellow secretion, which, in the greater number, had assumed a concrete form, having very much the character of fibrine; in others, it was in the form of a thick puriform mucus; where it occurred in the concrete form, it adhered, though slightly, to the lining membrane of the tubes."[4] It will not be questioned that these descriptions concur entirely with the views already laid before the reader in this paper, as to the origin of the lesions in question.

The researches of Jörg, in 1832,[5] gave a new impulse to infantile pathology, by showing the frequent occurrence in new-born children of a state of deficient expansion of the pulmonary lobules, having all the appearance of being congenital, but often persisting for some time after birth. This state, which Jörg called *atelectasis*, was identical in its characters with the collapse described above, and, like it, was often lobular in its distribution. It was figured as a disease of the new-born child by Cruveilhier,[6] who, however, understood nothing as to its real nature. Jörg was the first who pointed out the effects of inflation in this form of pulmonary affection as contra-distinguished from pneumonia. He ascribed the collapse to various causes, but particularly to weakness on the part of the child, and to

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[1] Recherches sur la Gastro-enterite, I. 361; and Recherches, &c., sur la Fievre Typhoide, I. pp. 328-334. It is worthy of remark that, from the observations in the preceding pages on the Edinburgh epidemics of 1847, as well as from Dr Jenner's recent careful descriptions of cases observed in London (Monthly Journal, Feb. 1850, p. 115, et seq.), this form of pulmonary lesion does not appear to be peculiar to any type or form of continued fever, at least in this country.

[2] Medico-Chirurgical Transactions, vol. xvi. p. 78.

[3] Loc. Cit., p. 91.

[4] Loc. Cit. p. 85.

[5] De Pulm. Vitio Organico; Leipz. 1832; and Die Fötus-Lunge im Gebornen Kinde; Grimma 1835.

[6] Anat. Pathologique, livraison 15, plate 2, fig. 1.

the circumstances of a too precipitate birth, which he conceived, somewhat fancifully, to militate strongly against the establishment of respiration. In this last view he has had few supporters.

In 1838, MM. Barthez and Rilliet published a monograph on infantile pneumonia, which afterwards became the basis of the extended treatises in their great work on the diseases of children.[1] While these authors describe with great care and general accuracy the appearances of the carnified lung, whether lobular or diffused, they fall into the error, at that time universal in France, of considering it as a form of pneumonia; and their reference to the researches of Jörg is so slight as to render it probable that they were not aware of the important facts established by him. An interesting remark in relation to the present subject, is, however, made by MM. Barthez and Rilliet, viz.:—that bronchitis, especially of the smaller bronchi, is a frequent concomitant of the pneumonia of children, *especially the lobular form* (collapse); the connection of bronchitis with the lobar pneumonia being more rare.[2] Similar observations had been made by M. Fauvel and other authors.

Rokitansky, writing in 1842, does not allude to any of the researches hitherto mentioned; nor does he appear to be sufficiently aware of the distinctive marks and real nature of collapse of the lung. In his description of *lobular*, *typhoid*, and *catarrhal pneumonia*,[3] however, the reader will trace many of the characters of this condition. In regard to the last of these affections, he says, "It is always lobular, concurring with catarrhal affection of the bronchial ramifications leading to the diseased lobules, and occurring frequently in catarrhal attacks in children, especially hooping-cough and catarrhus suffocativus." * * "The lobuli affected are blueish-red, dense, and rather tough; the walls of the air-vesicles are swollen, so as to obliterate their cavities, and contain, when less swollen, a sero-mucous, slightly frothy secretion; there is no trace of granulations." Rokitansky also notices the sinking in of the affected lobules, which he ascribes to an emphysematous condition of the surrounding parts.[4]

In 1844 were published the important researches of MM. Bailly and Legendre, before alluded to, which demonstrated the identity of the "lobular pneumonia" of children with the congenital collapse, or *atelectasis*, of Jörg. These authors also describe the catarrhal affections of infants as often attended with this change. But it is singular, that notwithstanding their own application of inflation of the lung to show the nature of this lesion, they consider it as produced, in some instances, by distension of the blood-vessels causing closure of the air cells. It is clear that, if this were the case, inflation could not effect any considerable change.

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[1] Traité Clinique et Pratique des Maladies des Enfans. 1843.
[2] Op. Cit., vol. i. p. 75. [3] Path. Anat. vol. iii.
[4] Path. Anat., vol. iii., p. 106.

The greater number of these authors, in so far as concerns the diseases of children, are passed in review by Dr West in his late carefully elaborated, and, at the same time, original lectures.[1] The descriptions of Dr West well deserve to be read, on account of their clearness and accuracy of detail; and his views as to the frequent connection of the collapse of the lung with bronchitis, and its dependence, in many cases, on that affection, harmonize so closely with my own, that I can scarcely regret the late period at which I became acquainted with them,[2] as it enables me to point out more clearly the identity of the conditions in the adult with those in the child. "In the child," says Dr West, "nothing more is needed than a copious secretion of mucus into the bronchi, or a feeble condition of the vital powers, to prevent the air from freely entering the pulmonary vesicles, and thus to induce the collapse of a large portion of the lung."[3] I have already endeavoured, in part, to show that in the adult the same causes are capable of producing the same effects; and I shall hereafter explain more at length what I conceive to be the exact mechanism of this change, both in the one and in the other. Dr West adduces three cases, examined by Dr Baly, of lobular collapse of the lung in the adult, in persons who died in a state of great exhaustion from fever and dysentery. In two of these cases there was much dyspnœa and distinct signs of bronchitis. The third is said to have presented no complication, and is considered by Dr West to be the "result of simple debility." I shall refer to this point in the sequel.

On reviewing the whole of the facts here presented to the reader, I think that the frequency of collapse of the pulmonary tissue, both in the adult and the child, must be considered as established, and its connection with bronchial obstruction rendered at least extremely probable. I have adduced evidence that the condensation of the pulmonary tissue thus produced, which in its slighter degrees is often overlooked by anatomists, has in its more marked forms been described by many careful and exact observers under different names, and with various ideas of its pathological significance,—and that, especially in the case of children, it has been accurately distinguished from ordinary pneumonic condensation. I have described the forms in which this lesion has occurred under my own observation, and showed that, in all essential characters, it is the same in children and adults; that in both a certain degree of pulmonary collapse may be almost invariably found as a concomitant of fatal bronchitis; and that, in some cases, this state of the lung bears so obvious and undeniable a

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[1] Lectures on the Diseases of Infancy and Childhood: Lond. 1848.

[2] Dr West's researches have come to my knowledge only since this paper was read, in its original form, to the Medico-Chirurgical Society.

[3] Op. Cit. p. 183.

relation to obstruction of the tubes, as to lead to the almost unavoidable inference of the dependence of the former upon the latter.

That this conclusion from anatomical data is, in all respects, consistent with clinical experience, and with correct views of the mechanism of respiration, I shall presently endeavour to prove; and, in the second part of this memoir, I propose to show the probable dependence of many important chronic alterations of the pulmonary texture on the condition of collapse from bronchial obstruction. The pathological history of this lesion, however, in relation to acute bronchitis, would not be complete without some notice of a condition which occurs very frequently in connection with it, and leads to some of its most important secondary consequences.

Bronchial Abscess.

It not unfrequently happens that, in the centre of the collapsed lobules of a lung affected with acute bronchitis, there are found small collections of pus, varying in size from that of a hemp-seed upwards to double or treble that volume. These small abscesses present, on section, an appearance so like that of softening tubercles, as to be very readily mistaken by many persons for these bodies; and the resemblance is all the greater on account of the peculiar limited form of the condensation by which they are generally surrounded, which, when felt by the touch from the exterior of the lung, is exceedingly deceptive. In their interior, however, these little abscesses contain, in the recent state, a very fluid pus; moreover, they are often met with as acute lesions produced by a few days of illness, and without a trace of tubercle in any other organ. This is peculiarly characteristic in the young child, in which tubercle of the lung so very rarely occurs without extensive deposits in the bronchial glands, whereas the present lesion is accompanied in its pure form by nothing more than slight enlargement. When the pus contained in these abscesses, in their recent form, is pressed or scraped out, they are seen to be lined with a fine villous false membrane, very different from the thick curdy mass which generally surrounds softened tubercles; in others they are not abruptly limited at all, the pus appearing to lie in contact with the surrounding pulmonary tissue. When the bronchi leading to the lung so affected are carefully incised, they are found much inflamed; the mucous membrane vascular, thickened, and covered with pus; and some of the bronchi thus affected can be observed to communicate with these purulent collections, the mucous membrane having evidently been, at the point of communication, destroyed by ulceration, and either stopping short abruptly, or becoming gradually incorporated with the false membrane lining the abscess. Sometimes these abscesses are found to break into one another, and form more considerable excavations; in one instance (Case VII.) I found them connected with a gangrenous condition; more commonly, however, they remain

of limited size, preserving perfectly the direction and relations of the bronchial tubes.

These abscesses occur in the diffused as well as in the lobular form of condensation from collapse; and both forms may sometimes be seen in the same lung. A similar lesion may accompany true pneumonia, but always in those cases where it is combined with intense bronchial inflammation. In this case, also, it tends to the formation of more considerable and more irregular excavations.

Such cavities have been pretty accurately described by Barthez and Rilliet (as well as by other French writers on the bronchitis and pneumonia of children), under the name of "*vacuoles*." They are, however, far from uncommon in adults, though, perhaps, more frequent in the so-called "lobular pneumonia" of children. As these "vacuoles" unquestionably arise from the accumulation of pus primarily in the extreme bronchial tubes of the collapsed lobules, the name of bronchial abscesses may serve to distinguish them from other forms of excavation of the lung.

That Laennec should have overlooked these small excavations is singular enough. Probably he may have considered them a form of tubercle, a term which has been used by him and others with sufficient vagueness to cover a multitude of anomalous lesions. His assertion that in many hundreds of pneumonic lungs, he had only met with collections of pus five or six times, can only be explained upon this principle. It is, indeed, quite true that in simple pneumonia there is little tendency to the formation of abscess, the pus escaping rapidly by the pervious bronchi; but as we have seen that Laennec has probably confounded the pneumonic with the bronchitic condensation, I think it also probable that he has not distinguished the bronchial from the tubercular abscess, especially as these lesions often resemble each other so much as to have led me habitually, for some time, to call the former *tuberculoid disease* of the lung, which name is inscribed over several dissections in the pathological register from which the following cases are taken.

In three of the four cases of fatal hooping-cough recorded by Dr Alderson, in the paper formerly alluded to, these lesions are described shortly as dilatations of the smaller bronchi by thick mucus or muco-purulent secretion. Most of the French writers on the pneumonia and bronchitis of children since Barthez and Rilliet, refer to them more or less distinctly; and in Dr West's lectures they are described as a *true* lobular pneumonia—the result, however, of bronchitis. As the work of Dr West is probably accessible to most readers, I do not think it necessary to transcribe the passage, which contains, however, a very accurate description of the lesion in question—(see p. 174, op. cit.) In regard to the characters of these abscesses, as distinguished from tubercle, my observations, as detailed above, almost exactly concur with those of Dr West. In one point alone I am disposed to differ from him. He seems to regard the abscesses as not formed in truly *carnified* lung, because the condensed por-

tions are not always *exactly* circumscribed by the interlobular tissue. I believe this appearance will be fully accounted for by the fact that when bronchial abscesses occur, the bronchitis is usually general and intense, and the collapse correspondingly diffused. Besides, I have found bronchial abscesses, and their results, in exactly circumscribed lobules, in repeated instances; and that the tissue in which they occur is mostly collapsed there can be no doubt, from the whole of its ordinary and microscopic characters. (See Case V.)

With these remarks I shall lay before the reader a few examples of this interesting lesion, which undoubtedly forms one of the most frequent consequences of intense bronchitis in children, and is also not rare in adults. With the exception of Case V., these observations, like many others which I have recorded, were made without the knowledge of those of the other authors mentioned above.

CASE V.—*Death from Dysentery—Extensive Collapse of Lung, with Bronchial Abscesses—Emphysema of Lung.*—(P. R. 350.)

A woman, æt. 30,—body examined June 8, 1850.

The body was much emaciated and pale.

Heart normal. Both pleuræ contained a small quantity of fluid, with some floating and adherent soft lymph. The lungs presented great variations in density; the anterior edges were partially emphysematous, but between the portions thus affected could be felt numerous condensed parts, which, when superficial, presented a somewhat sunk collapsed appearance, and a deep purple colour. At the posterior part of the lungs were considerable masses similarly condensed. On cutting into the pulmonary tissue, there were seen, throughout the condensed portions, numerous small yellow points resembling softened tubercles, but more irregular in outline; these, when scraped with the knife, were found to be bronchial tubes, or small cavities filled with and surrounded by pus. Except at these points, the condensed tissue yielded to the knife only a little sero-sanguinolent fluid, which, when examined under the microscope, contained mostly blood corpuscles, with a few epithelial cells and pus corpuscles.

The colon presented a marked example of follicular dysenteric ulcers, which had destroyed a considerable portion of the mucous membrane.

This case presents an example of the very first stage of the disease, the mucous membrane of the bronchi being as yet scarcely destroyed. It is the same affection of the lung as àppears to be figured by Dr Addison in Guy's Hospital Reports, Series 2d, Vol. III., Plate 3. Its resemblance to tubercle, as well as its distinguishing characters, have been perfectly correctly appreciated by Dr Addison.

CASE VI.—*Bronchitis after Fever, with Collapse of Lung—Death from this cause and Dysentery—Bronchial Abscesses.*—(P. R. 62.)

A boy, æt. 11, admitted under Dr Bennett, 13th Dec. 1848, in a delirious state, and with the symptoms of typhus fever. He continued in this state with weak pulse up to the 22d, when the tongue was cleaner, the pulse still weak, and there was slight erythema on the back. Next day cough attracted attention; the chest was somewhat dull at the lower part; there were sibilant râles, and on the 24th slight crepitation over the lower lobes of either lung. On the 27th dysenteric symptoms supervened, and continued till his death.

Erythema also spread over the face. On the 6th January there was dyspnœa with clogged bronchial tubes. He died in the course of the night.

The dissection was performed on the 9th January. The body was considerably emaciated.

The posterior part of the left lung was gorged with blood and serum, and crepitated very imperfectly; in most parts of the inferior lobe, and at the lower border of the upper, it was quite dense and contained no air. The condensed portions were not granular on section, but gave out on pressure a considerable quantity of reddish serum; throughout them were irregularly disposed yellowish opaque tubercular-looking points, which could be seen and felt through the pleura, being more resisting than the rest of the tissue; they did not exceed in general the diameter of 2 or 3 lines, and were irregularly circular in form. On pressure, they yielded a quantity of yellowish fluid mixed with a small quantity of air; and most of them could be seen to be perforated by an oval aperture admitting a probe, by which the communication of these cavities with the bronchi could be easily ascertained in all the larger ones. On slitting up a few of them, they were found lined with a reddened injected mucous (?) membrane. The lower edge of the right lung was partially condensed, and contained enlarged and thickened bronchial tubes (?) in the condensed portion.

The mucous membrane of the colon presented extensive ulceration, the characters of which need not be particularly detailed.

It will be seen from the expressions now marked (?) that this, like some of the other cases given, contains what I now consider the results of imperfect observation. The "thickened bronchial tubes" and the "mucous membrane" here adverted to, are in fact not normal structures, but false membranes supplanting those which have been destroyed by ulceration. Of this I am now well assured by other observations.

CASE VII.—*Intense Bronchitis—Bronchial Abscesses and Gangrene of Lung—Emphysema of Anterior Edges.*—(P. R. 284.)

A labourer, æt. 40, admitted under Dr Bennett, Jan. 6, 1850, with intense cough and dyspnœa. Had been sometime affected with these symptoms, but more severely for a week before admission. Face swollen, lips livid, difficulty in inspiration. Chest arched, very resonant on percussion. Expiration prolonged. Sibilant râles, with inspiration, occasionally. Expectoration tenacious, frothy, muco-purulent. On the 20th he was relieved; on the 25th much worse—feeling of "smothering." 26th, Quiet at night, but dyspnœa very great in the morning. Died at 9½ A.M.

On dissection, the heart was found soft, and its fibres granular under the microscope. Both pleuræ presented adhesions, not very firm. The bronchi were filled with frothy sanguinolent pus, and their mucous membrane was very red. The crepitation of the upper part of both lungs was impaired, but was nowhere completely destroyed over a large space. Distributed throughout the lung were small condensed patches, which gave to the touch somewhat the feeling of tubercles, but on section appeared to be less consistent and more diffused. A few condensed patches of larger size than the majority existed in the lower and anterior part of the left lung, and one of these on section displayed a distinct gangrenous cavity, capable of containing a cherry-stone, lined by a tolerably distinct membrane, and containing a fetid sanies intermixed with debris of fibrous tissue. Anterior edges of both lungs emphysematous.

CASE VIII.—*Measles, with intense Bronchitis—Collapse of Lung—Bronchial Abscesses—Partial Emphysema.*—(P. R. 272.)

A boy, æt. 8, admitted December 19, 1849, under Dr Bennett, suffering under measles, with mucous râles over the chest, which was clear on percus-

sion. On the 24th, he had an attack of croupy breathing, relieved by an emetic. On the 26th, chest symptoms aggravated, with loss of appetite, and fever; crepitating râles all over chest; but no dulness on percussion. *5th January* 1850.—Much dyspnœa, pulse quick and feeble. *6th.*—Dulness observed behind on both sides of the chest. Died on 10th January.

Dissection performed January 11th. The lungs collapsed imperfectly on opening the thorax; they were emphysematous at their anterior edges, and presented some irregularities of surface. Condensed lobules were felt in various parts,—frequently in close contact with, or in the midst of, the emphysematous portions. On section, the lower lobes presented irregularly disposed and irregularly formed yellowish deposits,—the largest of the size of a small bean, frequently perforated by bronchial tubes, and less sharply circumscribed than tubercles.

The last three cases, the history and symptoms of which during life are extracted from the clinical case-books of the hospital, will, I think, appear to the observant physician the type of many others which have come under his notice, especially if his field of observation have embraced the diseases of children, or of hospital patients enfeebled by fevers and chronic diseases. The rapid supervention, and sometimes equally rapid evanescence, of dulness on percussion, limited or diffused, in the midst of attacks of general bronchitis of greater or less intensity, and when the respiratory sounds are muffled or supplanted by large and small mucous râles in every part of the chest, is an occurrence which has impressed itself on my recollection in numerous instances,—of most of which, however, I have no records, or only notes too meager to be placed before the reader. Such an event comparatively seldom occurs in the acute catarrh of healthy adults; but, as a sequela of measles and hooping-cough in children, it must be familiar to every one; and I have seen it again and again in adults, in the bronchitis of fever,—in that which often terminates Bright's disease, or disease of the heart,—and still more frequently in those obscure and treacherous chest affections, which supervene in the last stage of exhaustion from hectic or malignant disease, and which are the accompaniments, rather than the active causes, of breaking-up of the enfeebled frame. Such affections have commonly been called latent pneumonia, and they undoubtedly may be so in some instances; but much more commonly they are nothing more than bronchitic collapse, determined by obstruction so slight as would in an otherwise healthy individual be easily overcome, and which, even in these emaciated and bloodless subjects, is only *not* overcome because the exhaustion is great and the need of respiration small,—the circulation being at a low ebb, and the attenuated and sluggish blood requiring very much less air, and consequently less lung, for its renovation than under ordinary conditions. Again and again has it occurred to me, under such circumstances, to open the bodies of persons in whom no suspicion of a respiratory affection existed during life, and in whom, nevertheless, condensation, abruptly lobular or diffused, having often the characters of the "peripneumonie des agonisans," or the "peripneumonie hypostatique" of M. Piorry (both of them varieties of the bronchitic collapse), has been

found after death affecting large portions of one or both lungs.[1] On the other hand, I can remember instances in which a superfluous zeal, or *nimia diligentia*, in stethoscopic studies, has detected the signs of these lesions, when not a single rational symptom existed to call for such examination; and very many cases where the extent of the lesion was altogether out of proportion to the gravity of the symptoms that attended its accession.

Apart, however, from such latent, or very obscure cases, it has often been remarked of late years, that the pneumonic affections springing from or accompanying bronchitis, are apt to present a peculiarly asthenic or typhoid character. So much has this been the case in France, that within the last few years "pneumonie catarrhale" and "pneumonie typhoide" have become almost synonymous terms with some authors. Seeing that a great number of cases of so-called broncho-pneumonia which have come under my notice have been instances, not of true pneumonia, but of bronchitic collapse, sometimes combined with engorgement or œdema of the lung, and sometimes with bronchial abscesses, I am inclined to believe that the solution of the asthenic or typhoid character of this disease will be found in a modified view of its pathology. The fact is, that in adults the tendency in bronchitis to complete collapse of the lung is comparatively small, excepting in exhausted or debilitated constitutions, or under the influence of typhoid affections of the system. In these the asthenia is not a result of the disease, but one of its most essential predisposing causes.

In the child, again, owing to causes which will be presently mentioned, collapse, in its most complete form, very readily occurs under all circumstances, as a consequence of acute bronchitic attacks. In commenting upon this peculiarity, Dr West, whose opinion on this subject I have before referred to, remarks that it is by the collapse of the lung "that we must explain many of the instances in which urgent dyspnœa, and all the symptoms of serious pulmonary disease, have developed themselves in the course of a few hours out of what had seemed to be a severe cold, or a bronchitis of moderate intensity. This, too, accounts for the occasional supervention of dulness on percussion, and of bronchial respiration in the child; so that you may discover them in the morning in a situation where, overnight, the percussion was good, and no sound of graver import than large crepitation."[2] To this remark I would only add, that dulness on percussion supervening under such circumstances is not necessarily attended with any increase of fever; and that careful attention to the state of the patient in this respect may be in some cases the only mode of forming an opinion with any degree of accuracy as to the presence or absence of true pneumonia in a case of bronchitis. Of

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[1] Instances of this are to be found in Cases I., III., IV., V.

[2] Op. Cit. p. 183.

this fact a striking instance occurred to me lately in the case of a little boy, three years old, whom I attended during an attack of acute bronchitis. He was a delicate child, and had suffered several times from bronchitic attacks. His chest presented a good deal of deformity, particularly on the left side, of the kind called "pigeon-breast." On the fourth day of the attack, which had all the usual acute characters during the first three days, decided dulness on percussion, with obscurity of respiration, appeared in the right back; the dyspnœa continuing considerable, but *the fever rather abating than increasing*. Under the influence of an emetic, and a forced decubitus on the opposite side to that on which the dulness existed, almost every trace of it had disappeared in thirty-six hours, and the dyspnœa was entirely relieved.

If these views be correct, they cannot be without practical importance. The rules, both of diagnosis and treatment, in regard to bronchitis and its complications, must probably undergo some modifications, in order to be safely followed. Such an axiom as the following, at least, which is one very generally conceived to be unquestionable, will not, I believe, stand the test of renewed observation, if the fact of collapse of the lung from bronchial obstruction be kept in view. "The stroke-sound of the chest," says Dr Williams, "is not materially impaired by catarrh; and, accordingly, the partial suspension of the breath-sound in a part of the chest in this disease *cannot be mistaken for that caused by hepatization*."[1] The reader will at once perceive that the clearness of the stroke-sound here alluded to is not always, perhaps not even generally, to be expected under such circumstances; nor can the opposite condition, even when combined with diminished or suppressed respiratory murmur, or with bronchial respiration, be allowed necessarily to indicate hepatization. The judicious physician has doubtless often been saved from error in the application of his remedies in this, as in many other cases where an unsound pathology has prevailed, by adhering to the great principles of our art, and the teaching of personal experience, rather than to received formulas and rules on matters of detail. But it is difficult to escape from the conviction, that the influence of a name, or the vanity of an *exact* diagnosis, may have misled many into such errors; and that in cases of supposed broncho-pneumonia or typhoid pneumonia, the practice may frequently have embraced blood-letting, heroic antimonials, or calomel and opium, when emetics and expectorants, with suitable stimulation, would have been better adapted to the circumstances of the case. This will be still more evident on considering the mechanism of these affections.

Since the preceding observations were in type, I have received the

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[1] Williams on Diseases of the Chest, 4th edition, p. 80. Similar rules of diagnosis are given by Laennec, Skoda, Watson, and most other writers.

recent work of Dr Fuchs, on the bronchitis of children,[1] which in its elaborate descriptions, both of symptoms and post-mortem appearances, furnishes additional evidence of the connection of collapsed lung with bronchitis. The state of the lung, called "lobular pneumonia" by most authors, and "état fœtal" by Legendre, is regarded by Fuchs as a direct consequence of bronchitis; and its relation to bronchial obstruction on the one hand, and to peculiarities in the infantile system on the other, is certainly more clearly stated than by any previous author. To distinguish this lesion from the congenital form of collapse, as well as from other varieties of condensation, Fuchs proposes the term *apneumatosis*;—but, although evidently anxious to point out a sufficient anatomical ground of distinction between the *unexpanded* and the secondarily *collapsed* lung, he admits that the *diagnosis* must rest chiefly on a consideration of the cause—the one being congenital and the other acquired (See pp. 112, 113). Under these circumstances it will probably, I think, appear to English readers unnecessary to burden the science of pathology with another scholastic term; and I am convinced that careful examination of both lesions will convince most observers that Legendre and Bailly are correct, and that there is no real ground for distinguishing them, excepting what may be inferred as to their mode of origin.

The simplicity and clearness of the pathological views entertained by Fuchs, as compared with most other writers on this subject, and the highly original character of his work, render it a most important contribution to the history of bronchitis; and as such, I shall have occasion to refer to it in the sequel. In the meantime, it is only necessary to say that he enumerates three stages of *apneumatosis*, each of which is described at great length. Into the anatomical description of these I do not mean to enter, as it refers exclusively to children below the age of five years. The reader of the original work will see many points of similarity between the characters there given and those which I have indicated as distinguishing bronchitic collapse in the adult as well as the child. In the case of children under five years, Dr Fuchs, indeed, denies having seen a true pneumonic condensation; and he appears also to have passed over, very lightly, the lesion above described as bronchial abscess; the only allusion to it being in p. 114, where he remarks, cursorily, on "the accumulation of yellow mucus in the bronchi and air-cells, *and their dilatations*;" this being, as he declares (in opposition to Friedleben, as well as to other authors), the only form of suppuration in the lung observed in early infancy. These assertions are entitled to due consideration, but can scarcely be accepted without further, and even more careful, examination.

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[1] Die Bronchitis der Kinder, &c., von Dr Caspar Friedrich Fuchs. Leipzig. 1849. The reader will find a notice of this work in the last No. of the British and Foreign Med. Chir. Review.

It appears from the work of Dr Fuchs, that, in 1837, a year before the publication of the monograph of Rilliet and Barthez, Seifert[1] had given an excellent description of infantile bronchitis, and of the peculiar pulmonary lesions in which it commonly terminates, which he considered to be a peculiar form of pneumonia. This work seems to have excited some attention in Germany, and it is not a little remarkable, under these circumstances, that the close relation of these lesions to the congenital affection described by Jörg, should have been overlooked until the observations of Legendre and Bailly, in France, seven years afterwards. Seifert also noticed the resemblance of the "broncho-pneumonia" of children, to the "peripneumonie des agonisans" of adults; and seems to have described the bronchial abscesses as a stage of suppuration.

Mechanism, Causes, &c., of Bronchitic Collapse.—The object of the remarks hitherto made, in reference to this subject, has been chiefly to establish the frequent coincidence and probable relation of cause and effect between the obstruction of the bronchi and the collapse of the air-vesicles. The nature and rationale of that connection now fall to be more particularly considered.

When a bronchial tube is in any way obstructed, or much diminished in caliber, at one or more points, the question arises, what is the mechanical effect of the movements of respiration upon the amount of air thus partially imprisoned behind the obstructed part? If the obstruction be complete, of course no change can take place, at least mechanically; but in the case of its being, as it usually is, incomplete, it may be supposed that the inspiratory act tends to draw in more air than the expiration can expel, and consequently that the air tends to *accumulate* in the vesicles; or, *vice-versa*, that the air behind the obstruction tends constantly to *diminish* in amount, owing to the comparative inefficiency of the inspiratory act; or finally, that the forces equalise each other, and the quantity of the enclosed air *remains unaltered.*

Now, it cannot be denied that from the pathological anatomy of bronchitis, a *prima facie* case might be made out for each or all of these theories; for, although only one side of the question has hitherto been brought prominently forward in the preceding part of this paper, the reader will not have failed to remark that in several cases in which bronchitic collapse of the lungs existed, other parts of the same organs were affected with emphysema or dilatation of the air-cells. (See Cases III., IV., V., VII., VIII.) Indeed, so familiar is this conjunction of emphysema with bronchitis, as to have suggested to Laennec, long ago, the first of the three theories above mentioned as to the cause of dilatation of the air-vesicles in that disease. Again, fatal cases of bronchitis undoubtedly occur, at least in adults, in which

[1] Die Bronchio-pneumonie der Neugebornen. Philipp Seifert. Berlin, 1837.

there is no change of the pulmonary texture so marked as to afford support to either of the first two theories.

Laennec's view of the consequences of obstruction was founded on the idea of the comparative weakness of the expiratory, as compared with the inspiratory, forces. "The mucus secreted into the bronchi, in consequence of pulmonary catarrh, must, especially if it is very viscous, present a great resistance to the free passage of air in inspiration and expiration; and we shall show, in speaking of the râle, that this resistance often goes the length of producing complete, though momentary, obstruction of a part of the bronchial ramifications. Now, as the muscles which subserve inspiration are strong and numerous, while expiration is produced only by the elasticity of the parts, and the weak contractions of the intercostal muscles, it must necessarily happen that the air, which has been forcibly driven beyond the obstruction in inspiration, will not be able to overcome it in expiration, and will be in a manner imprisoned, by a mechanism not unlike that in the butt-end (condenser) of an air-gun." I have given this passage from Laennec entire, because, notwithstanding the palpable fallacy it contains, it has been referred to, and its conclusion adopted without comment, by almost every systematic writer in this country as well as in France. The fact is, however, that though *ordinary* inspiration is more of a muscular act than *ordinary* expiration (merely because in the latter there is little or no resistance to be overcome, to which the elastic subsidence of the parietes is not adequate), yet the residual effective force for overcoming adventitious obstruction is very considerably greater in expiration. The *forced* or *muscular* expiratory act is, in fact, about one-third more powerful, as measured by its effect upon a pressure-gauge, than the extreme force of inspiration;[1] and it is this force which is thrown into action when obstruction in the tubes is to be overcome. In the act of coughing, moreover, we find a beautiful mechanism, by which the air within the vesicles is discharged outwards at a *maximum amount* of pressure, and brought to bear with all the additional mechanical advantage of a sudden impulse, on every obstructing substance within the bronchial tree,—a cumulative provision which does not exist in the case of the inspiratory force. There can be no great difficulty, therefore, on these grounds, in coming to the conclusion, that the data of Laennec's hypothesis are quite erroneous, and that the practical efficiency of the expiration in forcing air through obstructions must be, *cæteris paribus*, far greater than that of the inspiration. I have already alluded to the fact, that this is consistent with general experience; for while the inspiratory act is always, in bronchitis of considerable intensity, attended with extreme difficulty, the expiration is never so.

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[1] See the numerous experiments of Hutchinson and Mendelsohn, quoted in Dr J. Reid's article on Respiration: Cyclop. Anat. and Physiol. Part 32, p. 336.

The question of the origin of emphysema of the lung will be considered in the second part of this memoir, in which I shall endeavour to account for its connection with bronchitis, by referring its production to a totally different mechanism from that just mentioned. In the meantime, I would remark that, in order to establish a direct relation between this lesion and bronchial obstruction, it is necessary to show not merely that emphysema occurs in connection with bronchitis, but that it occurs especially or exclusively *in those parts of the bronchitic lung where obstruction can be shown to exist.* This is the proposition which I conceive the preceding pages have tended to establish as regards bronchitic collapse, and on the ground of which I have argued for the relation of cause and effect between this lesion and obstruction. In how far does emphysema fulfil these conditions?

It is well known to every one who has studied the anatomy of this pathological state, that the emphysematous portions of a lung can generally be inflated from the bronchi with the greatest ease. Indeed, so far as my own experience in this matter is concerned, I cannot recal any instance in which the pressure of the air was not found to reach the emphysematous parts with as great rapidity as the rest of the lung. In the collapsed lung, on the contrary, as I have already shown, very considerable resistance is often opposed to its inflation from the bronchi—a resistance only to be overcome by pressure many times greater than can ever occur in the vital act of inspiration. If this observation be correct, it is plain that the emphysematous parts of the lung are usually free, the collapsed parts obstructed.

Further, I cannot find that any unequivocal instances have been adduced, to prove that an obstruction, confined to a part of a lung, or to one lung only, is commonly accompanied by a corresponding distribution of emphysematous portions—a proposition which, if true, might surely be easily verified from the records of pathology. So far from this being the case, the habitual seat of emphysema leads to an inference of a directly opposite kind—a point which appears to have been overlooked in the discussion of this question. Let the reader reflect that, in the vast majority of instances, the seat of election of emphysema is the *anterior* border of the lungs, while the stethoscope, as well as the results of *post-mortem* examination, show that accumulations of mucus in bronchitis occupy in an equally numerous proportion of cases, the *posterior* and *lower* parts, which are also, especially in the adult, the principal seat of the bronchitic collapse.

Cases of the impaction of foreign bodies, and other palpable obstructions of the bronchi, are generally recorded with too little attention to the condition of the lungs to be available for the present discussion. Carswell has, however, figured the case of a monkey, in which the left bronchus was much compressed, or rather obliterated, by a mass of tuberculous glands; in this case the corresponding lung had diminished to less than a third of its normal bulk, while

the *opposite* lung, of which the bronchus was free, presented emphysema in several places.[1] Andral has adduced, as before mentioned, two cases of obstruction of the upper lobe of one lung, where, from the stethoscopic phenomena, he supposed emphysema to be present, but where the examination after death proved that this was not the case.[2] He has also recorded a case[3] in which the bronchi of the right lung were compressed by a melanotic mass, and the respiratory murmur greatly enfeebled. In the short note of the appearances after death, no notice is taken of any abnormal condition of the lung in this case. Andral, indeed, states (p. 196) that emphysema is one of the consequences of stricture of the bronchi; but adduces nothing whatever in proof of this assertion, which evidently rests on the ground of Laennec's theory.

All doubt, however, as to the real effect of a solid obstruction in the bronchi on the air in the lung is removed by the direct experiments of Mendelsohn and Traube on animals.[4] The former inserted a leaden shot into the trachea of a dog, pushing it down as far as possible into the bronchus with a probe. In another instance he inserted a ball of paper. In both cases, the parts to which the obstructed bronchi led were red and void of air. In the former there were emphysematous portions in the other parts,[5] and in the opposite lung.[6] Traube's experiments were similar, but more numerous. The general result was, that the artificial obstruction of a bronchus always produced expulsion of the air from the corresponding part of the lung, which had a dark-red colour, and presented the characters of collapse.[7]

It is clear, therefore, from experiment, as well as from pathological observation, that the most usual and most direct effect of obstruction, or of diminished caliber of the bronchi, however caused, is not accumulation, but diminution in quantity, of the air beyond the obstructed point. It is probable that this is due in part to the comparative weakness of the inspiratory power, and that the proposition of Laennec may, therefore, correctly enough be inverted. There is also, however, another mechanical condition which comes into play in producing collapse from obstruction, especially in the case of a viscid

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[1] Illustrations of the Elementary Forms of Disease—Atrophy. Plate iv., fig. 3.

[2] Clinique Médicale, v. 2, pp. 187-190. [3] Ibid, p. 193.

[4] For an account of these experiments I am indebted to the work of Fuchs, not having access to the original sources.

[5] The expression of this passage is not quite clear, but this is certainly the meaning, and corresponds with the author's inference.

[6] Der Mechanismus der Respiration und Circulation, p. 37. Berlin, 1845. Mendelsohn also threw a solution of gum into the air passages of an animal, with the result of collapse of some portions of the lung. In one instance I tried this experiment upon a rabbit, with a similar result; but the difficulty of limiting the fluid to particular parts of the lung makes these experiments less valuable.

[7] Beitrage zur experimentellen Pathologie und Physiologie, 1 Heft.

plug of mucus, which is most commonly, in bronchitis, the source of this affection. This condition is to be found in the form of the tubes.

The bronchi are a series of gradually diminishing cylinders, dividing, for the most part, dichotomously. If a plug of any kind, but especially one closely adapted to the form of the tube, and possessing considerable tenacity, be lodged in any portion of such a cylinder, it will move with much more difficulty towards the smaller end, and in doing so will close up the tapering tube much more tightly against the passage of air, than when moved in the opposite direction into a wider space. If such a plug be placed over a bifurcation, it will, even if freely moving in the larger space in which it lies, be of sufficient bulk to fall back upon one or other of the subdivisions during inspiration, in the manner of a ball-valve upon the orifice of a syringe, and thus completely to occlude it. (See diagram.) The consequence of this mechanical arrangement must inevitably be, that at every expiration a portion of air will be expelled, which, in inspiration, is not restored, partly owing to the comparative weakness of the inspiratory force, and in part to the valvular action of the plug. If cough supervene, the plug may be entirely dislodged from its position, or expectorated, the air of course returning freely into the obstructed part; but if the expiratory force is only sufficient slightly to displace the plug, so as to allow of the outward passage of air, the inspiration will again bring it back to its former position, and the repetition of this process must, after a time, end in perfect collapse of the portion of lung usually fed with air by the obstructed bronchus.

It is not a little surprising that this simple and clear mechanical mode of explaining the collapse should not have occurred to Dr Fuchs, who, in accounting for the disappearance of the air in the experiments of Mendelsohn and Traube, finds himself reduced to the theory of its absorption into the blood-vessels. (Op. Cit., p. 63.) Surely nothing can be more superfluous than such an explanation.

In considering, as a whole, the causes which tend to produce bronchitic collapse (as revealed in the preceding investigation), they seem to resolve themselves into the following:—*Firstly*, the existence of mucus in the bronchi, which is more liable to produce obstruction according as it is tenacious and viscid; *secondly*, weakness, or inefficiency of the inspiratory power, however caused; *thirdly*, inability to cough and expectorate, and thus to remove the obstructing mucus. Of these conditions the first must be considered as the immediate exciting cause, the others as predisposing causes, co-operating with the first, but incapable without it of producing collapse. Of the exciting cause enough has already been said. The inability to expectorate is obviously enough a formidable condition, and may be owing either to simple debility, or to a laryngeal affection. But I have

still a few remarks to make on the circumstances producing inefficiency of the inspiratory act, and their bearing on the present subject.

The inspiratory act is apt to be rendered inadequate from several causes. Of these the most obvious is weakness of the muscles of inspiration, usually concurring with general debility. I have already pointed out the great predisposition to bronchitic collapse which arises from an exhausted frame; so much so, that a barely appreciable amount of bronchitis, nay sometimes, I believe, the mere accumulation of the natural mucus in a debilitated subject (as in an individual near death), will give rise to a considerable extent of the pulmonary lesion. I cannot, however, see reason to believe with Dr West, that mere debility, apart from any obstruction in the tubes, is a sufficient cause for collapse in the child. The very fact of the lesion being usually more or less lobular, or partial in its distribution, appears to indicate special circumstances of a local kind, as having a marked influence on the production of this affection; and on this ground, as well as that of theory, I am disposed to think that, in the cases ascribed to debility alone (including the third case of Dr Baly, before alluded to, p. 19), the evidences of more or less obstruction might have been detected during life or after death. That this, however, is often of a very slight character, leading to scarcely any symptoms, and probably in some cases undiscoverable, except by physical examination, I have already indicated. (See p. 24; also, Cases I., III., and IV.)

A second circumstance tending to render the inspiratory act ineffective is distension of the abdomen, impeding the descent of the diaphragm. The influence of this condition in the production of collapse I have repeatedly witnessed; having had occasion to observe that when ascites, or any other cause of similar distension, is present, a very slight amount of bronchitis will determine extensive pulmonary collapse. In Case I. this cause was in action, combined with great exhaustion; and one of the first cases that awakened my attention to the subject of this memoir, was that of a boy of 17 years of age, who died of an enormous medullary tumour of the abdomen, and in whom a most marked form of lobular collapse was found distributed over both lungs, with a good deal of thick mucus in the tubes.

A third cause of inefficiency of the inspiratory act, and one of the greatest importance in relation to this subject, is the want of due resistance on the part of the thoracic parietes. The full dilatation of the lungs is only effected when the depression of the diaphragm is accompanied by the elevation of the ribs and widening of the thorax; and if the bones of the latter be very yielding, the external muscles of inspiration cannot, of course, act effectively under an obstruction. This is obviously the reason of the greater tendency in children to collapse of the lung as a consequence of bronchitis. The respiration of the child is at all times, even in health, more diaphragmatic than that of the adult; and the observations of Rilliet

and Barthez[1] afford satisfactory evidence of the comparatively small dilatation of the thorax in children, particularly of its lower part. When any obstruction exists to the entrance of air into the chest, even this small dilatation ceases, and collapse of the lung very readily takes place. Under such circumstances, Dr Rees[2] has pointed out that in very young children the motions of the chest are absolutely reversed, and instead of the descent of the diaphragm being accompanied by expansion of the chest, the ribs give way beneath the exhaustion caused by it within the thoracic cavity, and bend inwards to accommodate themselves to the collapsed lung in inspiration. This altered movement of the chest in infants is regarded by Dr Rees as pathognomic of *atelectasis*. It is also a prolific source of that permanent deformity of the chest which, in the early years of life, is often ascribed, with too little discrimination, to rickets.[3] Of this deformity I shall have something to say in the second part of this paper.

As to the so-called *atelectasis*, I have had but few opportunities of observing it accurately during life. I may, however, remark that in respect to its causation, it probably differs but little from the acquired collapse, and close examination would probably show that mere debility, without some obstruction in the bronchi, is as inadequate to prevent the expansion of the lung as to cause its collapse. The cases published by Jörg himself, although very imperfectly observed as regards physical signs, will, I think, on perusal, convince a careful reader that there is abundant ground for this opinion; but undoubtedly the subject requires renewed investigation.

Origin of Bronchial Abscess.—The mechanism of this lesion it is not difficult to explain satisfactorily. When pus accumulates in the central bronchi of a collapsed lobule, the evacuation of that pus is prevented from occurring, firstly, in consequence of the absence of the expiratory *vis a tergo;* and secondly, from the resistance opposed by the thickened mucous membrane and its secretion, closing up the bronchus in front. The coats of the ultimate bronchi, therefore, softened and injured by disease, gradually give way to ulceration; and the pus, which thus accumulates in still larger quantity, may at first scarcely be circumscribed, but soon begins to be surrounded by a false membrane exactly similar to that of an abscess in any other part of the body. The continuity of this membrane with that of the original bronchus, may be either maintained from its first formation, or it may be secondarily established. I believe, however, that

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[1] Op. cit., vol. iii., pp. 643, 644.

[2] Atelectasis Pulmonum. Lond. 1850.

[3] Rilliet and Barthez describe a reversed movement of the ribs in inspiration as taking place in rickets. There can be little doubt that rickets, combined with chest affections, forms a frequent source of the deformity; but the presence of the latter is probably essential. .Vol. iii., p. 646.

the first of these views is the correct one; and that the bronchus acts the part of an obstructed fistulous opening, not sufficiently pervious to prevent accumulation entirely, but not permitting of its increase beyond a certain amount.

When the bronchial abscess has been of some standing, and the patency of the tube leading to it has become re-established in time to prevent its obliteration, a process of repair takes place, analogous to the cicatrization of a wound, and perfectly similar to that which is observed in all healing excavations in the lung, however formed. The false membrane which lines the cavity becomes intimately blended with the bronchial mucous membrane, and indeed comes to resemble it so closely that it is almost impossible to tell where the true mucous membrane ceases and the new structure begins.

This reparation, however, is rarely, if ever, accompanied by restoration of the perfect function and structure of the lung; and on this account it will be considered under the permanent effects of bronchitis.

The length to which these remarks on collapse of the lung have extended, can only be excused by the immense importance of this lesion in relation to the pathology of bronchitis; the whole of the organic affections following from which seem to me more or less dependent on that which has formed the principal subject of these observations. These secondary effects of bronchitis and bronchitic collapse will form the subject of the second part of this memoir.

PART II.

SECONDARY RESULTS OF BRONCHITIS; OR, PERMANENT DISORGANISATIONS DEPENDING ON COLLAPSE OF THE LUNG, ETC.

In discussing the primary affections of the pulmonary texture resulting from Bronchitis, I have treated, at considerable length, of the collapse of the air-vesicles connected with obstructed tubes,—a lesion of which, as I have endeavoured to show, the true pathological significance has been much neglected or misapprehended, even by authors who have correctly enough described some of the morbid appearances. The more chronic and permanent lesions connected with long-continued bronchitis have now to come under consideration; and although these affections cannot be said, for the most part, to have engaged less than a due share of the attention of pathological writers since they were made familiar by the descriptions of Laennec, yet it will appear from the sequel, that the links which bind them together in a series, cannot be understood without reference to those primary results of bronchitis to which I have already adverted. In describing these affections, therefore, I shall avoid as much as possible dwelling on descriptions already familiar to well-informed medical readers, confining myself, for the most part, to the pathological considerations which flow more or less directly from what has already been advanced in the first part of this memoir.

Results of Bronchitic Collapse of the Lung—Curability of Pulmonary Collapse.—There can be little doubt that the condition of collapse of the air-vesicles, from obstruction of the bronchi, may, when recent, be completely removed, and give place to the normal condition of the pulmonary texture. The imitation of this result, by forcibly inflating the lung so affected after its removal from the body, not only proves the absence of any organic change, but shows conclusively that it is in many instances only a sufficiently strong inspiratory force which is required to disperse the obstructing mucus, and make a free passage for the entrance of air into the lung. The collapsed lung, however, is placed under a most serious disadvantage, as compared with that which contains air, in freeing the bronchi from causes of obstruction. The latter can render available the expiratory

force, and this, when aided by the impulsive effort of coughing, is by far the most efficient agent in displacing and removing the bronchial plug, which it carries outwards, and expels altogether from the system. The completely collapsed lung, on the other hand, can only bring into play the inspiratory force, a weaker power acting against a greater resistance, and able, at the best, only to disperse inwards, never directly to remove, the cause of obstruction. Under these circumstances, it might seem probable, that a lung when once brought into a state of complete collapse, should be mechanically incapable of perfect recovery; and even in minor degrees of the affection, it would appear that the tendency *inwards*, or towards the lung, of mucus, and all other obstructions, must greatly increase in proportion as the residual quantity of air in the vesicles diminishes. Again, where the obstruction is much localised, as in limited and lobular collapse, the mechanical forces tending to remove it, whether inspiratory or expiratory, will at all times be apt to diffuse themselves over the surrounding normal or comparatively unobstructed lobules, so that the removal of the bronchial plug under such circumstances is not easily understood, if we take into account only the forces we have hitherto been considering.

Does collapse of the lung, then, necessarily, or in the majority of instances, lead to organic and permanent change of structure? This doctrine I should be very slow to admit. A consideration of the cases in which bronchitis occurs, and is even repeated frequently in the same individual, without appreciable permanent change, while we know, from post-mortem appearances, and understand on mechanical grounds, that accumulation even to a very moderate extent in the bronchi, is often sufficient to cause a certain amount of the lesion, will, I think, even in the absence of more detailed clinical experience, constitute a strong case for believing in the existence of some more active remedial and conservative mechanism in such cases than that of the inspiratory and expiratory forces. Such a view is altogether borne out by the observations of writers on the bronchitis and lobular pneumonia of children, which, though often a grave, and even a fatal affection, is never regarded as being, in favourable cases, less capable of perfect resolution than any other form of pulmonary condensation. The remarks of most other practical writers are so much governed by pathological views, differing from those we have been considering, that it is quite impossible to eliminate the information they may contain as to the results of pulmonary collapse. I have already remarked that a great number of the varieties of so-called catarrhal and typhoid pneumonia are undoubtedly affections of this kind, sometimes combined with genuine pneumonia, and sometimes uncomplicated; and in particular, that the hypostatic pneumonia of M. Piorry, and the "peripneumonie des agonisans" of Laennec, are generally instances of the diffused form of pulmonary collapse. The former observer has devoted so much attention to the observation of this particular form of disease, as to render his remarks valuable,

even though probably modified by an erroneous pathology. He says:—"The first stage of hypostatic pneumonia, while the blood is still contained within the vessels (*i.e.*, while no exudation has occurred into the air cells,) is *very susceptible of cure;* indeed, it may be said that this state exists *in a large number of invalids* (chez beaucoup de malades), and *is dissipated during convalescence.*"[1] The other stages, especially the third and fourth, in which solid or purulent deposits exist in the air-cells, are, according to M. Piorry, more grave, and even generally incurable; but these again are obviously not uncomplicated instances of pulmonary collapse. I shall not at present enter more fully into the discussion of M. Piorry's views on this subject, than to remark, that the passage above quoted is in harmony with all that has been already submitted to the reader, more particularly with the observations alluded to in the last volume of this Journal, pp. 234–6. Indeed, I cannot entertain a doubt, judging from the facts there mentioned, that a more extensive and exact clinical experience bearing on this subject will demonstrate the extreme frequency, and in many cases the easy and rapid removal, of a certain degree of pulmonary collapse, which may or may not have led to serious symptoms during life.

De-obstruent Function of the Bronchial Tubes.—Supposing these views correct, the mechanism by which the viscid mucus is expelled to such an extent as to permit the return of air into the occluded vesicles, demands further consideration. We have seen that the expiratory forces are, under such circumstances, thrown out of action; while those of inspiration, even if strong enough to displace the obstructing plug, can never permanently remove it. Under these circumstances, it seems to be reasonable to ascribe to the bronchi themselves an active part in the expulsion of obstructive mucus, by means of the slow contraction of those circular fibres, the muscular character of which was demonstrated by Reisseisen, and whose physiological properties have been fully illustrated by the experiments of Dr Williams and others. It is now well established, that these fibres have no such vital endowments as would enable them to co-operate with the movements of respiration, influenced as these are by the will. "The contractility," says Dr Williams (of the bronchi), "resembles that of the intestines or of the arteries more than that of voluntary muscles or of the œsophagus, the contractions and relaxations being gradual and not sudden. They are, however, much less tardy than those of the arteries."[2] This kind of contractility is precisely that which empties the arteries of their blood after death, and which, in all probability, contributes to the passage of calculi along the ureters or gall-ducts. It is also more or less analogous to the peristaltic contraction of the intestines, or of the elongated tubular uterus of many of the lower animals, by which the

[1] Piorry—Pathologie Iatrique, vol. iv., p. 411.
[2] Williams—Diseases of the Chest, 4th Edition, p. 330.

solid or fluid contents of these viscera are gradually expelled towards their outlet. The experiments referred to appear to prove that the contractility of the air tubes is readily excited, not only by galvanism applied externally, but by mechanical and chemical stimuli in contact with their mucous membrane. It is easy, therefore, to understand, that the bronchi (or at least those which have not cartilaginous walls) may have a most important power of dislodging obstructions, altogether independently of the forces of respiration. When these forces are in active operation indeed, the tonic or slow contraction will be in abeyance, or very slightly manifested, as the air-tubes will then be dilated to their full extent at each inspiration and expiration. But, according as the admission of air to any part of the lung becomes less from obstruction, the detrusive action of the bronchial muscles will increase, being thus called into effective action precisely at the period when most required. Perhaps, also, the slighter contractions of these muscles may be in almost constant operation in the normal condition, to aid, by a kind of peristaltic movement, the outward passage of the physiological secretion. This secretion, comparatively small in quantity as it is, would almost necessarily tend to accumulate in the air-tubes (seeing that no efforts of coughing or forced expiration are made for its removal); and this would take place, particularly in the smaller bronchi, which we know to be especially subject to mechanical obstruction, and in which the ciliated epithelium, so abundant in the cartilaginous bronchi and trachea, gradually gives way to transition forms, not constantly furnished with cilia.

It may not be easy to adduce direct proof of the theory here proposed, as to the function of the bronchial muscles in health and disease; but as *no* theory upon this subject has yet been found consistent with our present physiological knowledge, and as the above speculation appears in all essential points to correspond with what is already known of the action of these muscles, it may be worth while to give it consideration, were it merely to rescue us from the unphilosophical predicament of supposing the circular fibres of the bronchi to be endowed with contractility, solely for the purpose of producing the asthmatic paroxysm. That these fibres are probably perfectly passive, as regards the respiratory act, is now generally admitted (contrary to the ancient opinion) by physiologists; and under these circumstances the theory of their de-obstruent action, even in health, but more especially in the diseased states of the pulmonary texture above described, appears to supply a gap in the chain both of physiological and of pathological phenomena.

The ordinary form of the paroxysm of spasmodic asthma, of the humoral kind, is full of instruction, when considered by the light of the preceding views. Notwithstanding the extremely doubtful and difficult pathology of this disease, it seems impossible to avoid referring its most obvious symptoms to some kind of irregular action of the muscular apparatus of the air-tubes. The copious expectora-

tion, with which the attack concludes, and by which it is immediately relieved, appears to indicate that undue accumulation of mucus has been taking place; while the absence, in some instances, of all considerable catarrhal symptoms, appears to demonstrate that this accumulation is directly connected with the spasmodic derangement which produces the paroxysm. The connection of these two phenomena it is by no means difficult to understand, according to the principles already laid down; in fact, if the removal outwards of the pulmonary mucus depends, in the normal state, upon the regular peristaltic contraction of the bronchial muscular fibres, it is obvious that accumulation must accompany the derangement of that action, just as constipation is the invariable concomitant of the analogous derangement of colic or ileus. In both cases the paroxysm ceases when the normal action is restored; and in general there is in both a copious discharge of the previously retained excretions.

Asthmatic persons are often subject to a slight habitual wheezing in some part of the chest, and also to an occasional cough, with or without slight expectoration, but with no other symptom of catarrh. These symptoms have been described to me as occurring on exertion in the open air after prolonged rest; they are accompanied with slight dyspnœa, and this, together with the rest of the symptoms, ceases when the exertion is continued long enough to produce some degree of re-action. These phenomena are unquestionably the minor degree of the paroxysm; they are probably caused by the same irregular action of the bronchial muscles as causes the latter, but do not reach the climax, because the nervous centres are awake to the first approaches of disorder, and the excitement and quickened respiration consequent on exertion produce the cure. The aggravated asthmatic paroxysm always occurs during sleep, when the energy of the nervous system is at the lowest, and the comparatively quiescent condition of the respiratory function favours the accumulation of mucus. It seems probable that the asthmatic paroxysm is attended with more or less of pulmonary collapse, the consequence of the accumulation in the bronchi; but I have not had an opportunity of direct observation on this point. It is certain, however, that this accumulation must seriously contribute to the production of the most distressing symptoms of the paroxysm. The spontaneous cure in the real paroxysm, as in the minor attack, or threatening of asthma, above referred to, usually takes place when the nervous centres have been thoroughly roused, and the whole system brought into a state of re-action by the exertion consequent on the dyspnœa.

An interesting fact, in connection with asthma and other spasmodic respiratory diseases, is the frequent occurrence of vomiting during the paroxysms,—a fact which points to the probable dependence of all these affections on some morbid condition in the communication of which the pneumogastric nerve and the medulla oblongata are the principal parts concerned. A phenomenon exactly the converse of that just alluded to, is the profuse and immediate expectoration

in cases of obstructive bronchitis after the administration of an emetic. Now, it is interesting to observe, in relation to both these facts, and their bearing on the subject we have been considering, that Volkmann has apparently succeeded in demonstrating the influence of stimuli applied to the trunk of the vagus nerve upon the muscular contraction of the bronchi,—a point left open to doubt, both by the experiments of Williams and by the subsequent ones of Longet.[1] The expeditious and complete relief afforded by an emetic in cases in which there has been extreme difficulty of expectoration, is one of the most striking phenomena connected with bronchitis; and one of which, I believe, no sufficient explanation has yet been afforded. It appears, however, to be completely in harmony with the theory I have advanced in the preceding pages.

Another fact tending still furthur to illustrate this view, is found in the experiments of Reid, Longet, Schiff, and others,[2] on the effects of section of the pneumogastric trunk or of its visceral branches, on the lungs and bronchi. All experiments concur in proving that these operations are followed by a very large accumulation of frothy mucus in the bronchi. Changes in the lungs have also been observed, which seem to be of the nature of congestion and collapse, but are imperfectly described. M. Longet has also found emphysema of the lungs, the relations of which to pulmonary collapse will hereafter be considered; and there can be little doubt that we have in these cases all the phenomena of bronchial obstruction and collapse following the division of the nerve which, according to the views above proposed, is the chief regulator or excitor of the bronchial de-obstruent function.

To sum up the results of this discussion, as respects bronchitis, I would recapitulate the following points, which, if not established, seem to be at least rendered highly probable. *Firstly*, That pulmonary collapse from bronchitis, when recent and uncomplicated, appears to be susceptible of cure, on removal of the bronchial obstructions. *Secondly*, That this is usually effected, not so much by the agency of respiration, as by the muscular contractions of the obstructed bronchi themselves. *Thirdly*, That the derangement or paralysis of this de-obstruent function becomes a cause of bronchial accumulation even in the normal state of the mucous membrane, and, *a fortiori*, in cases of bronchitis. *Fourthly*, That the de-obstruent function of the bronchial tubes may be impaired by various causes

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[1] Volkmann introduces into the trachea of a decapitated animal a tube having its outer end tapering, and perforated by a rather small opening. This being placed opposite a flame, he isolates and galvanises the vagus nerve, when, at every application of the stimulus, the flame is observed to be blown aside.—*Wagner's Handwörterbuch der Physiologie*, vol. ii., p. 586.

[2] Edin. Med. and Surg. Journal, April 1839; or Reid's Anatomical and Physiological Commentaries. Monthly Retrospect, 1849, p. 3. Longet—Systeme Nerveux.

acting on the pneumogastric nerve, either directly or through the nervous centres. And *Fifthly*, that it may be stimulated by remedies or other agents acting in a similar manner.

The application of these principles to pathology might be almost indefinitely expanded, if it were desirable at the present stage of the inquiry to indulge in much farther speculation. But enough has probably been brought before the reader to show that the symptoms, causes, and cure of bronchitis and other allied affections, even when not resulting in demonstrable or organic disease, are illustrated by a clear conception of the phenomena of pulmonary collapse and its attendant conditions.

I now proceed to the consideration of some permanent disorganisations, for the most part well known to anatomists.

Permanent Lesions of the Air-Vesicles and Bronchi depending on Bronchitis.

Relation of Bronchitic Collapse to Pulmonary Emphysema.—I have discussed above the mode in which the collapsed lung, under favourable circumstances, reverts to its natural condition. The mechanism by which this is effected, and particularly that portion of it which I have theoretically suggested under the name of the deobstruent function of the bronchial tubes, must be considered as of vast physiological interest, if we reflect that there is scarcely a case of fever, or any other debilitating disease, in which the signs of mucous accumulation and of partial pulmonary collapse may not be discovered at one period or other at the lower and back part of the lungs; and that the same forces which under these circumstances restore the lung to its normal state, by throwing off the load in the bronchi, are probably perpetually in action to prevent a similar accumulation in the state of health. A little careful reflection on the mode in which the free and unembarrassed play of the lungs is maintained in health, notwithstanding the constant presence of a viscid secretion from the bronchial mucous membrane, will probably satisfy every one that a special function for the removal of this secretion must form an element of the highest importance in normal as well as morbid respiration; and the ciliary apparatus, as I have already mentioned, is not calculated fully to perform this office, being less abundantly distributed to the smaller bronchial tubes, where its presence is apparently most required.

In many persons the removal of the bronchial mucus is habitually ill-performed. The quantity of mucus is not materially greater than natural, but it is not discharged as rapidly as it is secreted, owing to some defective condition of the deobstruent apparatus, or perhaps

a defective innervation of the bronchial muscles. In this condition, whatever be its cause, the tranquil and insensible processes of the normal economy are exchanged, sometimes for fits of coughing, which bring up pellets of tenacious pearly mucus, the result of undue accumulation; and sometimes for paroxysms of dyspnœa, which end in more copious evacuations of bronchial mucus, and a return to comparative health. These persons are the subjects of the *dry* or *humid asthma* of English authors, and of the *catarrh sec* of Laennec. Their disease, though simple and free from danger in its outset, is, according to the judgment of all physicians, apt to lay the foundation of organic disorder, which usually assumes the form of pulmonary emphysema. This consequence is peculiarly apt to occur, if care be not taken to guard against the supervention of bronchitis, which in these individuals generally assumes characters of great intensity, and is uncertain and protracted in its cure.

There are other persons who, with a habitually normal state of the respiratory functions, are subject to repeated acute bronchitic attacks of great severity. They are careless in their mode of life, frequently exposed to cold, or endowed with a peculiar sensitiveness of the pulmonary mucous membrane, while they are at the same time free from the tuberculous constitution, with its organic sequelæ. In such persons, also, pulmonary emphysema is known to be a frequent disease; being left behind as the legacy of the bronchitic attacks, and aggravated after each successive invasion.

Not unfrequently a considerable, or even an extreme, amount of pulmonary emphysema is observed to follow a single attack of acute disease in the chest. Thus, emphysema frequently arises in the earliest years of infancy and childhood, as the consequence of some form of severe infantile bronchitis; and all practitioners can bear witness to many cases in which shortness of breath and incapacity for exertion can be traced distinctly back to the date of an attack of hooping-cough or measles. Some of the most marked instances of emphysematous lungs in young subjects that have fallen under my notice in dissection, have had a similar history; and all authors on the diseases of children, who have carefully investigated the morbid anatomy and history of these affections, concur on this point. Again, in adults otherwise healthy, the severer forms of epidemic influenza are peculiarly apt to be attended with, or followed by, the development of emphysematous lesions; a fact which has been well observed and carefully recorded by Dr Peacock, in his excellent history of the last London epidemic of that disease.[1]

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[1] The Influenza or Epidemic Catarrhal Fever of 1847-8. By Thomas Bevill Peacock, M.D., &c.: London, 1848. See pp. 31-32, 134-135, 143-144, for graphic descriptions of the morbid appearances after death from influenza. Dr Peacock has favoured me with a letter on this subject since the publication of the first part of this memoir, and I am happy to be able to state, that this experienced pathologist—my predecessor in my present office—is convinced of the

Finally, a certain amount of emphysema of the lungs is of so frequent occurrence in the aged, as to be scarcely entitled to the name of a disease, distinct from the other evidences of corporeal decay. This fact was first pointed out by Magendie, and the form of emphysema here alluded to has since been described by many pathologists as a peculiar one, constituting a kind of senile atrophy of the pulmonary tissue. But there can be little doubt, that here also the pulmonary lesion is the concomitant of a bronchial affection,—the chronic bronchitis or bronchorrhœa,—which is almost constantly the companion of the more advanced periods of human life. In cases where this has been absent, I have repeatedly found the lungs of very aged individuals quite free from all trace of emphysematous lesion.

Considerations like these have, ever since the accurate descriptions of emphysema by Laennec became generally known, given rise to a general belief among practitioners that emphysema is related to bronchitis as effect to cause; and that it is indeed the organic lesion of the lung of all others most closely and invariably connected with long-continued or severe bronchial affections. In taking it, therefore, as the starting-point of the following researches on the permanent lesions of the lungs connected with bronchitis, I shall have the advantage, not only of beginning with a disorganisation so palpable and well-known as to be rarely overlooked at the present day by any one acquainted with pulmonary pathology, but one, the relation of which in some way or other to bronchitis, is almost universally admitted, notwithstanding the numerous differences of opinion as to its mechanism and causation. The observation of it somewhat more rigorously will serve, therefore, as a criterion of the correctness of the observations in the first part of this memoir, and at the same time will lead naturally to the consideration of other subjects.

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correctness of my explanations of the bronchitic collapse of the lung, and satisfied of the identity of that affection with many of those indicated by him in the pages to which I have referred.

TABLE OF FORTY CASES OF PULMONARY EMPHYSEMA, SHOWING ITS CONNECTION WITH OTHER AFFECTIONS, ESPECIALLY OF THE LUNGS.

Vol. and No. in Path. Reg.	Age.	Sex.	Amount, Position, and Extent of Emphysema.	Collateral Affections of Lungs.	Collateral Affections of other Organs.	Duration and Character of Symptoms.
XII. 10	37	F.	In L. L. sub-pleural bullae at apex, &c. In R. L. irregular emph. of inferior borders.	Condensation, non-granular at posterior portion of R.L. Lobular condensation. Cretaceous tubercle in apices.	None mentioned.	Cholera. No detailed history of symptoms. Died in reaction, with considerable dyspnœa.
XII. 25	31	F.	"Anterior part of both L. highly emphysematous."	"Posterior lobes much congested. Lower and back part of R. L. *somewhat hepatised* (?) and studded sparingly with miliary tubercles." Muco-purulent matter in bronchi.	None mentioned. "The abdominal organs healthy."	"Subject to want of breath from childhood." Dyspnœa, cough, lividity. Thick white sputa.
XII. 32	39	M.	The sub-pleural vesicles mostly solitary, — not larger than a barley-corn.	None mentioned. Upper lobe of R. L. compressed by a neuromatous tumour.	Neuroma of vagus nerve. Necrosis of scapula.	Frequent difficulty of respiration. Great exhaustion.
XII. 36	49	M.	Position and extent not mentioned. (The chest arched in front.)	L. L.—a cavity at apex, with irregular condensation. R. L.—miliary tubercles with condensation, as in L. L.	Cysts in kidneys.	Fever succeeded by bronchitis. No old history.
XII. 47	69	M.	Anterior edges of both lungs.	Chronic gray induration of upper and posterior parts of both L., with cavities and cicatrices of pleura (non-tubercular).	Hypertrophy of heart.	Six months' (?) cough, dyspnœa, &c.
XII. 67	22	M.	"Ant. edges very emphysematous." (Chest arched in front.)	Adhesions. L. L. hepatised in lower part of upper lobe. R. L., scattered lobular condensation in posterior part. Bronchi containing much mucus.	None mentioned.	Acute attack; 8 days. An *equestrian* in Batty's circus—intemperate, and exposed to cold.

XII	108	19	M.	"Exceedingly emphysematous at ant. edges."	Sulci between emphysematous portions. Gray condensation. Tubercle, with small cavities.	Extreme emaciation. Hypertrophy of liver and spleen. Tubercle of intestines.	Chronic phthisis; 4 years.
XII.	169	17	M.	Anterior portions. (Sternum arched.)	Tubercular condensation with cavities, scattered through both lungs, except a small part of anterior edges. Adhesions.	Tubercle of mesenteric glands, and slight ulceration of intestines.	No history. Symptoms acute.
XII.	220	—	M.	Whole anterior part of both lungs.	Some recent tubercle. Chronic and irregular condensation. Old puckering in apex of L.	Aneurism of aorta opening into œsophagus.	No information.
XII.	222	—	—	"Highly emphysematous at some points."	"Lobular condensation, alternating with crepitating and emphysematous lung. Outline very irregular, from sinking of the condensed, and prominence of emphysematous parts."	Softening of cerebellum.	No information.
XII.	272	—	—	Emphysematous at anterior edges; presenting irregularities of surface.	Lobular condensation "frequently in the midst of the emphysematous portions." Bronchial abscesses.	None mentioned.	Acute affection, succeeding measles. Fatal in 3 weeks.
XII.	274	38	F.	"Considerable emphysema of both lungs anteriorly."	Calcareous deposits scattered through both L., especially at apex. No condensation or tubercle. Adhesions.	Calculus in left kidney.	No information.
XII.	275	38	M.	"Both lungs very emphysematous anteriorly." (Chest considerably arched in front.)	"Slight condensation of both L. posteriorly." "R. L., a portion completely condensed. Bronchi full of muco-pus."	Hypertrophy of heart.	No information.
XII.	288	50	M.	"Air-vesicles much enlarged beneath the pleura at whole anterior part."	"L. posteriorly condensed, and scarcely crepitating." In the midst of the crepitating and emphysematous portions small lobular condensations. Adhesions.	Hypertrophy of heart.	Repeated attacks of chest symptoms for many years.
XII.	317	45	M.	"Ant. and lower edges excessively emphysematous."	Lobular condensation. Bronchi clogged with thick mucus. Soft lymph on pleura.	None of importance.	No information.

TABLE OF FORTY CASES OF PULMONARY EMPHYSEMA, SHOWING ITS CONNECTION WITH OTHER AFFECTIONS, ESPECIALLY OF THE LUNGS.—(*Continued.*)

Vol. and No. in Path. Reg.	Age	Sex.	Amount, Position, and Extent of Emphysema.	Collateral Affections of Lungs.	Collateral Affections of other Organs.	Duration and Character of Symptoms.
XII. 323.	60	F.	"Both lungs emphysematous anteriorly."	Bronchi contain much mucus. "Right side yields copious frothy serum. No condensation."	Hypertrophy of heart (right auricle).	No information.
XII. 326	—	—	Both lungs very emphysematous.	Irregular lobular condensations. Bronchi contained much mucus. Bronchial abscesses. Adhesion at apex of R. L.	Hypertrophy of heart.	No information.
XII. 327	35	M.	"L. emphys., particularly the anterior borders."	Lobular condensations; bronchial abscesses; cretaceous matter in bronchial glands.	Granular kidney. Necrosis of thigh.	No information.
XII. 345	45	M.	Both L. remarkably emphysematous. (Sternum very arched; sides of chest flattened.)	Hepatisation and collapse. Cicatrices at apex of R. L. Bronchi congested and thickened. Adhesions.		Cough *constant* for a year. Hæmoptysis. Emaciation.
XII. 349	36	M.	L. L. generally emphysematous.	L. L. containing miliary tubercle. R. L. contracted. Empyema, with contraction of right side of chest.	None mentioned.	3 months. Acute attack. Signs of empyema.
XII. 350	30	F.	Anterior edges partially emphysematous.	Between emphysematous portions, collapsed lobules; and a considerable amount of condensation post. Bronch. abscesses.	Ulceration of colon.	No information.
XII. 352	17	M.	R. L.,—intense emphysema of upper lobe; marked also in lower. L. L.,—much less emphysema, but in same situations as in R. L.	R. L.,—deep furrows between emph. portions, corresponding to dark atrophied parts. L. L.,—upper lobe much atrophied, and containing calcareous matter. Adhesions.	Hypertrophy of heart. Firm indurated deposits in spleen.	No information.

XII. 355	—	—	Great dilatation of air-cells (to size of a bean) in the anterior prolongation of upper lobe of right lung.	Collapse of upper lobe of R. L., with bronchial abscesses. Diffused collapse posteriorly, and lobular anteriorly, along with emphysema. Adhesions.	Slight hypertrophy of right side of heart. Stricture of urethra, &c.	No information.
XII. 360	7	M.	Interlobular emphysema confined to anterior prolongation of upper lobe of left lung.	R. L. healthy, except imperfect and partial collapse. L. L. contains a mass of tubercle in lower lobe, obstructing some of the bronchi. At the anterior edge, opposite this mass, lower lobe completely collapsed.	Tubercular lymph at base of brain.	Sub-acute hydrocephalus.
XII. 364	30	F.	Both lungs very emphysematous at anterior edges. Some air-cells dilated to size of walnuts.	Posterior portions of lungs completely collapsed. Emphysematous prolongation of upper lobe of L. L., connected with the rest by partially collapsed tissue.	Cancer of uterus, ovaries, and peritoneum.	No information.
XII. 390	16	M.	Both L. highly emphysematous at anter. borders, especially at the anterior part of lower lobes.	Condensation of posterior portions. Adhesions.	Contraction of mitral orifice. Slight hypertrophy of right side of heart, with granular degeneration of fibres.	No information.
XII. 400	50	F.	Lungs very emphysematous anteriorly.	Atrophied portions mingled with the emphysematous. Posterior parts but slightly crepitant.	Contraction of mitral orifice. Hypertrophy of heart. Disease of kidneys.	No information.
XIII. 1	40	M.	Anter. portions in both; in left, all the lobes anteriorly, and the lower and outer border of lower lobe.	Collapsed, intermixed with emphysematous portions. Firm concrete pus in some parts, forming small roundish masses of nearly cartilaginous consistence. Partial hepatisation. Adhesions.	Purpura spots on surface. The intestines healthy.	No information.

TABLE OF FORTY CASES OF PULMONARY EMPHYSEMA, SHOWING ITS CONNECTION WITH OTHER AFFECTIONS, ESPECIALLY OF THE LUNGS.—(*Continued.*)

Vol. and No. in Path. Reg.	Age.	Sex.	Amount, Position, and Extent of Emphysema.	Collateral Affections of Lungs.	Collateral Affections of other Organs.	Duration and Character of Symptons.
XIII. 9	—	F.	Highly emphysematous in anterior half.	Lobular collapse at edges, alternated with emphysematous portions. Posterior part of left lung crepitated imperfectly. Bronchi contained much muco-purulent matter. Adhesions.	Tubercle in bronchial glands and kidney.	No information.
XIII. 10	—	M.	In both lungs, but greatest in left ; in upper part, anterior and lower borders, and at the diaphragmatic surface.	Emphysematous parts had furrows corresponding to parts affected with lobular collapse. A part of the lower margin of R. L. crepitated very sparingly. Bronchi filled with pus, and dilated.	Bronchial glands enlarged and dark. An old sinus opening into right bronchus at the root of the lung. Old miliary tubercle of kidney.	No information.
XIII. 18	40	M.	"Highly emphysematous in their anterior half."	Lobular collapse and atrophy at edges. Posterior half of both L. slightly collapsed, but still crepitating. Bronchi contained much muco-purulent matter.	Commencing cirrhosis of liver. Very slight and doubtful hypertrophy of right ventricle of heart.	Bronchitis. Duration not stated.
XIII. 27	45	M.	Both lungs very emphysematous anteriorly.	Posteriorly, imperfect collapse, with œdema. Small points of extravasation.	Hypertrophy of heart (20 ozs., chiefly left). Bright's granulations of kidneys.	No information (Report of examination imperfect in many points.)
XIII. 30	45	M.	Lungs emphysematous over anterior third.	Atrophied portions in emphysematous edges. Collapse of posterior third. Mucus in bronchi.	Clot in left hemisphere of brain.	No information.

XIII. 34	40	F.	Lungs highly emphysematous over ant. third, and in lower border. Air-vesicles dilated to size of mustard seed.	Completely atrophic portions in midst of emphysematous. Mingled collapse and atrophy in post. two-thirds. Hard encysted nodules scattered through both L., containing much carbonaceous pigment.	None mentioned.	No information.
XIII. 49	68	M.	Both lungs extremely emphysematous anter. In upper lobes, bullae size of a hazel-nut.	Diffused and limited condensation, collapse, atrophy, induration, with encysted concretions in the midst of emphysematous parts. Bronchi loaded with mucopurulent matter.	Superficial fibrous degeneration of brain, &c.	No information.
XIII. 74	40	M.	Lungs emphysematous anteriorly.	Emphysematous edges present scattered collapsed lobules. Posterior—incomplete collapse of right, and hepatisation of left lung. Lymph on pleura.	Hemorrhagic ulcers of stomach, &c.	No information.
XIII. 81	22	M.	Lungs at anterior part pretty generally emphysematous.	Considerable collapse at posterior part, and scattered in midst of emphysematous portions. Tubercles—miliary, yellow, softened; small cavities, with puckering of pulmonary tissue (atrophy). Encysted masses, scarcely cretaceous.	Ulcers of intestines. Bright's disease of kidneys.	Symptoms acute; lasted 10 days.
XIII. 84	30	F.	Extensive emphysema at ant. edges. In left L. several pedunculated lobules, and greater dilatation than in right.	Collapse, diffused and scattered among emphysematous parts. Hemorrhage into bronchi.	Hypertrophy of right side of heart.	Pain in chest, cough, and expectoration, for 6 months.
XIII. 86	54	M.	Extensive and extreme emphysema; at upper part of left lung, one bulla, size of a pigeon's egg.	Much atrophy, with induration at apices, and scattered through emphysematous parts. Some concretions, cicatrices, a single small cavity (bronchial abscess) in left lung. Chest almost cubical in form.	Bright's granulations of kidney, &c. &c.	Symptoms of asthma of many years' standing.
XIII. 88	25	F.	Extensive and tolerably uniformly diffused emphysema of ant. edges. Nowhere great dilatation of air-vesicles.	Traces of atrophied lobules at anterior edges. Diminished crepitation of lungs behind.	Other organs not examined.	No history beyond the ultimate attack.

Analysis of Cases of Pulmonary Emphysema.—The preceding six pages contain a table of forty cases of emphysema of the lungs, the object of which is chiefly to show the connection of that lesion with other collateral affections of the pulmonary tissue. The state of the bronchi is not always noted, nor indeed can it be fairly assumed that, in a chronic lesion such as emphysema, the condition of the bronchial mucous membrane at the period of death has any direct relation to it in the majority of cases. The statements in the table accordingly show, not the evanescent and inappreciable conditions of the bronchial membrane, but the more permanent and evident affections of the air-vesicles and pulmonary tissue. The other columns are added for the satisfaction of those who may be studying the same subject under different aspects, and references are given in all the cases to the Registers of Dissections in the Royal Infirmary, where more detailed reports of them may be found. It is right also to state, that no cases of conside rable emphysema have been excluded from the table, excepting a few, in which the report was, from one cause or other, considered to be inadequate or untrustworthy.

The most cursory inspection of this table will show that pulmonary emphysema is in by far the greater number of instances accompanied by other lesions of the air-vesicles and pulmonary tissue; and that, in fact, its occurrence as an isolated affection of the lung, is not only uncommon but doubtful. In every instance it was found connected with some mode or form of condensation of the pulmonary tissue, except in the two cases marked XII. 32, and XII. 323. In one of these the report was not drawn up by me; and in neither of them do I now feel certain of its accuracy in this respect, as some of the lesions which I shall have to describe in the sequel as concurring with emphysema are easily overlooked, and have, in fact, been constantly overlooked by Laennec and other writers on this subject.[1]

The appearances in the other cases may be arranged as follows:—

Hepatization in four cases, or 10 *per cent.*, viz., XII. 67, 345; XIII. 1, 74.

Tubercle (or tubercle with condensation) in eight cases, or 20 *per cent.*, viz., *tubercle without excavation*, XII. 25, 220, 349, 360; *with excavation*, XII. 36, 108, 169; XIII. 81.

Condensation (presumably *bronchitic collapse*, and often described as such) in 27 cases, or 67·5 *per cent.*, viz., XII. 10, 25?, 67, 222, 272, 275, 288, 317, 326, 327, 345, 350, 355, 360, 364, 390; XIII. 1, 9, 10, 18, 27, 30, 34, 49, 74, 81, 84.

[1] In Louis's essay on Emphysema, in the Mem. de la Societé d'Observation, he describes at considerable length numerous cases of this lesion affecting the *entire* lung, without any concurrent affection,—a condition which, I do not hesitate to say, is not found in nature.

Bronchial abscesses, or non-tubercular ulcerations, in seven cases, or 17·5 *per cent.*, viz., XII. 47, 272, 326, 327, 350, 355; XIII. 86.

Chronic induration or atrophy, in ten cases, or 25 *per cent.*, viz., XII. 47, 108, 345, 352, 400; XIII. 30, 34, 49, 86, 88. Besides some of the tubercular cases, and the following:—

Contraction of opposite lung (absorbed pleuritic effusion) in one case, XII. 349.

Concretions in eight cases, or 20 *per cent.*, viz., XII. 10, 274, 352; XIII. 1, 34, 49, 81, 86.

Even a superficial inspection of this catalogue of morbid appearances will serve to corroborate many of the views previously expressed, and will conduct us to conclusions simplifying very much the whole subject. We find, in the first place, that 67·5 per cent. of the whole cases of emphysema were connected, at the time of death, with those forms of pulmonary condensation which have been ascertained in the preceding pages to be most frequently the result of bronchitis, and which, indeed, concur with it in the great majority of cases. The per-centage of bronchitic lesions, however, as we shall see in the sequel, will be increased from those found under other heads. Contrast with this the proportion of cases referred to hepatization or tubercle, two of the commonest of pulmonary lesions, the former of which is, nevertheless, found only in 10, the latter only in 20, per cent. of the emphysematous cases; and it will be at once evident, to what a large extent the bronchitic lesions predominate over all others. Nor is this apparent predominance merely the result of their greater absolute frequency; for I find, by my manuscript returns of the results of 502 post-mortem examinations of all kinds of disease performed in the hospital, during the periods referred to in the table, that, among these mixed cases, hepatization occurred 48 times, or 9·8 per cent., and tubercle of the lung 100 times, or 20 per cent.; while the other forms of condensation alluded to occupy a medium position in frequency, viz., 59 times, or 11·8 per cent., being only a little more frequent than hepatization, and very considerably less so than tubercle.[1]

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[1] As all the circumstances tending to affect the accuracy of these returns ought to be mentioned, so far as known to me, I may here indicate that I believe the frequency of these bronchitic lesions to be understated in both classes of cases, from the slighter forms being not always recognised or recorded, especially before I became quite familiar with their character and significance. These omissions would affect both classes of cases quite equally, and therefore be of little moment, were it not that the distinction between hepatization and collapse of the lung is not always sharply drawn in the earlier cases; and I think it possible that, especially in the larger series of returns, a few cases of the latter may have found their way under the head of the former. Even if we suppose, however, the per-centage of hepatization *slightly diminished*, and that of collapse of the lung *slightly increased*, in the series of mixed cases, the difference between the two affections in regard to the production of emphysema, will remain too broad and well-marked to be explained by any accident.

I am aware it will be said, that tubercle stands in a wholly peculiar relation to this subject, as several pathologists of great eminence have maintained the doctrine of the incompatibility of emphysema and tubercle; supposing, on the ground of their comparatively rare co-existence, that the former confers on those attacked an immunity from the latter affection. Rokitansky has, indeed, given the sanction of his high authority and immense experience to a doctrine which may be considered inclusive of this assertion of the French pathologists; viz., that all affections producing *venosity*, or imperfect oxygenation of the blood, such as cyanosis, curvature of the dorsal spine, emphysema, &c., confer an immunity from tubercle. Without entering here on the discussion of this doctrine, in its more general relations, it may be confidently stated, that the portion of it relating to emphysema gains no support from the numbers just quoted. According to this doctrine, it might reasonably be expected, that among persons dying with emphysematous lungs, tubercle would bear a decidedly lower proportion to the whole numbers than in a mixed hospital mortality; whereas, by a remarkable enough accident, it happens that the proportion is, in the above numbers, precisely the same, viz., 20 per cent. in both classes of cases.[1] And although it would be too much to argue from this coincidence, that emphysema and tubercle exert no influence upon each other, yet I think it may justly make us pause before accepting a doctrine which has not, *a priori*, much argument in its favour, and the evidence of which has never been presented to the public under a form approaching to exactness. I shall have occasion hereafter to state my own views on this subject.

The following table exhibits, in one view, the per-centage of most of the lesions referred to above, in emphysematous and in mixed cases of disease,—the numbers from which it is calculated being derived from the same hospital returns, so as to assimilate the conditions of observation as nearly as possible.

	In mixed Cases.	In Emphysematous Cases.
Hepatization,	9·8 per cent.	10·0 per cent.
Tubercle,	20·0 —	20·0 —
Condensation (collapse),	11·8 —	67·6 —
Bronchial abscesses,	5·5 —	17·5 —
Induration and atrophy,	7·5 —	25·0 —
Concretions,	4·1 —	20·0 —

It will be seen that while the first two lesions in the preceding table appear to have no special numerical relation whatever to

[1] Rokitansky admits the conjunction of obsolete or cretaceous tubercle with emphysema. But, in the cases above referred to, all the instances of obsolete tubercle have been excluded from both lists. In the cases conjoined with emphysema, it will be seen that there existed cavities in four cases; the others were miliary or yellow tubercle without excavation.

emphysema, their per-centage being nearly the same in this affection as in the general returns, the remaining four are found to be greatly more frequent in connection with emphysema than under other circumstances. But this is not all; for, as tubercle is almost invariably connected with some form of condensation, and was so connected in many of the cases here referred to, and as all the cases of hepatization are also to be found under the head of bronchitic condensation, it becomes nearly certain that, of the whole forty cases of emphysema, not one had any direct connection with either hepatization or tubercle, as such, but only through the medium of the other lesions mentioned. Tubercle and hepatization, therefore, are in all probability merely the accidents, and not either the causes or effects, of emphysema of the lungs.

If now we consider the all but invariable connection of emphysema with one or other of the remaining lesions of the lungs, and the frequency with which all of them occur in emphysematous as compared with mixed cases, we shall be driven almost inevitably to the conclusion, that some circumstance, common to them all, and not necessarily present in hepatization and tubercle, is closely connected with the production of emphysema, if not, indeed, its real pathological cause. What that circumstance is, we may now endeavour to discover.

Mechanism of Emphysema.—Emphysema of the lungs was said by Laennec, in one of the most original and accurate of his descriptions, to have two varieties: the one being a dilatation of the air-cells, and finally a rupture of them one into another by removal of their septa; the other, a rupture of the air-passages directly into the interlobular areolar tissue. It is needless to repeat these descriptions, the distinction of vesicular and interlobular emphysema being well known to every one, or at least accessible to all, in words which cannot be improved. It is only necessary to add, that the microscope and other modern means of investigation, which have done so much for morbid anatomy, have scarcely availed here to augment our knowledge; having only succeeded in demonstrating more clearly the fact, known to Laennec, of the gradual breaking up of the vesicular septa, and the obliteration of their capillary network.[1]

Emphysema, therefore, is an abnormal distension of the pulmonary tissue with air. In its earliest stages, whether interlobular or vesicular, or, as frequently happens, both combined, nothing can be more certain than that it is essentially a mechanical lesion: in fact, the distension of the air-cells, giving the peculiar cushion-like and

[1] After frequent personal observation on this subject, I am compelled to regard the late theory of Mr Rainey, in regard to the dependence of emphysema on fatty degeneration of the lung, as fallacious. The granules described by him certainly do not always occur in emphysematous parts; and when they do so, they are so few, and so little characteristic of this particular lesion, that it is plain Mr Rainey's views have been founded on an imperfect appreciation of the relations of the so-called "fatty granules" to morbid tissues.

pale appearance to the lung, can be exactly imitated by inflating it with undue force artificially. Moreover, the whole of the subsequent structural changes implied in the gradual removal of the septa and obliteration of the capillaries, are readily explained by the mechanical effects of distension. Upon this subject M. Poiseuille, to whom we owe so many interesting facts in mechanical physiology, has a very beautiful experiment.

An instrument being adapted to the pulmonary artery of an animal, by which a given quantity of liquid was propelled with a given force through the capillaries of the lung, he found that this was effected, in the normal condition, in 29 seconds. M. Poiseuille now inflated the lungs so as exactly to fill the cavity of the chest; the time was still 29 seconds. On distending the lungs, however, farther, so as to produce the appearance of a partial emphysema, the time required for the passage of the fluid became lengthened to 62 seconds; when the emphysematous appearance was increased, 95 seconds; when it pervaded the whole lung in consequence of excessive distension, 129 seconds were required, and the fluid returned from the pulmonary veins mixed with some bubbles of air.[1] From these results, it is evident that whenever the air-cells are distended beyond the amount required or possible in the healthy condition, the flow of blood through the ultimate capillaries of the lung must be retarded or obstructed;—a condition not only corresponding with the appearances observed in emphysema, but readily accounting for the structural changes, the absorption of the walls of the air-cells, and the tension and obliteration of vessels observed in the latter stages of the disease.

It is, therefore, nearly certain that the source of emphysema is to be sought in a derangement of the mechanism of respiration, and not in any previously morbid condition of the affected part. Every thing denotes that the emphysematous parts of a lung are usually free from all diseased changes, with the exception of those which are the result of inordinate distension. The freedom from œdema and from morbid deposits, when other parts of the lung are so affected; the absence of accumulation in the bronchi, or at least its comparatively slight character, allowing of the perfect and easy inflation of the emphysematous parts when others are collapsed; finally, the habitual seat of emphysema in those parts of the lung which are usually most exempt from other disease,—all tend to prove what I have now stated. The diminished elasticity, the dryness, the anæmia, which have all of them been supposed to be the predisposing cause of this lesion, are manifestly nothing more than the effects of the distension with air upon the circulation and nutrition of the compressed walls of the delicate pulmonary air-cells. Even the

[1] Bulletin de l'Academie Royale de Médecine, vol. viii., p. 705.

small accumulations of granular deposit found by Mr Rainey may be accounted for by these secondary nutritive changes.

But emphysema is not merely a lesion resulting from inordinate distension of previously sound portions of lung; it is, as we have already seen, the product of mechanical derangement in the *sound parts* of lungs *otherwise diseased*. The existence of bronchitic condensation, of induration, of concretions, &c., if not a necessary cause of the production of emphysema in the sound air-vesicles, is at least in some way related to it. The theory of emphysema by Laennec, besides the objections offered to it in the former part of this memoir, in no way accords with the facts now adduced. Mucous obstruction of the bronchi, even if proved to exist, cannot determine, *directly*, both condensation and rarefaction of the lung; and we have already learned, from unquestionable and multiplied evidence, which of these two is its real result. The opinion of Louis, derived, apparently, chiefly from a consideration of the seat of election of emphysema as compared with that of bronchitis,[1] is opposed to the idea of any precise relation between these two affections; but this negative opinion would appear to be sufficiently answered by the numerical facts above adduced.

Some writers, conceiving, like Laennec, that emphysema is produced in the act of expiration, believe it to be the result of violent efforts of coughing, or other forcible expiratory acts. But have we really any direct proof whatever that cough, however violent, or any similar act, can produce emphysema, apart from the other accidents of bronchitis? In croup, in laryngitis, in aneurism of the aorta, we have cough even more violent and distressing than that of bronchitis; yet these affections are not known usually to cause emphysema, and I have repeatedly seen cases opposed to the idea of their having any such influence. The alleged unusual frequency of emphysema among players of wind-instruments is likewise totally devoid of proof, and rests upon one unsupported assertion of Laennec; whereas, if the real cause of emphysema were such as above described, no singer or wind-instrument player could in all probability remain long exempt from this disease. But it would require further to be known whether an increased liability to emphysema in this class is not accompanied by a similar proclivity to other pulmonary affections, before the question could be decided on such grounds.

But the most serious objection to the expiration-theory of this disease is, that the expiratory act is *mechanically* incapable of producing distension of the lung, or of any part of it. The act of expiration tends entirely towards emptying the air-vesicles by the

[1] "Si l'on se rappelle que le *maximum* de l'emphysème ordinairement a son siège au bord tranchant des poumons et dans leur voisinage, tandis que le catarrhe pulmonaire aigu intense a le sien en arrière et en bas, on sera forcé de conclure que si ce catarrhe a une influence quelconque sur la développement de l'emphysème, cet influence est peu considerable et ne s'exerce sans doute que bien rarement."—*Memoires de la Societé Médicale d'Observation*, tome premier, p. 253.

uniform pressure of the external parietes of the thorax upon the whole pulmonary surface; and even when the air-vesicles are maintained at their maximum or normal state of fulness by a closed glottis, any further distension of them by the expiratory force is as much out of the question as would be the further distension of a bladder blown up and tied at the neck, by hydrostatic or equalised pressure applied to its entire external surface. The air-vesicles can sustain no distending pressure from the column of air *within* the tubes, as that air only becomes compressed in virtue of a force acting on the *exterior* of the lung, which opposes exactly as much resistance without as it creates pressure within. It is singular that a theory so radically unsound, and so devoid of direct proof, as this of the production of emphysema by expiration, should have been allowed to maintain a place in medical literature.

The only theories of emphysema which remain, are those which refer it to the act of inspiration. The most usual form assumed by these theories, is the supposition that emphysema of the lung is a physiological *compensation* for the occlusion of a diseased portion of lung;—a view not only giving no real explanation, but totally inconsistent with the fact, that in truly morbid emphysema there is always a diminished respiratory surface and consequent dyspnœa. Dr Williams, however, and some others, have placed the inspiration-theory in a more tenable position,—supposing that, when certain portions of the lung are occluded, the air is brought by inspiration to penetrate with greater force, and in greater volume, into the remaining parts.

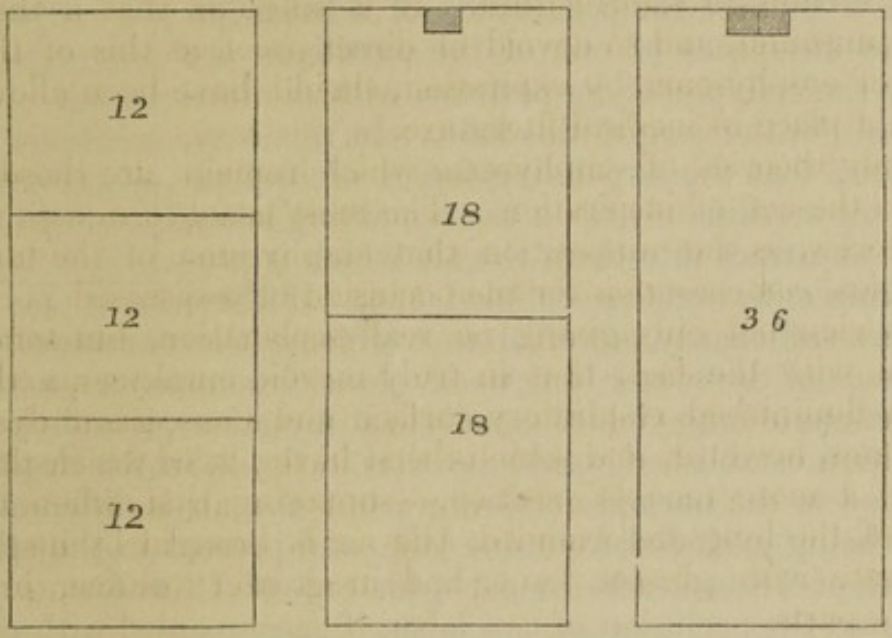
This view is certainly near the truth, and is quite consistent with clinical stethoscopic experience. But it is clogged in Dr Williams' work with a reference to the incompetent expiration-theory of Laennec, as if the author did not see his way clearly to the explanation of all cases of emphysema by his own. Moreover, it is not the whole truth; because certain obstructive lesions have, as we have seen, no appreciable influence in causing emphysema; and also because it is evident that the inspiratory or expansive power of the chest is exactly limited by its capacity, and that even when a portion of lung is impervious to air, as in hepatization, the inspiratory force can no more distend the sound air-cells to the degree observed in emphysema than it can do so in the normal state. This fact will appear more clear from the following observations.

It appears to me that none of the writers on this subject have clearly apprehended, or at least clearly expressed, the single obvious condition which is necessary to the mechanical completeness of the inspiration-theory of emphysema. Emphysema is, according to this theory, a *complementary* lesion, dependent upon the previous existence of some form of occlusion of the vesicles, and invading the remaining sound portions of lung. Thus far it corresponds with all that we have hitherto seen, to an extent certainly not anticipated by Dr Williams, when, after enunciating his own view, he brings

forward Laennec's theory to account for residual unexplained cases. But there is yet another condition necessary, besides mere occlusion of the air-vesicles in a part of the lung: this is *partially diminished bulk;*—in other words, collapse or permanent atrophy of a portion of the lung.

The operation and importance of this condition will be at once seen by the aid of a diagram. Suppose that in the accompanying fig. 1 the three equal partitions represent the *maximum* air-space, in

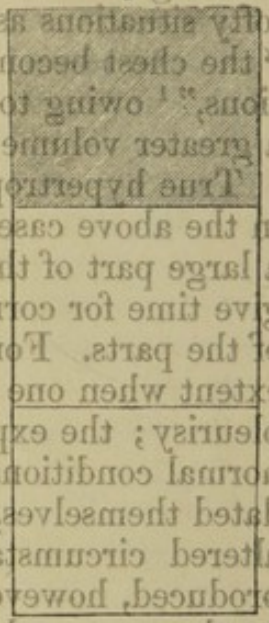
Fig. 1. Fig. 2. Fig. 3.



the normal condition of full inspiration of three lobes or portions of a lung (represented equal for the sake of simplicity). Each lobe holds, on a full inspiration, say 12 cubic inches or other measures of air; and it is adapted normally to hold this quantity, without pressure on the capillary circulation, or risk of violence to the texture of the organ. It is at once obvious that no amount of lesion, which leaves the upper partition or lobe of its normal volume, can at all affect the maximum expansion of the other two. They will continue, under all circumstances, to be capable of receiving their normal 12 measures of air; they will be prevented from receiving more, not by the tendency of the pulmonary texture to *resist* further expansion, but by the inadequacy of the mechanical apparatus *for producing* further expansion. No strain can in this case be thrown upon the walls of the air-cells; these still preserve their normal relation to the capacity of the chest which contains, and, by its dilatation, expands them. The inspirations will indeed be multiplied,—they will also be increased in fulness and force beyond the ordinary condition; but this can have no more effect in producing emphysema in the free air-cells of a diseased lung (under the above conditions) than running or violent exercise can have in relation to a healthy well-organised chest. This is the state which occurs in pneumonia, tubercle, and

all other lesions primarily affecting the air-cells themselves; it is represented in fig. 4, where the upper partition is supposed to be blocked up, at its full volume, with some abnormal deposit occupying the air-spaces. It may be granted that this diagram differs from what occurs in nature thus far, that even in the purest instances of hepatization the volume of the lung is seldom fully maintained; but it will be found, that exactly in proportion as it is so, the liability to emphysematous lesion is less.

Fig. 4.



But now suppose the occurrence of a lesion, in which the air-spaces of one of the partitions are closed by the collapse of its parietes, with diminution of bulk of the lung in this lobe. In this case, it is obvious that the expanding forces of inspiration will act inordinately upon the remaining lobes, and tend to attract into them the air which is prevented from entering the occluded one. If these forces were sufficiently powerful to overcome the resistance offered by the tissue of the sound lung under these circumstances, and if the sound portions of lung yielded equally in all directions, it is obvious that the condition established would be that in fig. 2, in which the lung is expanded to the normal maximum; but the air is differently distributed, being excluded from one lobe, and present in the others to the extent of 18 measures in each, instead of 12 as formerly. In like manner, the occlusion of two lobes, if accompanied with collapse of the tissue, would necessarily lead, in the event of the lung being fully dilated, to the accumulation of the whole 36 measures of air in the remaining lobe, as in fig. 3. A lobe thus distended would certainly suffer obstruction of the capillary circulation, as in the experiment before mentioned of M. Poiseuille; and the original purely mechanical condition would pass into one complicated by those structural changes which are actually produced in chronic emphysema.

It may be well to explain here, that a certain amount of overdistension, *when gradually effected*, is sometimes borne by the lung without the supervention of a distinctly morbid condition. The lung, under those circumstances, probably undergoes a genuine hypertrophy, the air-vesicles becoming slightly enlarged, but with a nutritive adaptation of the vascular and other structures to the changes thus effected. This enlargement of the lung, without the pathological characters of emphysema, is sometimes observed in disease, when the whole of one side of the chest has been contracted from pleurisy, the opposite lung passing, as the stethoscopist well knows, for an inch or two across the median plane in front, and having all its parts seemingly adapted to its increased size and function. A large power of adaptation of the lung to external circumstances is also shown (as has been pointed out by an acute

critic of my original communication on this subject to the Medico-Chirurgical Society of Edinburgh) "among the inhabitants of such lofty situations as the high table-land of South America," in whom "the chest becomes of a size considerably beyond its ordinary dimensions," [1] owing to the permanent and constant necessity for inspiring a greater volume of air than in less elevated situations.

True hypertrophy of the lung is most readily produced when, as in the above cases, the distending force acts equally on the whole or a large part of the pulmonary tissue, and when it is so gradual as to give time for corresponding changes in the nutrition and circulation of the parts. For this reason, emphysema seldom arises to a marked extent when one lung replaces the function of another destroyed by pleurisy; the expansion of the sound lung remaining limited by the normal conditions until all the structures have gradually accommodated themselves, under the influence of exercise and habit, to the altered circumstances of the system. That emphysema may be produced, however, to a certain extent in such instances, is shown by the case marked XII. 349, as well as in others of a similar kind which I have witnessed. The forms of pulmonary disease in which emphysema is most readily produced, on the other hand, are those where the primary lesions have been much disseminated, so that every part of the chest, in its expansion, acts at once directly upon corresponding portions of lung partially collapsed or atrophied, and yet containing many comparatively unobstructed lobules, which yield readily to the distending force. Hence the most frequent of all combinations with recent emphysema, as may be seen by reference to the table, is a certain extent of collapse of the posterior portions of the lungs, with a number of disseminated lobular condensations between the emphysematous parts. As these lesions are also very rapidly produced, and give rise to dyspnœa extremely urgent, they are apt to induce accelerated and laborious efforts at inspiration, in the midst of which emphysema, either of the interlobular or vesicular kind, or both combined, very readily arises.[2]

The theory here proposed has already been advanced by various writers, and with different degrees of precision of statement, to account for those cases of emphysema which are connected with the cicatrisation of tubercular cavities and other kinds of pulmonary atrophy. It is obvious, however, that its true significance, and the extent of its application, cannot be understood, till it is clearly apprehended that all cases of considerable obstruction in bronchitis bring with them, *as a necessary consequence*, a certain amount of diminished volume in the obstructed parts of the lung; and, therefore, that the connection of emphysema with bronchitis need present no difficulty

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[1] Med. Times, July 20, 1850, p. 72.

[2] The relation of emphysema to the violence of the inspiratory efforts, rather than to the apparent importance of the pulmonary lesion, is noticed by Rilliet and Barthez, "Maladies des Enfans," vol. i., p. 139.

to the pathologist, even when the latter affection has not been so violent or long-continued as to lead to any considerable amount of permanent and evident occlusion. That emphysema prevails in the opposite parts of the organ to those in which the direct effects of bronchitis are observed, becomes, in this point of view, one of the strongest evidences of its connection with that affection. That in the great majority of cases it is found in company with bronchitic collapse, or some lesion implying diminished size of the organ, amounts, I think, almost to demonstrative proof of the correctness of the theory here advanced.

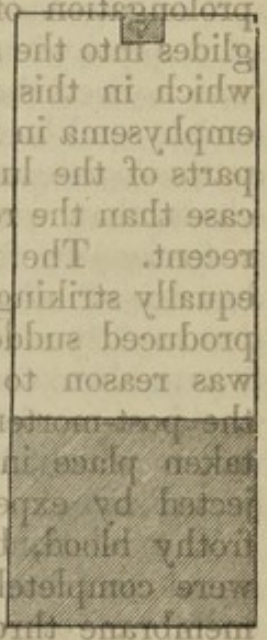
I am prepared, then, to maintain, that emphysema of the lung may, in all cases which I have witnessed, be satisfactorily accounted for by considering it as *a secondary mechanical lesion, dependent on some condition of the respiratory apparatus leading to partially diminished bulk of the pulmonary tissue, and consequently disturbing the balance of air in inspiration.* I therefore submit this principle to the judgment of the profession, in the confident anticipation, that it will prove no less constant and satisfactory in the hands of other observers, and will establish itself as the exclusive law of the production of this most important lesion.

A very few facts, in addition to the evidence already adduced, appear to be so striking as to deserve to be placed in an isolated form before the reader. One of these is found in Case IV., formerly narrated (p. 15), and also inserted in the table (XII. 360.) A child, in whom the right lung was normal, excepting imperfect bronchitic collapse, had in the left lung a mass of tubercular bronchial glands pressing on the bronchi passing to the anterior prolongation of the lower lobe, which was accordingly perfectly collapsed, void of air, and flaccid. The corresponding prolongation of the upper lobe, which in the act of inspiration glides into the same angle of the pleural cavity, and the bronchi of which in this case were free, presented very marked interlobular emphysema in its early and perfectly recent condition, and the other parts of the lung were normal. Nothing can be more clear in this case than the relation of the collapse to the emphysema, both being recent. The following case, which occurred to me lately, is an equally striking illustration of this point. An aneurism of the aorta produced sudden death, by bursting into the air-passages. There was reason to think, however, from the symptoms, as well as the post-mortem appearances, that bleeding to a less extent had taken place internally some time before death, without being rejected by expectoration. The bronchi on both sides contained frothy blood, but the *lower* bronchial branches of the left lung were completely stopped up with coagula of blood. The mucous membrane throughout the air-passages was quite healthy, though stained purple. The right lung appeared externally uniformly emphysematous, or at least distended with air throughout the upper and middle lobe, and less so in some parts of the lower lobe. The

surface was marked with purplish irregular mottlings, which could be seen to be quite beneath the pleura, and shining through from the substance of the lung (blood-stains, without condensation). Considerable portions of the lower lobe presented distinctly-marked lobular collapse. The left lung was also generally emphysematous in the upper two-thirds of the superior lobe. The inferior third was partially condensed and flaccid. The whole of the lower lobe was violet-coloured, completely condensed, and flaccid, having all the external characters of carnified lung. The whole sequence of the phenomena is here again most evident: the coagulated blood in the lower air-passages, especially of the left lung, producing obstruction and collapse, while in the upper part of both it had merely produced staining or mottling of the tissue, the bronchi being free, and the tissue generally emphysematous. In like manner, in emphysematous lungs having, as is usually the case, distinctly marked collapsed lobules or portions in the anterior edge, I have frequently been able to demonstrate the excess in the bronchi of the latter of muco-purulent matter; and in all cases the greater amount of obstruction may be demonstrated by the attempt to inflate the lungs, when the emphysematous portions will be found to yield at once, while the others follow slowly and often imperfectly.

Relation of Emphysema to Hepatization and Tubercle of the Lung.—It has already been shown, that no apparent numerical relation exists between emphysema and hepatization or tubercular deposit in the lung; the per-centage of cases of emphysema accompanied by these affections being nearly the same as in the general hospital dissections. These facts agree in all respects with the theory just stated, which shows that morbid deposits, affecting the ultimate tissue of the lung, can have no direct connection with the production of emphysema, unless they lead, in the first place, to diminution of bulk, or atrophy of the parts involved. This is not the case either with tubercle or hepatization in their recent condition, except when connected with bronchitis, in which case they may lead to the condition represented in the diagram, fig. 5. If a lobe of lung be, in the first place, completely hepatised, a subsequent attack of bronchitis may produce the collateral lesions of collapse and emphysema in the remaining lobes, the hepatized part remaining indifferent both to the one and the other tendency. On the other hand, bronchitis and its attendant phenomena may be succeeded by hepatization or tubercle. In either case they exert no direct influence upon the mechanical conditions under which respiration is accomplished.

Fig. 5.



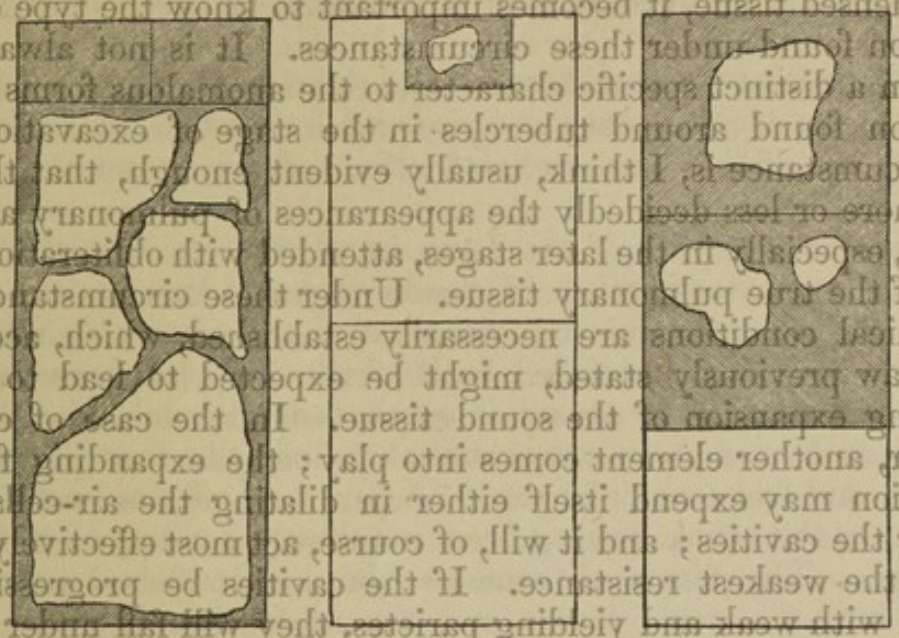
Relation of Emphysema to Excavations, and to Tubercular or Bronchial Abscesses.—The above remarks apply to tubercle in the

state simply of morbid deposit, before it has proceeded to excavation. The formation of cavities, however, in the lung, from whatever cause, is calculated to modify so considerably the mechanical conditions for the production of emphysema, that it is necessary to devote some consideration to this subject. As cavities are usually surrounded by condensed tissue, it becomes important to know the type of condensation found under these circumstances. It is not always easy to assign a distinct specific character to the anomalous forms of condensation found around tubercles in the stage of excavation; but one circumstance is, I think, usually evident enough, that they assume more or less decidedly the appearances of pulmonary atrophy, and are, especially in the later stages, attended with obliteration of all trace of the true pulmonary tissue. Under these circumstances, the mechanical conditions are necessarily established, which, according to the law previously stated, might be expected to lead to a compensating expansion of the sound tissue. In the case of cavities, however, another element comes into play; the expanding force of inspiration may expend itself either in dilating the air-cells, or in dilating the cavities; and it will, of course, act most effectively where it finds the weakest resistance. If the cavities be progressive, ill-defined, with weak and yielding parietes, they will fall under the influence of the inspiratory force, and be expanded to the full amount necessary to compensate the atrophy; and this result will follow the more easily, in proportion as they occupy, collectively, a larger proportion of the lung. On the other hand, if the cavities be healing, and surrounded, as they usually are in such circumstances, by very dense fibrous tissue; and if there be much sound pulmonary tissue, the cavities being few in number, and small, the expansion during inspiration will principally take effect upon the air-vesicles, and emphysema will be produced. These conclusions harmonise in all respects with the results of experience. Most pathological writers have noticed the frequent connection of emphysema with cicatrised cavities and healed tubercle; and even Rokitansky, whose opinions on the incompatibility of the two affections I have already noticed, admits that, with healing or healed (obsolete) tubercle, emphysema is frequently found. Is it too much to suppose that this whole doctrine of alleged incompatibility has been founded on the fact, that rapidly ulcerating and extending cavities, form a condition unfavourable to the development of emphysema; and that consequently, *in this stage*, they rarely co-exist? Such is, in truth, the whole extent to which, I believe, experience will be found to corroborate this hypothesis.

The relation of cavities to emphysema, then, may be shortly stated as follows:—1st, Large or numerous cavities, with flaccid walls, are, even when accompanied by atrophy—(see fig. 6.)—unfavourable to the development of emphysema; 2d, Cavities in process of cicatrization, if few or small in extent, and surrounded by firm atrophied walls—(see fig. 7.)—are extremely favourable to

the production of this lesion; 3d, Cavities, not surrounded by atrophied walls, whether large or small, exercise no particular influence in relation to pulmonary emphysema.—(Fig. 8.)

Fig. 6. Fig. 7. Fig. 8.



The great confusion which has hitherto existed on this subject, may be shown by reference to the best pathological works of the present day. The theory of Rokitansky, which derives one of its chief proofs from the relation of emphysema to tubercle, has been already noticed. Not to mention the absurd propositions made at various times to cure tubercular lungs by establishing emphysema, the following passage, from the careful and truthful work of Hasse, will show conclusively the correct apprehension he had of the facts, and the difficulty of finding a sufficiently general solution for them. "Those much in the habit of examining the dead body, cannot but be struck with two circumstances,—First, The almost invariable existence of emphysema in lungs, which bear the characteristic marks of recovery from phthisis;—and, Secondly, The *proportionate* rareness of tubercular deposits in emphysematous portions of lung. This would seem to show, that dilatation of the air-cells constitutes one of the conditions under which the cure of phthisis is possible, and again, that it forms *an obstacle to the development and progress of tubercle.*" Hasse then gives an accurate picture of the mode in which the cure of phthisis tends to the production of emphysema; and it is therefore clear, that, from the same series of facts, he makes two distinct inferences: one, that the cure of tubercle causes emphysema; the other, that emphysema causes the cure of tubercle. One of the two is, as I need not point out, obviously superfluous. In a subsequent paragraph he says:—"The different forms of active hyperemia are likewise subject *to the control of emphysema.*" Emphysematous portions of lung seldom become

affected with œdema. Pneumonia, for the most part, *leaves exempt such lobules as happen to be emphysematous.*" [1] There is here the same inverted inference as in the other case; but no hint of the correct solution of the phenomena. The principle that emphysema is always a secondary lesion, and always complementary of other states involving diminished bulk of the lung, at once solves all such difficulties.

There remains for consideration the relation of emphysema to the chronic forms of pulmonary atrophy and induration, including concretions, and of these lesions to affections of the bronchi.

Permanent Atrophy of the Lung in relation to Bronchitis. — In the preceding investigation of the causes and mechanism of pulmonary emphysema, the connection of that lesion with all causes leading to partially diminished bulk of the lung has been fully displayed. Among these causes of emphysema, bronchitic collapse of the lung and its sequelæ unquestionably hold the first place in importance and frequency; about 67.5 per cent. of the cases of emphysema in the table, having been manifestly connected with these affections. The forms of condensation here referred to may be divided into the more recent and the more chronic; the former being pure instances of bronchitic collapse, usually capable of easy removal by artificial insufflation of the dead lung, and therefore probably remediable in their character; the latter, on the other hand, having acquired more or less of a permanent type. The latter kind of condensation I distinguish by the name of *pulmonary atrophy*, a condition of the lung, the various forms of which are very imperfectly described by systematic writers, and by no one, so far as I am aware, except by Dr Stokes, ascribed to bronchitis.

The connection of atrophy with bronchitic collapse of the lung, can require but little explanation to the reader of this memoir. That the lung affected with collapse should after a time become altered in its structural relations, and be the subject of a permanent contraction or even obliteration of the air-vesicles, is no more than might have been apprehended from the knowledge of what takes place in the pulmonary tissue when subjected to long-continued pressure from pleuritic effusion. In such cases, it becomes, after a time, more or less impermeable to air, and incapable of its former expansion, even when the fluid has disappeared from the pleura, and all the mechanical conditions are favourable for its return to the normal state. Observation and experiment on the dead body also shew, that in those cases the proper tissue of the lung has in part disappeared, and that the air-vesicles which remain are incapable of assuming their original volume by any amount of expanding force.

[1] Hasse's Path. Anat. (Sydenham Society), pp. 313, 314.

In some instances, indeed, where compression has not been too long continued, we have good evidence that time will do much towards restoring the lung to its former condition; but in the majority of cases of chronic pleurisy, a permanently contracted side, with some degree of hypertrophy in the opposite lung, form the nearest approach to a perfect cure.

A precisely similar series of changes may be observed in the collapsed bronchitic lung, and has, indeed, been already alluded to in the first part of this memoir (Cases III. and IV., p. 15). In the dead body, the following gradations may be traced:—1. In the quite recent forms of the affection, the collapsed lobules yield before a force somewhat greater than that which inflates the sound portions of the lung; when inflated to the utmost, they are pale, emphysematous in appearance, and of volume equal to the surrounding parts; and when allowed again to subside to the ordinary condition of the dead lung, they are undistinguishable from the originally sound portions. This is the condition of simple collapse without atrophy. 2. The collapsed lobules cannot be inflated without the application of considerably greater force than in the former case; they then yield, however, and though perhaps not gaining altogether the full volume of sound lobules, are, on subsidence, not very easily distinguishable from them. 3. On insufflation of the lung, the collapsed lobules yield after very considerable resistance, but evidently not to the full extent; on allowing the lung to subside, they return more or less completely to the collapsed and non-crepitant condition. These phenomena are of course best observed in the well-defined lobular collapse which affects the anterior and lower edges of the lungs, as in Case IV., already alluded to. 4. The collapsed lobules cannot be inflated with air, except perhaps by a force sufficient to rupture the tissue; and then the air passes more readily into the interlobular spaces than into the obliterated air-vesicles. This is the condition of complete simple atrophy.

In *simple atrophy of the lung*, the result of uncomplicated bronchitic collapse, the affected parts usually present somewhat different characters from other forms of pulmonary atrophy. They are, in fact, reduced to a lax fibrous or areolar texture, inclosing the remains of bronchi and vessels; perfectly flaccid, free from all induration or abnormal exudation, and very frequently, in the purest form of the lesion, free even from that excessive deposit of carbonaceous pigment, which is so apt to accompany all chronic affections of the lung. Such atrophied lobules will almost invariably be found, on examining the free anterior or lower margins of old emphysematous lungs; and, in more recent specimens of emphysema, the anatomist will generally be able to trace several of the stages which I have indicated above, as intervening between collapse and atrophy. The atrophied lobules at the edge of the lung, correspond to the indentations and grooves between the emphysematous parts. On examin-

ing them closely, there will often be found a thin lamina, spread out between two emphysematous prominences, like the web of a frog's foot, and composed of the two pleural layers, enclosing the attenuated remains of the pulmonary tissue. Such portions are generally clearly and definitely marked off by the interlobular septa from the emphysematous lobules in their neighbourhood. In other instances, scarcely even this amount of tissue can be traced, and the two pleural layers may appear to be almost in contact over a small space, with little or no intervening substance. To any one who attentively studies a variety of such specimens, it will be apparent that simple atrophy of the lung, in its most complete form, is a lesion only to be distinguished by negative characters. The proper and special elements of the pulmonary tissue have disappeared; but they are not replaced (as in atrophy from other causes) by any adventitious structure, or even by the thickening or induration of the fibrous basis. For this reason, simple atrophy is sure to be overlooked, unless its traces be sought for in the manner I have described. In the centre of a lung, very many lobules may be entirely atrophied, and leave no visible or tangible evidence of their previous existence.

Simple atrophy, like the lesion which gives rise to it, occurs in the lobular and the diffused form. The latter is chiefly found in the posterior portions of the lungs, near their root, among the great bronchi and the bronchial glands, which are often, in these cases, dark coloured from infiltrated carbon, even when the lungs are by no means remarkably so. In diffused simple atrophy, the lung is rarely entirely condensed, generally retaining a certain degree of crepitation, but being dense, tough, and fibrous; sometimes dark slate-coloured, at other times not so; and in the most marked and exaggerated examples, crossed in every direction by fibrous processes, or septa of considerable thickness and density, corresponding to numerous depressions and irregularities on the surface of the lung, which is usually in these cases very emphysematous in front, and over the surface generally. Such lungs will always be found, when a fresh section is inspected with or without a lens, to present the most remarkable varieties in the size of the vesicles; some of which are entirely obliterated, or very small, and others greatly expanded beyond the normal volume; the latter condition prevailing, of course, towards the anterior margins in the most emphysematous parts.

The changes impressed upon the form and movements of the chest, by the chronic sequelæ of bronchitis, form so marked an illustration of the doctrines above recorded concerning the supervention of atrophy on bronchitic collapse, that some reference to them here is quite necessary to the complete treatment of this subject. I have already alluded in the first part of this memoir to the modifications of respiratory movement which take place in acute bronchitis in children, while the bones and cartilages are, as yet, inadequate to the task imposed on them of expanding the chest under conditions of in-

creased resistance. Under such circumstances, it is not very unusual to find the movements of the lateral regions of the chest actually reversed, the parietes being, as it were, sucked inwards at each descent of the diaphragm, owing to the external atmospheric pressure overcoming their power of expansion. This yielding of the ribs I have indicated as probably one of the causes of the extremely frequent occurrence and great extension of bronchitic collapse in very young subjects. In rickety individuals, it is not only more marked, but apt to become permanent, especially when such subjects are affected with any considerable or persistent bronchitic affection. In such cases, the reversed movement of the ribs is stereotyped, as it were, in the form of chest called *pigeon-breast*, in which the sternum is protruded, particularly below, and the whole lateral region, including also the lower costal cartilages in front, flattened, or even at some points rendered irregularly concave.

Many slighter and more partial permanent irregularities in the form of the chest are no doubt owing to infantile bronchitic attacks, either modifying the original expansion of the lung, or producing subsequent partial collapse of its tissue. The immense frequency of such diseases in childhood, and the unquestionable tendency which they are now shown to have towards structural changes, will probably go far to account for many of those disorganizations in the lung, revealed by morbid anatomy in a large proportion of cases, and which often seem to have no connection with any thing in the history of the individual. To a similar source may, in all probability, be justly traced most of the so-called "physiological heteromorphisms" of the chest, described and investigated with such elaborate minuteness by M. Woillez.[1] According to this writer, these slight and trivial deviations of the apparently healthy chest, occupy, for the most part, the same situations as those which are known as the results of disease; and, indeed, it would appear that the "physiological" and "pathological" irregularities are by no means separated by a very distinct line of demarcation. It is quite true that, in many of the individuals presenting these changes, no history of chest-disease can be procured; but every one accustomed to the task knows that the elimination of information in regard to diseases of early life, is, in the cases of most hospital patients, nearly impossible, even where the disease has been of considerable importance. In so far, therefore, as these "physiological" irregularities are worthy of consideration at all,[2] I cannot but

[1] Recherches Pratiques sur l'Inspection et la Mensuration de la Poitrine. Paris, 1838.

[2] This qualification is not unimportant, seeing that M. Woillez, by means of rather ponderous statistical machinery, has arrived at the singular conclusion, that only 1 in 3 of the *healthy* chests, and about 1 in 5 of *all* the chests examined by him, present a strictly *regular* conformation. It is obvious that, with a few more refinements such as those to which this observer has devoted so much labour, the ideal of regularity would require to be sought altogether beyond the

think that infantile bronchitis may probably have a large share in their production.

In adults, the motions of the chest are altered to a considerable extent in bronchitic affections, though not nearly to the same degree as in children. The greater solidity and firmness of the bones and cartilages opposes an effective resistance to that abrupt and well-marked retraction of portions of the thoracic wall which has been noticed as occurring in infantile bronchitis; the chest expands more uniformly and forcibly over its whole surface, and the phenomenon which gives rise to the pigeon-breast is not observed when the bones are healthy. That the respiratory motion may be seriously limited, however, and sometimes even reversed, at certain points in the adult chest during bronchitis, is demonstrated by the observations of Dr Sibson, made with the aid of his ingenious and useful instrument, the chest-measurer.[1] These observations I regret that I have not yet been able to repeat, but of their general accuracy I can entertain no doubt. It is evident, indeed, to the eye (which, when employed with the requisite care, is in this case a far less deceptive, and more instructive instrument, than the ordinary measuring tape) that, while even in the severer forms of bronchitis, the chest on the whole expands both in its upper and lower zones, the movement of the latter is much more restricted than that of the former; and that while the *lateral* expansion of the thorax is circumscribed, the *anterior* movement of projection of the sternum and costal cartilages is usually even exaggerated. The result of this curious modification of respiration is, that in cases of long-continued chronic bronchitis, even during the intermissions of accumulation in the air-tubes, an altered habit of breathing is acquired and permanently retained; and the stethoscope, as well as the inspection of the chest, can often determine in such cases that respiration is effected chiefly by the upper and anterior portions of the lung, and by the movements of elevation and projection of the sternum; while the parts of the lung corresponding to the lateral and posterior regions of the chest, remain comparatively little affected by the respiratory act. The modification in the permanent form of the chest which supervenes upon this condition, is tolerably well-known as the "cylindrical" or "emphysematous" chest; it is marked by increased fulness and prominence of the whole anterior thoracic vault; often also, but not invariably, by in-

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pale of humanity. It appears very doubtful whether even the Apollo or the Antinous could withstand the search for "physiological heteromorphisms" by M. Woillez. At all events, artists and anatomists are well aware that, among the poor sons of Adam, strict symmetry and regularity in every point of form, is an occurrence of almost fabulous rarity. The very general lateral curvature of the dorsal spine, and the all but invariable lateral deviation of the nose, are glaring instances known to every one. How often do the phrenologists find a *regular* head?—or would any two of them agree upon the subject?

[1] Medico-Chirurgical Transactions, Vol. xxxi.

creased arching of the sternum from above downwards; and perhaps yet more characteristically by a diminution in the lateral, and a relative increase of the antero-posterior, diameter of the thorax.

The true relation of these changes to the existence of collapse posteriorly, and emphysema anteriorly, in the lungs, is not altogether so clear as it may at first sight appear. That the permanent modification of form is the consequence of the peculiarly altered movement of the chest which I have described above, will admit of little doubt to those who have witnessed this movement in characteristic cases of acute and chronic bronchitis. It may also be freely admitted that the diminished lateral motion is the direct effect of the diminished expansion of the lung in consequence of bronchitic accumulation, with partial collapse, and perhaps subsequent atrophy of its tissue. But to ascribe the increased movement and consequent deformity of the anterior part of the chest to the production of emphysema, appears to me an error both of logic and of observation. I believe, on the contrary, that whatever be the relation of emphysema of the lung to the "emphysematous chest," *it is not directly or indirectly the cause of that deformity*. And this conclusion appears to be borne out by the following considerations :—

In the first place, the increased respiratory movement in the anterior part of the chest, which appears in all cases to be connected with the generation of emphysema, as well as of the deformity above-mentioned, exists in a large number of instances of bronchitis, before either the one or the other condition has yet arisen; indeed, in its slighter degrees, I believe the increased anterior thoracic movement to be an almost constant concomitant of that affection. In the second place, the existence of very well-marked emphysema, though unquestionably concurring with the highest degrees of the deformity, has always appeared to me to tend to diminution of the abnormal excess of motion; this excess being always detected most characteristically in company with simply puerile, not emphysematous, respiration. It appears, therefore, impossible that the generation of emphysema can be the cause of that exaggerated motion. Lastly, according to arguments and observations already laid before the reader, it appears that emphysema is a lesion directly due to the forcible expansion of the chest under peculiar circumstances, which seems fairly to exclude the opposite proposition, that undue permanent expansion of the chest can ever be owing to the existence of emphysema.

From observations on this subject, it appears to me susceptible of demonstration,—that the abnormal motion of the chest, in the cases above alluded to, always precedes both the deformity and the emphysema; that the emphysema frequently precedes the deformity, but in its more chronic and exaggerated forms generally follows in its wake; that a certain amount of emphysema may exist without deformity, and a certain amount of deformity without marked emphysema; and that, in any given case, when emphysema

supervenes on exaggerated anterior movement, with or without deformity, its natural effect is to diminish that excessive movement. This last proposition corresponds with the state of the lung in extreme emphysema, in which the emptying of the air-vesicles is effected with great difficulty, or even may be absolutely impossible, owing to the existence of an apparently valvular obstruction to the egress of air; a condition which suggested to Laennec his theory of emphysema, but which I believe to be a secondary effect, and not a cause of that structural alteration.

Were I to hazard a speculation as to the mutual connection of this complicated series of phenomena, it would be that indicated in the following propositions, which I submit to the reader, not as ascertained truth, but simply as being the most probable conclusions at present attainable in relation to this subject:—1. The direct tendency of bronchitis is to produce bronchial accumulation, and thereby to restrain the expansion, or even to produce retraction, of the whole lung, and consequently of the chest. 2. To overcome this tendency, forced respiration is at once thrown into action, and the breathing, from being, as in the normal state, mostly diaphragmatic, becomes in a high degree costal and thoracic. 3. In overcoming resistance, by means of costal superadded to diaphragmatic respiration, those parts of the chest, whose movements are performed by the most powerful muscles, acting at the greatest mechanical advantage, tend to assume the principal function, while the remaining portions fall into abeyance, or yield in part to the opposing resistance. 4. On this principle the elevation of the sternum, and of the anterior ends of the true ribs, which is effected by the powerful aid of the cervical muscles in addition to the intercostals, becomes the predominating movement along with the descent of the diaphragm; while the motions of the posterior and lateral parietes of the chest, which are maintained, in the normal state, by a much weaker force, tend to fall into abeyance. 5. The respiratory forces, instead of acting equally on all parts of the pulmonary surface, and tending to expand it from all points at once, are thus spent in greater measure upon the anterior edge and upper part of the lung, which are in contact with the most mobile parts of the thorax, as well as upon the lower edges and diaphragmatic surface; and these parts, therefore, become the principal seats of respiratory movement, while the root of the lung and its lateral and posterior surfaces only receive the inspiratory impulse secondarily, or in greatly diminished ratio. 6. The consequence of the inferior power of movement in the posterior and lateral parts of the lung, is accumulation and stagnation of mucus in the tubes; thence a greater liability to pulmonary collapse and atrophy as the consequence of bronchitis. 7. The consequences of the superior power and greater extent of movement at the edges and upper parts of the lung, and on the diaphragmatic surface, are comparative freedom from mucous accumulation, and consequently from pulmonary collapse and its consequences, and on

the other hand, much greater tendency to the development of emphysema from violent and repeated forced inspiration, when partial collapse or atrophy is present elsewhere. 8. The irregularities of movement of the thorax tend ultimately to affect its form, producing in the child the *pigeon-breast*, by lateral flattening of the yet flexible and soft ribs, with depression of the lower costal cartilages, and protusion of the sternum; in the adult or older child, slighter lateral flattening, with expansion or bulging of the cartilages, and arched protrusion of the sternum; and in both the child and the adult, increase of the antero-posterior diameter relatively to the lateral, and of the upper zone relatively to the lower. 9. The deformity of the chest usually accompanying emphysema of the lungs is neither a cause nor an effect of that lesion, but both emphysema and the "emphysematous chest" depend on the altered respiratory movements in bronchitis, and the exaggerated respiration necessary to overcome the tendency to bronchitic collapse of the lung.

It may appear to some readers that the above explanation of the seat of election of pulmonary collapse and emphysema is superfluous, and that the gravitation of the mucous obstructions in bronchitis to the posterior portions of the organ, is a sufficient reason for the occurrence of collapse in that situation, and of emphysema in the opposite region. To this opinion, however, some facts stand in direct opposition. The most important is that in the horse, in which emphysema and the other diseases of the lung are common, and in which the position of the lung as respects the effect of gravitation is precisely the reverse of what occurs in man, the seats of election of emphysema and of pulmonary condensation are nevertheless nearly as in the human subject. In various experiments on the rabbit, also, I have noticed the same tendency of emphysema to the borders of the lung, and of collapse to its root, although the animals were allowed to maintain the natural position, in which the force of gravitation ought to have had an opposite tendency. For these reasons, I have been induced to ascribe very much less effect than most observers to the simple [illegible]atical condition of the fluids in pulmonary diseases, and to look for some dynamical cause which would explain the position of the lesions found in bronchitis, pneumonia, and emphysema, in a more satisfactory manner than hitherto. To what extent the preceding paragraph is a successful attempt at such an explanation, must be left to the judgment of the reader, and to the future observation of facts bearing on the subject.

Pathological alterations of the Bronchi in Pulmonary Atrophy and in Emphysema.—The memoir of M. Reynaud[1] on obliteration of the bronchii, has been referred to by most subsequent writers as having enumerated and described with great completeness all the more

[1] Mémoires de l'Académie de Médecine. Tome iv.

ordinary varieties of permanent contraction or dilatation of the air-passages. Indeed it is difficult to conceive anything more completely exhaustive than this memoir, when considered purely in an anatomical point of view, and solely with reference to the air-passages; and having frequently had opportunities of verifying nearly all of his observations, I find it, like most others who have referred to them, not easy to state anything novel upon this subject. But M. Reynaud's researches, though full of anatomical truths, are strangely barren, at least in his own hands, of real pathological interest; which arises chiefly from his having too exclusively pursued the inquiry relatively to the bronchi themselves, and not having sought to connect their alterations with those of the pulmonary tissue, with which they are, according to my experience as well as that of others, constantly and indissolubly associated. Somewhat of the same objection applies to Laennec's observations on dilatation of the bronchi, which first gave to this disease a place in pathological anatomy. Accordingly it has been reserved for future observers to discover, that both the dilatation and the contraction of the bronchi are almost always secondary lesions, or at least invariably connected with some kind of disorganization of the pulmonary air-cells. Several of the later pathologists have adopted these views, with more or less decision; among whom may be mentioned Hasse, Rokitansky, Stokes, &c.; but the ideas of Laennec and of Reynaud have still been adopted by many writers on the diseases of the chest, with perhaps too little discrimination, and very little real advance has been made in the pathology of these affections.

The forms of obliteration and contraction described by Reynaud are numerous. It is scarcely necessary to enter into the consideration of the anatomical varieties. The different kinds of dilatation, as described by Laennec and Reynaud, and their relations to obliteration of other parts of the same bronchial divisions, are of more interest. The most frequent are the following:—A small bronchus of normal caliber suddenly opens out into a sacculated dilatation, lined by smooth thin membrane, and of more or less rounded form. This dilatation sometimes terminates all trace of the bronchus; in other specimens, the contracted and obliterated remains of bronchii pass from its opposite end towards the circumference of the lung. Again, a bronchus may show a succession of marked irregular dilatations through its whole length, at some parts having the sacculated character, at others being irregularly cylindrical, with partial annular projections, and transverse septa arising from the walls of the dilated tube. Finally, the whole of a lung or of a lobe may be broken up into a series of cavities, having free communication with each other, and with the main bronchi of which they are presumed to be dilatations.

On all the changes here described, there is one important remark to be made—they are invariably found in close connection with atrophied lung, either of that kind which results, as we have seen,

from bronchitic collapse, or some of the more complex varieties which proceed from other lesions, such as tubercle or chronic hepatization. With regard to obliteration or contraction of a bronchus, it is indeed self-evident that this must be the case; and the reader of the foregoing parts of this memoir will see without difficulty by what steps the obstruction of the air-tubes leads to their contraction, along with the collapse and gradual atrophy of the vesicles to which they lead. In the case of dilated tubes, it is an observation of all the later pathologists, in which I fully concur, that the pulmonary tissue around these dilatations is usually impermeable, and in a condition of fibrous atrophy, most commonly without marked induration. It was this circumstance which gave rise to the theory of bronchial dilatation by Dr Corrigan, which led him to call this disease "cirrhosis of the lung,"[1] conceiving that the formation of a peculiar contractile fibrous tissue in the interstices of the bronchi, and the obliteration of the air-cells, led to the expansion of the tubes, by the gradual operation of the inspiratory forces, and constituted the true pathological condition of the disease. Although I have little doubt that Dr Corrigan's theory is fallacious, in so far as regards the existence of any *new* or *peculiar* fibrous element in this affection, yet there is no doubt that his observations were in other points correct, and that in particular he has the merit of being the first to draw attention in a decided manner to the morbid alteration and obliteration of the air-cells, a fact singularly enough nearly overlooked by Laennec.

What, then, is the origin of bronchial dilatation? The explanations of Laennec and others, which ascribe it to violent coughing, to distension by accumulated mucus, &c., are clearly unsatisfactory, on precisely the same grounds as have been already indicated in the case of emphysema, and which there is therefore no occasion again to repeat here. As in the case of emphysema of the vesicles, it seems more consonant with reason to ascribe these dilatations (as is done by Dr Corrigan) to the expansive forces of inspiration acting upon the bronchi of atrophied lung. But it is difficult to understand, on this principle, the occasional partial character of the lesion—the expansion of one portion of a bronchial tube into a sacculated globular enlargement, while adjoining tubes and adjoining portions of the one affected, retain their natural size. In such cases it becomes necessary to suppose the existence of some more local affection, rendering the bronchial tube dilatable at the point in question.

To those who have studied this subject only in the light of Laennec's description, the following remarks will probably appear too bold and sweeping a generalization. They are, nevertheless, the result of much consideration, both of the descriptions of authors,

[1] Dublin Journal. May 1838.

and the facts observed in very numerous dissections, as well as in most of the public pathological collections of this country. The conclusion to which I have been led by this survey is, that almost all the so-called bronchial dilatations, and all of those presenting the abrupt sacculated character here alluded to, are in fact the result of *ulcerative excavations* of the lung communicating with the bronchi. That such ulcerations are not uncommon in bronchitis, especially in the case of children, has been already sufficiently indicated in the first part of this memoir, under the head of bronchial abscess. I have, in fact, incised the bronchi with great care in cases of recent bronchitis with bronchial abscess, and have found the small cavities so described to occupy precisely the same relations to the caliber of the tubes as the larger dilatations which are found in connection with chronic atrophy. The expansion of these small cavities, either by increase of ulceration, or by the act of inspiration, would clearly in these cases have led to an appearance closely resembling the so-called dilatations of the bronchi in everything except the fine, smooth, and consistent lining. On the other hand, the examination of a very great number of unquestionable instances of chronic pulmonary excavation from tubercle and other causes, has satisfied me that cavities originally formed by ulceration, may become lined by membranes exactly resembling those found in the "dilated bronchi" of Laennec. In no instance that I have seen, has this membrane exactly resembled the mucous membrane of a bronchus; even in the cases of so-called true bronchial dilatation, it is thin, dense, very smooth and glistening, and with comparatively few vessels; in fact, more resembling a serous than a mucous tissue. This description altogether concurs with those of Hasse [1] and others as applied to bronchial dilatation. It is, I think, almost conclusive upon this question, that in chronic cavities, evidently of tubercular origin, I have been able to trace quite satisfactorily the gradual assimilation of their lining to this type; and in several instances to observe cavities, which, but for the existence of others in the same lung in a different condition, would scarcely have been distinguishable from the so-called bronchial dilatations; they being lined by membrane perfectly smooth and glistening, and gradually passing into that of the undilated portion of the tubes leading into them. I have also observed that in such cases there is even the formation of an incipient epithelium upon this new membrane; or, at least, of numerous cells which, under the microscope, sufficiently resemble the columnar epithelium of various parts of the air-passages, differing, however, from that of others, and especially of the trachea and larger bronchi, in being of inferior size, and never, so far as I have observed, furnished with cilia. The opportunities of making such observations under the requisite conditions to ensure accuracy,

[1] Pathological Anatomy—article, Bronchiectasis.

are too rare to permit of my entering into this subject at greater length.

If these observations are admitted as bearing on this question, it will, I think, become probable that the usual origin of bronchial dilatations is in cavities formed in atrophied lung, in consequence of bronchitis or tubercle, and afterwards expanded beyond their original dimensions by the inspiratory force. The conditions that conduce to such expansion have been already considered under the head of emphysema, and its relation to excavations. It will be at once evident that the tendency to expansion of a cavity must be great in proportion to the flaccidity of its walls, and the absence of crepitant lung in its neighbourhood. It is owing to this circumstance, that bronchial dilatation and emphysema of the lungs have been found, to a certain extent, mutually exclusive. The cases most fitted for the development of such permanent excavations, are those in which the whole of one lung has been converted into a series of cavities, with no intervening crepitant tissue. Of this I have seen several examples. One such instance, figured by Cruveilhier,[1] forms an admirable illustration of Corrigan's cirrhosis, and of the real mode of its origin, viz., by ulceration. The whole upper lobe of the lung is converted into the condition of chronic cavities, lined by smooth membrane, and communicating freely with each other; while in the lower lobe are found recent excavations; and every intermediate condition between the two varieties can be readily traced. Of this case I have on one occasion seen an almost exact counterpart, in a boy, aged about 12 years, affected for a long time with hepatic and pulmonary disease, under the care of Dr Renton.

Pulmonary Concretions and Cicatrices.—The existence of cicatrices and puckerings in the pulmonary tissue, sometimes accompanied by distinct induration, with much thickening of the pleura in the neighbourhood, and sometimes by rounded whitish masses of atheromatous, chalky, or stony consistence, imbedded in the tissue of the lung, and surrounded by a fibrous cyst, has been long known to morbid anatomists, although more attentively studied of late years. Morgagni, summing up his own experience with that of his predecessors, signalised their existence in connection with asthma and other symptoms of disease of the respiratory organs.[2] Portal considered calculous concretions of so much importance, that he indicated, by means of them, a particular species of phthisis, the "phthisie calculeuse." He maintained the entire dissimilarity between the calculi of gouty and those of scrofulous origin, showing

[1] Anatomie Pathologique, Livraison, 32. Pl. 5. Fig. 3. The case was considered to be of tuberculous origin, yet "the cicatrization (of the excavations) was perfect; the parietes presented the appearance of accidental mucous membranes."

[2] De Sed. et Causis Morb. Epist. xv. 17, ad finem.

their frequent connection with phthisical symptoms, cough, hemoptysis, dyspnœa, &c.,[1] which, however, he did not consider as exclusive of the gouty diathesis, inasmuch as he has a "phthisie arthritique et rhumatismale." Portal also considered these calculi as formed in some instances by inspissation of the bronchial humours, as well as by dust inhaled from without, — an opinion which has received some recent confirmation in the special case of the stone-hewer's phthisis, but which, as a general deduction, is contradicted by the chemical nature of these bodies; the pulmonary calculi being now known to consist in a great part of phosphate of lime, and other calcareous and magnesian salts, evidently of organic origin. Laennec was the first distinctly to protest against the opinion that these pulmonary concretions were necessarily attended by symptoms; having, as he says, frequently found them "in subjects who had presented no sign of oppression or embarrassment in the respiratory organs." The description by Laennec of these bodies bears the marks of a very attentive observation of all their pathological relations. He notices their frequent occurrence with or without the accompaniment of other lesions, and remarks that they are occasionally found in the centre of tubercles, and very frequently along with pulmonary cicatrices, like those which are found in tuberculous individuals; from which circumstance he concludes, that, "in the greater number of instances, they are the result of a cured tubercular affection." He does not, however, deny that osseous and cretaceous concretions may be developed independently of tubercle, but regards this as occurring very rarely.[2]

Since the enunciation of the above opinion by Laennec, as to the source of these pulmonary lesions, it has been for the most part acquiesced in by pathological authorities; and, as usually happens when a doctrine gains general support, even the prudent reservations of its author have been in some danger of being consigned to oblivion. The accuracy of Laennec's observation as to the frequency with which these lesions occur, is more than justified by the later researches of M.M. Rogée and Boudet of Paris, and Professor Bennett,[3] who have found that pulmonary contractions and puckerings, with or without concretions and thickenings of the pleura, occur in a very large proportion of the bodies subjected to examination in hospitals. In the cases of Dr Bennett, the proportion is about 40 per cent. of the whole; while M. Rogée and M. Boudet give respectively 51 and 86 per cent. in their different

[1] Phthisie Pulmonaire. Edition of 1809. Vol. i. 478 et seq.; vol. ii. 321, 349. The connection of calculi with phthisis was also maintained by Bayle, as well as other still older authors. See Sauvages' Nosologia Methodica—article Phthisis.

[2] Auscultation Mediate; tome 2, chap. 4.

[3] See the paper of Dr Bennett, in Ed. Med. and Surg. Journal, vol. lxiii., p. 406.

spheres of observation. It thus becomes a matter of considerable importance to determine whether the connection of these lesions with obsolete tubercle be subject to no exception; more especially as none of the observers above noticed have, in drawing inferences from the facts adduced by them, indicated any doubt upon this subject. I have thought it right, therefore, to make the following remarks, tending to limit the application of the doctrine of Laennec, which in being made to include all, or nearly all, pulmonary cicatrices and concretions, under the designation of healed or obsolete tubercle, appears to me to have been scarcely warranted by the facts of the case. That I may not be suspected, however, of an equally exclusive bias upon the other side, I may state that the healing of tubercles in this particular manner admits, in my opinion, not of the smallest doubt; and that to any one who has seen, on a sufficiently large scale, the progress of these lesions, as exemplified in the lungs of those dying of unquestionable tubercle, the conclusions of Laennec as to the frequent cure of tubercular lesions, especially in their early stages, must appear perfectly irrefragable. Nor is there anything in the doctrines of Carswell, Cruveilhier, or other subsequent writers, from which I feel called on to dissent, in so far as they illustrate the different modes in which this cure is accomplished. But, as regards the precise frequency with which the early stages of tubercle become obsolete, I believe that we are not yet in possession of accurate statistical results; and that neither the 40 per cent. of Dr Bennett, nor the 51 and 86 per cent. of the French observers, represent correctly the proportion of such cases in our hospital bills of mortality.

That simple bronchitis must be responsible for a certain number of pulmonary contractions and puckerings, will be at once evident to the reader of the preceding pages. Every instance of pulmonary atrophy, from whatever cause, which is abruptly defined and surrounded by normal or emphysematous tissue, will necessarily present the appearance of a cicatrix,—more especially if the pleura over it be, as often happens, thickened. It might be supposed that such cicatrices would in simple bronchitis occur chiefly or exclusively at the back part of the lungs; but this is by no means the case; for while partial atrophy occurs with extreme frequency at the back part of the lungs, it is rarely complete, and almost always in the diffused form; while the obvious pulmonary cicatrices arise from lobular atrophy, which occurs chiefly at the edges and upper parts of the lung. I know of no means by which a simple cicatrix, formed by bronchitis or broncho-pneumonia, surrounded (as such cicatrices often are) by a certain amount of induration and carbonaceous infiltration, could be distinguished from a tubercular lesion, unless the absence of tubercular traces in other organs, and presence of lobular atrophy along the whole edges of the lung with diffused incomplete atrophy behind, and the comparative exemption of the summit of the lung, be considered to indicate such a distinc-

tion. Even to one aware of all the characteristics of both forms of disease, cases will constantly occur in which no distinct opinion can be formed; at least such is the result of my experience since my attention has been particularly drawn to this subject. Nor can I for a moment doubt, considering the frequency of bronchitis at all periods of life, that a proportion of pulmonary cicatrices, quite large enough to modify considerably the statistical results alluded to, has been included among the tubercular lesions without having a just claim to this designation. This is particularly the case with M. Boudet, whose enormously high proportion of 86 per cent. could only have been attained, as it appears to me, even among old subjects (and it is not asserted that his were exclusively such) by diligently seeking out every trace of pulmonary contraction, by whatever cause produced. By such a method the traces of extinct pulmonary disease may indeed be discovered in a very large proportion of cases; but certainly not of extinct tuberculous disease.

With regard to concretions, which were found by Dr Bennett in about 22 per cent. of the bodies opened by him, I have little doubt that they have a tuberculous origin in a large proportion of cases. But something in the way of reservation requires even here to be kept in view. The occurrence of such obsolete masses of exudation in connection with old-standing bronchitis is far from uncommon; and as Laennec, and after him many others, have clearly traced tuberculous matter through all its stages into that of complete calculous induration, so has it occurred to me repeatedly to see old bronchial abscesses, having evidently the characters and the usual distribution of bronchitic lesions, in every stage of conversion into these bodies. Even when the conversion was complete, it has appeared to me that a certain proportion of those I have met with might be reasonably referred to this source, from their prevalence at all the borders of the lung, or from their being surrounded rather by what I have called *simple lobular atrophy*, than by any considerable induration. But in this, as in the former case, the observer who looks to all the possibilities of the case is apt to find instances of a sufficiently equivocal and doubtful character in the present state of our knowledge.

Without being prepared to defend the following conclusions as absolute, and without at present entering at length into the grounds for some of them, I may here endeavour to state briefly the inferences to which I have been led by the preceding and other researches in regard to such lesions of the lung as may be suspected to be connected with the extinction of tuberculous disease. 1. There can be no reasonable doubt that open excavations, one or many, completely cicatrised on their internal wall, and lined by a membrane possessing the appearance, and in some degree the minute structure, of an epithelial membrane, may be of tuberculous origin;[1] but such exca-

[1] See the text of Cruveilhier, loc. cit.

vations (the *dilated bronchi* of Laennec) may also originate in simple bronchitis, broncho-pneumonia, simple isolated abscess (a very rare disease), multiple or metastatic abscess, syphilitic ulceration, gangrene of the lung,—which last lesion also may have several causes. Of these forms of healed excavation, the tuberculous is probably the most frequent; but there are as yet no good characters to distinguish it from the others, unless collateral lesions, sufficiently characteristic, be found in other organs, or unless the original lesion of the lung be, in other parts of the same or opposite organ, in an earlier stage of its development. 2. Concretions of inspissated pus, atheromatous matter, cretaceous or calculous matter, surrounded by atrophy, with or without induration, are in the great majority of cases the remains of obsolete tubercles, softened or miliary; but they may also arise from any other form of pulmonary ulceration or abscess, as above enumerated. 3. Pulmonary atrophy, simple or with induration, and carbonaceous deposit, accompanied or not by adhesions and thickening of the pleura, may arise from obsolete tubercles, from bronchitis, from broncho-pneumonia, and probably also, though more rarely, from simple pleuro-pneumonia; and pulmonary cicatrices arise frequently from all these sources except the last. The *relative* frequency of these lesions as leading to cicatrices is as yet undetermined. 4. The lesions above mentioned (1, 2, 3) *are probably tubercular*, if they occur exclusively or chiefly at the apices and back parts of the upper lobe of both lungs at once; or in the apex of one lung only, without trace of a lesion elsewhere; or generally diffused throughout both lungs, but chiefly in their upper lobes, and especially at their back part and apex; or in any case in company with characteristic traces of tubercular lesions in other organs. In reference to this last clause, it is doubtful whether cretaceous or other deposits in the bronchial glands can, in a case of such pulmonary disease, be considered as a characteristic indication of the tuberculous taint; but such deposits in other parts of the lymphatic system, especially in the mesenteric and cervical glands, or the traces of old deposit or ulceration in the mucous membrane of the [illegible]l intestine, would necessarily determine the tuberculous nature of the affection, except in some cases, rare enough in this locality, but of which I have seen examples, in which typhoid or cancerous disease might throw doubt upon the diagnosis. 5. All these lesions *are probably non-tubercular* if they occur in one lung in a generally diffused form, without traces of tubercle, even obsolete, in the other lung; or in the lower lobes to the exclusion of the upper; or at the edges of the lung in both lobes and not at its apex; or at the root of the lungs only; being in all these cases unaccompanied by tubercles, or the traces of tubercles, elsewhere. 6. After employing all these characters of distinction, a certain number of cases of all the lesions in question will remain of indefinite or unknown origin.

In concluding this contribution to the history of bronchitis, I am

well aware that many important practical relations of the various subjects discussed have been necessarily left almost untouched; but if I have succeeded in throwing any light on these relations, or in giving the practitioner a key to their apprehension in any degree simpler and of more extensive application than has hitherto been accessible, I am confident that the numerous minds at work in this country and elsewhere in the furtherance of practical medicine, will not allow these researches to remain destitute of the assistance which they are calculated to derive from more extensive opportunities of clinical research. With this conviction, I leave the foregoing pages in the hands of the profession.

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CASE OF ANEURISM OF THE AORTA,

ARISING FROM THE BACK PART OF THE ARCH,

SIMULATING LARYNGEAL DISEASE, AND FATAL BY SUFFOCATION.

BY W. T. GAIRDNER, M.D., F.R.C.P.,

PATHOLOGIST AND ASSISTANT-PHYSICIAN TO THE ROYAL INFIRMARY OF EDINBURGH

[REPRINTED, WITH AN ADDITIONAL NOTE, FROM THE MONTHLY JOURNAL OF MEDICAL SCIENCE, FOR AUGUST 1851.]

(Read to the Medico-Chirurgical Society of Edinburgh, 18th June 1851.)

THE following case of aneurism of the aorta is interesting, 1st, from the absence of physical signs, and the prominence of the symptoms of a laryngeal affection; 2d, from the free communication of the sac with a mucous canal, without causing serious hemorrhage; 3d, from the termination of the disease by suffocation, and the remedial measures suggested by this termination. On these grounds I beg to lay it before the Medico-Chirurgical Society, along with a preparation showing the parts involved in the disease.

Thomas O'Brien, æt. 46, a robust labourer, was admitted into the hospital at the hour of visit on the 30th May 1851. He complained of great dyspnœa, which, in the recumbent posture, was so extreme as to threaten suffocation. The breathing was sonorous, with a distinctly [illegible] character on inspiration. The countenance anxious and flushed; no fever or pain complained of. Expectoration considerable; the chest was examined as well as his state permitted, and revealed only slight bronchitic râles, the harsh laryngeal breathing being heard over all the bronchi. The voice was evidently produced with effort, scarcely husky, but having a somewhat muffled character; there was no tenderness over the larynx; the epiglottis and throat were natural. Shortly after his removal to a ward, the paroxysm subsided to some extent. I then learned that this was only an accidental exacerbation of a state which had existed for some months, and for which he had undergone active treatment. A blister was applied to the nape of the neck; and he was ordered ipecacuanha wine 3ss every second hour.

On the 31st he had slept well; but the dyspnœa had returned in