decays and yields; then the nature of the valvular lesion, whether static or progressive, ceases to be of much importance; the mural failure is then the object of our thoughts. If there be really fatty degeneration spreading through the heart-wall, then all our efforts will in no long time be futile to stave off the inevitable failure of the ventricle in diastole. But let me impress upon you this fact, that the heart, as a muscle, is liable to be affected by malassimilation, or general defective nutrition, and may be temporarily weakened in consequence. Such a condition I have described as "Heart-Starvation;" a term which holds its own, despite the not very polite remarks of one critic, who perhaps wrote in haste.

Another critic in speaking of this matter says: "Dr. Milner Fothergill enters fully into the diagnostic symptoms of fatty heart, distinguishing, in his usual forcible language, between a fatty and a starving heart, to discriminate which is most important. Each is the effect of malnutrition: one, however, is a perfectly curable disease; whereas fatty degeneration of the heart, with atheroma of the arteries generally, and of the coronary arteries especially, is a truly senile change, which carries with it the gravest prognosis." Now, you will find that partial starvation of the heart-wall is the common cause why a patient, with a valvular lesion, comes for relief. The lesion has been there for years; no change has taken place in it. What, then, has made the difference? The nutrition of the heart has broken down; may be, because the demand made upon the crippled heart has been in excess of its nutritive powers; may be, because the assimilative processes have broken down. Whatever the cause, the heart cannot longer hold out, and the person is laid up, possibly with dropsy. In such cases (and they are common enough), it is usual to speak of the valvular lesion as the cause of the patient's break-down; yet the lesion has been there for years, and is unaltered. It is really the compensating hypertrophy which has become defective; it is the condition of the heart-wall rather than the mutilated valves which should command our attention, and give direction to our therapeutic endeavours. Where there is merely an ill-fed heart, rest, good nutrition, and appropriate remedies will in no long time restore the patient to his ordinary state, and allow him to go on again. But when the ventricle is yielding, because its muscle is the seat of a necrobiotic process, and there is fatty degeneration spreading through the muscular fasciculi, it is impossible to arrest, or even to appreciably delay, the downward progress of the case.

From what has just been said, I hope I have convinced you that, when you have determined the seat, the extent, the nature, of the pathological process in any valvular affection of the heart, there remains the associated condition of the muscular walls to be calculated. The detection of the presence of the lesion by its appropriate murmur is not all that is required. It is but the first step taken, the first rung of the ladder climbed; and much more remains to be done. You have not only to make sure that the murmur tells of actual change in the valves; you have to ascertain the extent of the lesion; its origin, and whether it is static or progressive; whether the muscular wall is well fed, or ill fed, or decaying. And all this can usually be done if the requisite attention and thought be given to the case. The stethoscope is invaluable; but other evidence is essential. That a murmur means diagnostically a valvular lesion, prognostically sudden death, therapeutically digitalis and iron, is a shallow view; which, however, obtains too largely. I trust that this lecture will leave all of you with a much wider grasp of the subject; indeed, if you had remained satisfied with that simple, but comprehensive, if erroneous, view, you would probably not have honoured me by your presence here to-day.

HEALTH OF CAMBRIDGE.—If Dr. Anningson's reports are brief, they are to the point. For 1881, the death-rate represented 15.7 per 1,000; for 1882, 16.2; and for 1883, 18.0 per 1,000. Scarlet fever was very fatal in 1882, the mortality, indeed, being higher than any previously noted. Dr. Anningson has little doubt that this disease is kept active by the migration of persons attending various fairs held in the town. Assemblies of this kind exercise a pernicious influence in the spread of infection; and, on more than one occasion, sanitary authorities have endeavoured, sometimes with success, to forbid them. The reiterated recommendations of the health-officer for the provision of a sanatorium seem at last to be approaching realisation, since a site has been selected and approved, and the boundary-wall built, and provision made in the rates for the current year for the cost of its erection.

SUPERANNUATION.—Mr. Robert Jones, late medical officer for the Conway District and the workhouse of the Conway Union, has obtained a superannuation allowance of £40 *per annum*.

THE PATHOLOGY OF PULMONARY EMPHYSEMA.

Introduction to a Discussion in the Section of Physiology, and Pathology at the Fifty-Second Annual Meeting of the British Medical Association.

By D. C. McVAIL, M.B.,

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LAENNEC was the first observer who placed on anything like a certain basis the pathology of pulmonary emphysema. So far as mere anatomical description of the condition is concerned, nothing of importance has had to be added to what Laennec had told us. He wrote very accurately regarding the enlarged air-cells, showing that the enlargement affected more especially the cells of the surfaces, the margins, and apices of the lungs. He attempted more than mere description of the condition: he endeavoured to account for the abnormal size of the cells. He tells, concerning this, that in certain cases of dry catarrh there is secreted a very thick tenacious mucus; that little portions of this become lodged in certain of the smaller air-tubes; that inspiration (in his opinion), being a stronger force than expiration, keeps adding to the air in the air-cells beyond the obstruction, while the mere expiratory force can send little or none out of these same cells past the obstruction; and thus he supposed that, by an action that he compares to the charging of the air-gun, the vesicles beyond the little portion of tough mucus become more and more distended with air, until they attain to the sizes we see in the *post mortem* room. But, by and by, Louis pointed out that it was not in the parts of the lung that were actually suffering from bronchitic action that the emphysematous condition was chiefly found; he showed that, while the bases of the lungs were most prone to bronchitis, the condition of emphysema was, for the most part, located—as Laennec himself well knew—in the anterior surfaces, margins, and apices.

About 1835, Dr. C. J. B. Williams suggested that the emphysema in the portions of the lung, where it is found, is complemental, or vicarious, to other portions of the organ which from bronchitis had become obstructed or collapsed.

In 1850 and 1851, Dr. W. T. Gairdner greatly amplified and strengthened the vicarious theory of Dr. Williams, and gave it the permanent place it has since occupied. He strongly insisted that the inspiratory force caused the non-occluded cells so to dilate as to fill the space that was occupied by the collapsed cells, when they were in their normal state of patency. He further gave an explanation of the cause of pulmonary collapse, that up to the present time has been almost universally received, and concerning which I shall presently have something to say.

Some years prior to the time when Dr. Gairdner wrote, Fuchs and Mendelssohn had advanced the theory that expiration had the chief or only share in bringing about the emphysematous dilatation. They alleged that, in the strong expiratory act of coughing, while the glottis is partially or wholly closed, there is a very violent compression of the thoracic walls by the expiratory muscles, and chiefly by the expiratory muscles that act on the lower portions of the thorax; and by this means the compressed air will be driven with great force into the parts of the lungs that are not supported by the chest-walls, and so will dilate greatly the air-vesicles of the margins and apices of the lungs. This view was advocated, twelve years later, in this country, by Dr. Jenner, who, not knowing anything of Fuchs and Mendelssohn's work, argued the case in favour of expiration very much on the same lines, and so ably and lucidly, that all writers accord him on the matter a share of honour quite equal to that attaching to his two predecessors. He argued most resolutely against inspiration having anything to do with the vesicular dilatation. He has since, in his article in Reynolds's *System of Medicine*, changed considerably in his attitude towards inspiration as a cause of emphysema, although he still holds to expiration as being chief agent in the majority of cases.

Niemeyer, while adopting the expiratory theory as applicable to very many cases, and the vicarious view as being true of some, sought to explain a number of cases of emphysema where there was no collapse—cases of substantive emphysema—as being produced by a heightened negative inspiratory pressure. In England, he is supported by Dr. Waters, of Liverpool, who, quite independently of Niemeyer, explained, by the inspiratory force, cases of vesicular dilatation, where there had been no cough, and where there was no collapse of lung-tissue. But both these eminent writers limit this explanation to a comparatively small number of cases. Prior to Niemeyer, Freund ad-

vanced the view that ossification of the costal cartilages, by bringing about a loss of elasticity of the chest-walls, led to a condition of permanent dilatation of the thorax, and that this, acting on the lungs, caused an emphysematous condition of the air-cells.

Time forbids an examination of all these different views to-day, and I will now at once proceed to state the thesis that, at this meeting, I will seek to defend. Briefly stated, it is that the inspiratory forces alone, without the aid of expiratory force, can account for all the structural phenomena of pulmonary emphysema; and that all the phenomena known to us in pulmonary emphysema must be produced if the inspiratory movements be for a sufficiently long period of time markedly greater in degree than under normal circumstances (the length of time being, of course, greatly influenced by the nutritional conditions of the vesicular walls); and vesicular collapse does not necessarily precede pulmonary emphysema. I thus seek to extend the inspiratory theory much further than has been done by Williams and Gairdner on the one hand, or by Niemeyer and Waters on the other. I do not to-day in any way controvert the proposition that violent expiration may cause dilatation of air-cells.

In seeking to establish the view that has just been stated, I ask your attention first to the manner in which, in inspiration, the air-cells are dilated. The distending force is from without. The diameters of the thorax are all increased, and the lung is expanded by being drawn after the receding parietal walls. The force acts on the external surface of the lung necessarily, and thus first of all, and directly, on the most superficial of the layers of air-cells. Confining the attention to any single cell of the superficial layer, that cell, in inspiration, is moved in space to a position further from some point in the centre of the lung than in the expiratory condition it occupied. But it cannot be so moved in space without dragging after it the cell beneath it, and that in turn affects the cell deeper still; and so a chain of cells is acted upon. At one end of the chain is the superficial cell spoken of; at the other end, some cell close to the bronchial root of the lung. Now, a force acting on such a chain of cells, and in the direction of the axis of the chain—that is, perpendicularly to the surface of the lung—must operate on the cells in the following manner. The outermost cell, on which the force directly acts, having a certain *inertia*, will undergo a certain degree of elastic distension, until this tension equals, and becomes greater than, the inertia, when the cell will begin to move bodily in the direction of the force pulling on its outer wall. But attached to the opposite wall of the first cell is the second cell, with an inertia equal to that of the first; and, in virtue of this, it also undergoes a certain degree of distension before being moved bodily in space. But the first cell, in being dragged away in space, has now necessarily superadded to its own proper inertia the inertia of the second cell; and so, when the second cell begins to move in space, the first cell is being acted upon by a distending force twice as great as it would be were it alone and independent of all inertia save its own. And, when the third cell begins to move in space, the first cell is acted upon by three units of distending force, the second cell by two units, and the third cell by one; and so on, for every succeeding layer of cells that dilate and move outwards, there is added an unit of distending force to each of the cells that lie more superficial. As in hardly any section of the lung, from its surface to the bronchial root-divisions within, are there less than two or three hundred such cells, it follows that the superficial cells are acted upon by a distending force acting in the perpendicular direction that is enormously greater than that which acts on the deeply placed cells within. But the distending force acts not only perpendicularly to the surface; we have forces at all angles; and, as these necessarily act in straight lines, the outer cells are acted upon by a distending force infinitely greater than that which operates on the cells towards the centre of the organ. Still more is this the case with the cells of the angular margins of the lung; on these, the force, in its very highest degree, is exercised.

The force that distends a cell is spent chiefly in stretching or uncoiling the elastic tissue of the cell-wall, which, when the force ceases to act, contracts and recoils itself. This occurs with every respiration, is repeated more than twenty-one thousand times every day. But no animal or organic material, with which we are acquainted, is endowed with perfect elasticity. When stretched, no organic structure ever quite returns to its former proportions; every time it is strained, there is a certain degree of permanent yielding in the direction of the strain. This is true of the best made India-rubber elastic bands. It is true even of inorganic materials—of the best tempered steel spring; a spring weighing-machine becomes, in time, useless, from permanent partial giving way of the spring in the direction of the pull. It is most emphatically true of the vesicular elastic tissue of the lungs. There is a progressive giving way of this from youth to old age; the

cells are constantly becoming larger; every breath we take effects a change of this kind—infinitesimally minute, but, in the course of years, producing very evident results. The cells of the adult have a diameter twice as great as those of the child; and while, in the child, the diameter of all the cells, superficial or deep, is the same, in the adult the superficial cells are greater in diameter than the deep cells, and the marginal cells have the greatest size of all. In Quain's *Anatomy*, eighth edition, vol. ii, pp. 275-6, it is stated that, "in the adult human lung, their (the air-cells) most common diameter is about $\frac{1}{15}$ of an inch, but it varies from $\frac{1}{20}$ to $\frac{1}{10}$ of an inch; they are larger on the surface than in the interior, and largest towards the thin edges of the organ; they are also said to be very large at the apex of the lung. Their dimensions go on increasing from birth to old age; and they are larger in men than in women. In the infant, the diameter is usually under $\frac{1}{30}$ of an inch."

With every inspiration, in ordinary quiet breathing, the pulmonary vesicles and intervesicular passages are collectively distended, so as to make room for thirty cubic inches of air. While this distension is occurring, a graphic apparatus, applied to any part of the thoracic wall, shows a forward, or outward, and upward movement of a certain extent. If, instead of a making an ordinary inspiration, a healthy young adult man makes the very deepest forcible inspiration, much more air enters the chest, and the forward and upward movement of any point of the chest-wall is very much greater than in ordinary quiet breathing. Normally, everyone possesses a very large "breath-reserve" space; he may voluntarily breathe at any level between the ordinary quiet respiratory level, and the highest limit to which he can raise the thoracic walls; and, involuntarily, he breathes at different levels according to the muscular or other effort in which, at the time, he happens to be engaged. Here, for example, is a tracing (diagram was shown) of the quiet breathing of a person sitting in a chair, his back resting firmly against the back support of the chair. At this point he had heavy weights put into his hands, he sitting perfectly still as before; and you see how the respiratory level rises, and continues to rise, until, at this part of the tracing, the weights were dropped, and the breath-level falls very suddenly through a considerable space, and then more gradually declines to the original level.

In a young healthy adult man, this breath-reserve space is of very considerable amount. The lower end of the sternum can be advanced in a forward direction at least about an inch and a half by the greatest forcible inspiration, in front of its position in ordinary quiet breathing. But every year, even in the most perfect health—absolutely without bronchitis—this reserve-space is being encroached upon from below, as the air-cells, from the causes of which I have already spoken, are undergoing slow progressive enlargement. There is, in fact, in every person, from childhood onwards, being certainly developed what I may term physiological emphysema. We are all the subjects of it, and, if we lived only long enough, we would exhaust this reserve entirely, and would die from being unable to inspire; and all this without disease or the operation of any abnormal condition whatever. In the case of a perfectly healthy man, whose reserve I carefully measured ten years ago, and who has not since had any pulmonary disease, and is now in perfect health, I find that his reserve is lessened by fully a quarter of an inch, as regards the forward movement of the lower end of the sternum.

In connection with this, I was greatly interested in a demonstration by my friend, Dr. Knox of Glasgow, who, some years ago, showed that the aorta immediately above the semilunar valves, while quite cylindrical in children, becomes somewhat globular with advancing years. This seems to me to be really a condition of physiological aneurysm, analogous, in its mode of production, to physiological emphysema.

But if a physiological emphysema be thus developed when the air-cells undergo only the ordinary dilations of moderate breathing, how much more rapidly will it develop under circumstances where the respiratory movements are, for long periods of time, greater by far in range. Such a condition we have in bronchitis. The mucous membrane lining the bronchial tubes in the inflamed condition becomes thicker, from the congested state of the capillary vessels in its wall, and from cell-proliferation, and the diapedesis of leucocytes. The lumen of the tube is thus narrowed, and the air to enter the terminal vesicle has to undergo increased friction on its way. This quite involuntarily brings about greater respiratory movements, and these are at a higher level, nearer the top of the reserve space. When, for example, in the case of a person whose thorax is writing its respiratory movements by means of this tracing machine, the spiograph, I apply to his face this mask, having only a very narrow opening to the outer air, the air entering his lungs is subject to much greater friction than under ordinary circumstances; and you see from the tracing here

how the movements become greater in range, and how the level rises. This tracing, you see, is very similar to the fatigue-tracing that I showed you, brought about by the person holding heavy weights. How greatly are the cells, under such circumstances, stretched and strained; and in chronic bronchitis this is continued for weeks, and months, and years, so that emphysema must follow in its wake as surely as the shadow follows the substance. Under these circumstances, also, the enlargement of the cells must be greater at the surface of the lung, and greatest of all at its sharp margins, owing to the manner in which the distending force acts on the cells, as I have already explained.

But it will here be said the enlargement of the pulmonary cells is also extremely great in the apices of the lungs, and that, under ordinary circumstances, the inspiratory movement there is less than towards the lower parts of the chest; and that, therefore, there, at all events, we must call in aid the expiratory theory of Mendelssohn and Jenner. Such seems to be the idea, at all events, of Niemeyer and Waters. But the inspiratory force will amply account for this also. It is true that, under ordinary circumstances, the movement in respiration of the lower portions of the thorax is greater than in the upper parts; but, in far advanced bronchitis and emphysema, this is entirely reversed.

Time will not now permit me to enter into a description of the mechanical thoracic phenomena, by which this reversal of action is brought about, but I simply appeal to what every one who has observed the breath-movements of advanced cases of emphysema knows to be the case—that the sternum, at its lower end, does not advance in inspiration at all, but actually recedes. The whole forward movement is at the upper part of the thorax, and the inspiratory force must therefore, in such cases, give rise to great emphysematous enlargement of the cells at the apex.

With altered and impaired nutrition, the process of cell-dilatation will necessarily be greatly hastened and intensified; but time will not allow me now to state this aspect of the question, nor can I now allude to the share in the process due to loss of elasticity of the thoracic wall itself.

But, before sitting down, I desire to say a few words on the relation of emphysema to pulmonary collapse. Dr. Gairdner regards pulmonary collapse as the cause of emphysema, and undoubtedly emphysema must ensue in all cases of collapse; but I have shown that without collapse at all emphysema occurs; and now, further, I have to say that in many cases pulmonary collapse results from emphysema. Dr. Gairdner, rightly, in his celebrated series of papers, to which I have already referred, considers that collapse is very frequently owing to little pellets of tenacious mucus obstructing certain of the smaller air-passages. He supposes that, under ordinary circumstances, the unstripped muscular fibres of the walls of the air-tubes, by peristaltic action, carry outwards the mucus that he supposes to be constantly secreted in the tubes, even in health; and that, in bronchitis, the mucus secreted is often extremely tenacious, and the muscular fibres possibly less active, and so the mucus here and there is not carried away, and partially blocks up a tube. He further supposes that, in expiration, the air more readily escapes from the cells beyond than it can enter in inspiration, and soon there is little or none left to escape; that now, by pressure of the air on the upper surface of the pellet of mucus, it is driven more firmly in, and reaches, by-and-bye, a part of the tube so narrow that it becomes permanently impacted there, and cuts off completely, from all external communication, the cells beyond; and whatever air remains within these finally disappears.

That in certain parts of the air-passages these muscular fibres may have some such peristaltic action, I am not prepared to deny; and if I were to object to it, I certainly am in no position to prove that these muscles may not so act. That is a point on which, in the meantime, no one can adduce positive evidence either one way or another. It is mere conjecture; but quite certainly, as regards many parts of the air-passages, these muscles can have no such action. In the bronchial tubes, from those of the largest size down to those of one-thirtieth of an inch in diameter, they cannot so act, as the walls are stiffened by cartilaginous plates, and cannot so contract as to act peristaltically. Then again, ere the tubes break up into vesicles, the muscular fibre disappears. Thus, in a very large part of the tubular structure of the lungs, peristalsis is impossible. How then, in these portions of the tubes, is the mucus carried outwards, against gravitation, and in spite of its adhesive clinging to the tubular wall? The general answer would be that it is moved on by the action of the cilia of the columnar epithelial cells. No doubt this has something of an expulsive action; but, in the finest of all the tubes, and in the ultimate cells, there are no cilia present; and in bronchitis, where from the abundance of mucus to be got rid of there is most need of

such aid, it is well known that just in these very cases large portions of the tubes are entirely stripped of the ciliary cells that normally line them.

By what means, then, is mucus moved in those parts of the bronchial tubes that can neither undergo peristaltic action, nor be swept by ciliary motion? This is effected by circumstances that, in so far as this particular action is concerned, have not hitherto been taken into account. A blast of air in a direction that is greater in volume than a blast in the contrary direction will—other things being equal—have greater effect as a motive agent than its opponent. The expiratory blast of air is greater in volume than the inspiratory. It owes its increased volume to two circumstances. It is, on an average, about 15° C. warmer than the inspired air, and as air expands by the $\frac{1}{273}$ of its bulk for each such degree, this will increase its bulk by about one-eighteenth. Again it is saturated at this high expiratory temperature by aqueous vapour, which, at the particular tension due to that temperature, will add about one-thirtieth to its original bulk. We have, thus, from these united causes, the expiratory blast greater than the inspiratory, by about one-twelfth.

Mucus or other material adhering to the interior surface of the air-tube is thus, by the inspiratory blast, moved from its original position inwards; and by the expiratory blast, in a direction outwards; but as the latter blast is the greater, the object will be moved to a position further outwards than before that particular respiration took place. This inspiratory flow and expiratory ebb of the mucous tide goes constantly on, but the ebb is always greater than the flow. In health, the tide is always going out, and never going in.

But another circumstance more potent still is at work. In health, expiration is accomplished in a shorter time than inspiration—shorter by about a fifth; and from this alone it would be more effective in driving before it obstacles than the inspiratory air, apart altogether from the difference in volume of the two blasts. This is well known in mechanical operations; the blow of a hammer drives the nail, which the same force more gradually applied would fail to move. The influence of the more sudden impingement of the expiratory blast on the substance to be moved is very great; and, coupled with the increase in volume of which I have just spoken, it moves upwards and outwards the mucus that in health is secreted less or more abundantly, without voluntary assistance on our part—without our consciousness even of the movement at all.

In emphysema, how are these two factors in the expulsion of mucus affected? So far as the increase in volume is concerned, that may for the present be regarded as being practically under the same conditions as in health: it will be warmed, and it will receive aqueous vapour. But the second factor undergoes profound modification. Expiration in relation to inspiration lengthens; it becomes more and more prolonged, until it occupies twice the time that inspiration does, or even more than twice the time. It now impinges on the mucus so gently as hardly to move it outwards at all, while the inspiratory air keeps moving it inwards. The tide of mucus is now turned; the flow inwards is greater than the ebb outwards. The inward flow is not so powerful as it would be were the blasts equal in volume; still the advantage soon comes to be on the side of inspiration, and the mucus pursues a course downwards towards the air-cells of the lung. Only by coughing can the mucus now be got to travel outwards—coughing, which is simply an exaggeration of an ordinary respiration, in so far as the shortening of expiration in relation to inspiration is concerned. So soon as the cough ceases, the mucus in the emphysematous patient again begins to travel backwards with every respiration; and so he coughs again and again, the frequency depending on the amount of mucus secreted, and the relative increase of the duration of the expiration to that of the inspiration.

But by-and-bye, here and there in the lung, portions of mucus, by the inward flowing tide, become impacted in the smaller tubes in spite of all the efforts of coughing; and then we have collapse of lung-tissue subsequent to, and, in the manner I have described, caused by the emphysema. This reversed mucous tide is usually, too, in the end, the immediate cause of the patient's death. The efforts of coughing become more feeble as the patient's muscular strength declines, and as the elasticity of lung and thoracic wall still further lessens. More and more the expiration lengthens; the inward movement ever becomes greater; the outward ever becomes more feeble, until the small tubes are so filled with mucus, that air sufficient for the wants of life cannot enter, and thus death occurs by the so-called capillary bronchitis; the secretion, however, that fills these minute tubes being for the most part produced elsewhere, and only conveyed to them in the manner that has just been described.

Other points I would have gladly spoken of, but I have already far exceeded the time allotted to me.

Professor GAIRDNER (Glasgow) regretted that time would not allow him even to attempt to argue out all the points raised by Dr. McVail's communication. The main point to which he would address himself would be the theories he advanced thirty years ago, and the views he then expressed. Had he to revise these now, he did not think he should do so on the lines adopted by modern writers on the pathology of emphysema. He could not agree with the eclecticism which sought to bind together all the views regarding the production of emphysema, and affirmed that all or any might be pathogenic factors. In Dr. Gairdner's opinion, emphysema had, in all probability, one mechanism, and not many mechanisms. In no case could he regard expiration as being productive of dilatation of air-vesicles. The immediate mechanical force must, to his mind, be the inspiratory, and no other. How it was actually determined in all cases, or by what intermediate processes in any individual case, he could not say. But this he would say, a perfectly healthy man could not overdistend his air-vesicles. Mechanical conditions absolutely precluded the possibility of overdistension of a single air-vesicle. Emphysema only arose when disturbance of air-currents existed, which prevented the air from entering equally into all vesicles. Relative obstruction of bronchi meant relative collapse of corresponding air-cells, and the tendency of inspiration was to overdistend collateral air-vesicles. As a rule, the vesicles which tended to relative dilatation were those which were most superficial in position. Dr. Gairdner next referred to the possible uses of the bronchial muscles. These, he considered, had probably definite physiological functions. He could not conceive the tidal air of the lungs passing in and out, and giving a clear scour of the bronchial mucus; he advanced the theory that the peristaltic action of the bronchial muscles was an important factor; therefore, he regarded these as quiet but constant scavengers, and termed them "deobstruent muscles." As another possible function, Dr. Gairdner thought that the bronchial muscles might regulate the admission of air to particular air-vesicles, and so possibly tend to prevent overdistension, or even rupture of them, when subjected to temporary undue strain in inspiration.

Dr. DOUGLAS POWELL (London) was greatly interested to hear the elastic agencies of the body so fully recognised by Dr. McVail in his able paper, these agencies having been, in his opinion, too little recognised in regard to health and disease. He could not but think that more causes than one must be allowed for emphysema. There were at least three principal causes. 1. There were structural changes involving impairment or degeneration of tissues; these naturally ensued upon those senile changes which, as had been shown by Dr. McVail, commenced at an early age, but were in health very gradual, proceeding *pari passu* with similar changes in other parts. In diseased conditions of various sorts, however, these structural changes were hastened, and became an important cause of emphysema in early middle life. 2. Overstrain from inspiratory effort, in the manner pointed out by Professor Gairdner, must be admitted as a very frequent determining cause of emphysema. 3. Dr. Powell was convinced that overstrain of elastic tissue of the lung from expiratory effort, particularly that of tussive expiration, could not be ignored. He thought that the action of inspiratory effort in inducing emphysema was well shown in cases of diphtheritic obstruction, that of expiratory effort in whooping-cough. He thought Dr. McVail had not attached sufficient importance to the state of the pulmonary vessels in emphysema, upon the narrowing and obliteration of which the secondary cardiac and other congestive results of emphysema were mainly dependent. Without these changes, the consequences of emphysema would not be serious. Dr. Powell was much interested in hearing Professor Gairdner's account of the functions of the bronchial muscles in health and disease. He thought that, amongst other functions, these muscles helped to preserve the bronchi, which were naturally held patent by the elastic traction of the lung, from becoming overstretched during powerful inspiratory effort; the muscles, by their contractile tonicity at such moments, counteracting the excessive inspiratory force.

Professor REDFERN (Belfast) remarked on the clearness and care with which each step of Dr. McVail's argument had been put before the Section. He asked if it had been ascertained with certainty that the superficial and deep air-vesicles were of the same size at the earliest periods of aerial respiration, that being essential to sustain the argument of the paper. He believed that the great cause of emphysema was collapse, as Dr. Gairdner pointed out; but he could not resist Sir William Jenner's expiratory theory as applicable to certain cases, and wished that Dr. Gairdner would reconsider whether it was necessary that there should be only one cause of emphysema. He stated his belief that one function, probably the most important function, of the bronchial muscles was to act like the muscles of arteries, regulating the supply to different parts of the organ, and under different circum-

stances. In most cases, the arterial muscles regulated the whole supply. In the lung, the bronchial muscles were as much required to regulate the quantity of air, as the arterial muscles were to regulate the quantity of blood.

Professor GAIRDNER explained that he did not say there was only one cause of emphysema, but that there was only one mechanism.

Dr. EWART (London) expressed himself as having some difficulty in understanding how the function attributed by Professor Gairdner to the bronchial muscles could be satisfactorily explained. Any mucus supposed to collect at the bottom of the lung could hardly be propelled along the air-tubes by the bronchial muscles, unless it filled the bronchiole, and, if the bronchiole, then ultimately the trachea. The fact that masses of mucus filling the calibre of the trachea were not expelled in health, strongly militated against the view that mucus was, under normal circumstances, excreted from the lungs. The mucus was probably decomposed *in situ* by vital agencies, or absorbed as such by the pulmonary lymphatics, which were as widely spread as the mucous membrane itself. Dr. Ewart looked upon the bronchial muscles as mainly expiratory, although acting likewise, as Dr. Douglas Powell had remarked, during inspiration. In forced expiration with closed glottis, intrapulmonary pressure was greatly increased, and, if unopposed, would lead to dilatation and bulging of the air-tubes. This could be obviated by the action of powerful muscles. In criticising some of the remarks contained in the paper, Dr. Ewart suggested that infundibula in various parts of the lung were practically in identical conditions of pressure. Inasmuch as their dilatation was effected by atmospheric pressure acting from within, whilst they received corresponding support from without, a natural inspiration, however full, would lead in them to no injurious strain. During the forced expiration of muscular effort, and of cough, true pressure might be set up at different parts in the lung, to which the weakest part would yield. Emphysema might owe its origin to various causes. Any long-continued inspiratory dyspnoea would lead to its production where the pulmonary tissue lacked resiliency or power of growth. In most cases, the expiratory theory would afford, however, the best mechanical explanation. But, above all other agencies, the influence of disturbed nutrition of lung-tissue, and of variations in the tone and fulness of the pulmonary vessels, should be recognised as a leading factor.

Dr. CARTER (Birmingham) said that the circumstances favouring emphysema might be divided into two groups: those inducing emphysema; and, secondly, those affecting the localisation of emphysema. With reference to the first of these, Dr. Carter thought the popular views were too exclusive. He agreed with Dr. Ewart as to the primary importance of nutritive changes. So long as the elasticity of the walls of the air-vesicles remained in a healthy condition under ordinary circumstances, no stretching would cause emphysema; but when nutritive changes concurred, a very moderate amount of increased tension might result in permanent emphysematous distension. The primary factor was nutritive changes in the elastic tissue of the lung; then anything producing tension, no matter how produced, tended to the production of emphysema. No mention, Dr. Carter said, had been made of the tension which permanently existed, not only during inspiration and expiration, but also during pause. This pressure was increased during inspiration, and might be increased during expiration, by coughing. Sometimes it was the one, sometimes the other, and sometimes both combined, which determined any given case of emphysema.

Professor CHARLES (Cork) disagreed with the proof Dr. McVail gave of the expiratory force being greater than the inspiratory. Dr. McVail stated that, in the pneumographic tracing, the part corresponding to expiration was shorter than that to inspiration. Dr. Charles held that, in normal healthy breathing, there was scarcely any pause; in fact, what had been styled the pause might be regarded rather as the continuation of the expiration; consequently, the expiratory part of the tracing would be really longer, as a rule, than the inspiratory.

Professor GREENFIELD (Edinburgh) had to confess that he was eclectic so far as regards the causation of emphysema. He believed the expiratory force had some efficiency, and asked Dr. McVail if the emphysema of those who played wind-instruments had been adequately explained on the inspiratory theory. Dr. Greenfield would attach more importance to the degenerative changes which, he believed, preceded emphysema, and in many cases, he thought, might be the primary cause. He asked, further, whether the inspiratory theory accounted for the rupture of vesicles in whooping-cough. With regard to the action of the bronchial muscles, Dr. Greenfield was inclined to agree rather with the second view advanced by Professor Gairdner. No reference had been made to the dilatation of the smaller bronchi in emphysema; and he pointed out that dilatation followed inflammation of the bronchial tubes.

Dr. McVAIL, in reply, said that, at that advanced hour of the meeting, he would say only a very few words. He in no sense, at that meeting, sought to controvert the view that, under certain circumstances, the strong expiratory act of coughing might give rise to emphysematous dilatation of the cells, and he would therefore pass over all that had been said by speakers on that point. Also he had distinctly guarded himself from any remarks on the nutritional causes of emphysema, as time had not permitted the extended explanation of their share in the production of emphysema, that was desirable. As, however, nearly all the speakers had alluded to this side of the question, he would say that he was convinced that nutritional, or trophic emphysema, apart altogether from bronchitis, was exceedingly common. He found that young men, who came to the dispensary of the Western Infirmary of Glasgow, were peculiarly subject to it: young men from ship-building yards, and other public works, in Glasgow, who had begun, at a very early age, to drink whisky on pay-nights, and who, at a very little over the age of twenty years, had, in many cases, already lost as much as a quarter, or a third, of their breath-reserve space, and whose arteries also were becoming zigzagged from impairment of their elastic tissue. In fact, this question of trophic emphysema was one largely affecting life-assurance, and all questions of health-prognosis; and its full significance had yet to be wrought out by the aid of such instruments as the spiograph that he showed them. It was because this question was so vast, that he had almost entirely left it out of view in the remarks with which he opened the discussion. He hoped, however, shortly to bring a more extended account of it before the profession. He could not understand how Dr. Gairdner could object to the view that prolonged breathing, at a high respiratory level, would give rise to vesicular dilatation. If the collapse of, for example, four cubic inches of lung-tissue, must necessarily, in Dr. Gairdner's opinion, cause a corresponding dilatation, distributed less or more evenly among the other cells of the lung, surely an extra dilatation of from fifty to two hundred cubic inches, brought about and maintained by any cause, no matter what, would much more give rise to cellular emphysema. Dr. Ewart had objected to the explanation given of the mode in which the dilating force acted on the air-cells; but, as to that, there could be no doubt at all; the distending force was from without, and, for every successive layer of cells acted upon, the outer layers must successively undergo an extra unit of strain, just as the addition of an extra wagon to a railway-train, going up a hill, added an unit of tension to all the couplings between it and the engine.

ON THE OPERATION (ALEXANDER - ADAMS) OF SHORTENING THE ROUND LIGAMENTS FOR UTERINE DISPLACEMENTS, WITH THREE CASES.

Read in the Section of Obstetric Medicine at the Annual Meeting of the British Medical Association.

By W. L. REID, M.D.,

Physician to the Dispensary for Diseases of Women, Western Infirmary;
Physician to the Maternity Hospital, Glasgow.

FROM my friend Dr. James A. Adams, of Glasgow, I had heard of his operation for replacing displaced uteri a considerable time before I knew it had been proposed and practised by Dr. William Alexander, of Liverpool; but I did not attempt the operation until I had seen the latter gentleman perform it on the dead body, and had heard particularly of his results in the living subject. Although, from the position I occupy, I had abundant opportunities, it seemed right to wait until I met with what appeared to be test-cases, in women who were willing to have practised upon them an avowedly new and so far untried operation; and it is to the histories and results of three such cases that I now direct your attention. Possibly, some little extra interest may attach to them, in so far as, to the best of my knowledge, they are the three first successful cases operated on in Scotland, speaking of them simply as operations for replacing displaced uteri.

CASE I.—Mrs. McG., aged 33, began to menstruate at 16, and was regular till pregnancy, two months after marriage, at 20. She had one child naturally, and had never been well since. The uterus was found much hypertrophied and retroflexed. The left ovary was prolapsed. She complained greatly of backache, pain in the left ovarian region, and inability to walk any distance. There was great and continuous leucorrhœa; and intercourse was almost unbearable. Dysmenorrhœa was severe, and there was frequent micturition, especially during the night. These symptoms had continued more or less

steadily for ten years. She was treated by me at the Western Infirmary for nearly two years, during which time she obtained temporary relief from pessaries, glycerine tampons, scarification of the cervix, and intra-uterine applications of iodine, and of nitric and carbolic acids.

On November 1st, 1883, assisted by Drs. James A. Adams, George A. Turner, and James Ross, I performed the operation of shortening the ligaments. The pubes was shaved and washed with carbolic water, and carbolic spray was used during the operation. An incision was made on the left side from the pubic spine upwards and outwards for two inches, and carried down to the tendon of the external oblique muscle. The external abdominal ring was felt, and the tissues coming out of it were gathered up by an aneurysm-needle, and seized with the fingers. They could not be drawn out, even by the exercise of considerable force. They were then grasped by a pair of broad-pointed dressing-forceps, and freed from the surrounding tissues with the help of the scalpel; and, on continued pulling, they very gradually came out, until the firm round ligament appeared. The same procedure was repeated on the opposite side. A sound was then passed into the uterus, and the organ braced well up in its normal position, and the ligaments were pulled on until they were found to be fixing it there, when fully two inches were found drawn out on each side. Three strong chromicised catgut stitches were passed through the pillars of the ring, each transfixing the cord. These were not tied very tightly. The ends of the cord were folded up in the wound, and the edges of the skin brought together by gut-stitches over a rubber drainage-tube. To take the strain off the ligaments, a pessary which the patient had worn before was introduced. A dressing of gauze and cotton-wool was applied, and kept in place by an elastic bandage. In the evening, the pulse was 90, and the temperature 100.2°. The patient complained only of something being "very tight" in the lower part of the abdomen. The wounds were dressed without spray, with the idea that the scar-tissue would be firmer if suppuration were allowed to take place. It would serve no good purpose to give careful details of her progress; suffice it to say, that the wounds were dressed twice a day for four days, and once a day thereafter, until she was dismissed on the twenty-sixth day, there being then only a corner of one of the wounds unhealed. Her highest temperature was on the second day, namely, 101.4°; on the third day, it fell to 98.6°.

At the time of her dismissal, she complained of frequent desire to pass urine when standing or walking, but this soon passed off. The pessary was left in position, and was not removed until a month afterwards.

On April 21st, six months after the operation, I saw and examined her. The cervix was found high up, and normal in position. The fundus could not be felt in the posterior vaginal fornix, but the point of the sound passed backwards, showing that the organ still remained flexed.

On July 23rd, when asked to what extent she had improved since the operation, the patient mentioned the following points. The dysmenorrhœa and dyspareunia were both gone; she did not require to micturate during the night; defæcation was much more easy; and the weakness of her legs was so much improved, that she could walk two miles without special fatigue. On the other hand, she still complained of backache and ovarian pain; the uterus was still three and a quarter inches in depth; there was erosion of the cervix, and a mucopurulent discharge from the os uteri. I may add that, since the operation, the ovary has never come within reach.

CASE II.—Mrs. H., aged 31, was first seen by me on September 4th, 1880, when the following note was taken. She began to menstruate at 10, and was regular, with some dysmenorrhœa on the first day, until pregnancy, two months after marriage, at 21. She menstruated one month after her labour, which was normal, except that the second stage was long. Milk died away when menstruation appeared. She had been quite regular, but never felt well since her confinement. She complained of backache, leucorrhœa, frequent micturition, and of a sharp attack of diarrhœa, which came on every morning immediately after rising, and did not return during the rest of the twenty-four hours. She had no ordinary dysmenorrhœa, but suffered severely from headache before and during each menstrual period. This symptom was often so severe as to be accompanied by delirium, and to confine her to bed for three or four days. The patient was stout and florid. The uterus was found retroverted, three and a half inches in depth, very heavy. The cervix was hypertrophied, and patulous. The perineum was partially destroyed, and much relaxed.

Her history for the next three years may be summed up in saying that, by the use of the means mentioned in connection with the case already detailed, she obtained at various times great relief, which, however, never lasted more than a few weeks; and, on November 3rd,