remedy failed, I succeeded in procuring almost instant relief, by injecting two grains of morphine and a drachm of tincture of assafœtida. These were cases where mental distress appeared to be the exciting cause.

I have often sat at the bedside of one, suffering from the severest form of the disease, watching with great anxiety the result of prescribed remedies, and it has not unfrequently happened that many have been tried without relief, the patient all this time gasping for life with sufferings the most intense, when relief has at length come from a remedy apparently the most unlikely to procure it—so capricious is the disease, and so uncertain the remedy in asthma cases of this particular character.

Having now recorded my treatment and opinion as regards a considerable number of cases of asthma which have come under my notice during the space of some years, I would, in concluding these papers, make a few additional remarks, which may, perhaps, lead those disposed to follow up the subject with scientific research, to throw still more light as to the actual cause of this disease. So long as asthma is ranked amongst diseases which are called peculiarly nervous, so long can we never hope to come to any correct conclusion; the idea being so vague, and the laws which govern the nervous system for the most part so incomprehensible. Is it not more likely that the real cause of the disease is, as in other hereditary diseases, some impurity or deficient element in the circulating fluid. For instance, do we not find in scrofula, remedies in iodine, and in the phosphates of lime and iron? In phthisis, do we not see that cod liver oil will arrest the disease, and in some instances eradicate the predisposition to it? And do we not find in some forms of gout that colchicum and alkalies will keep at bay this disease? In other instances, all traces of gout will be lost by simply drinking at meal-times what is commonly called rough cider. How are these results brought about, but through the medium of the circulating fluid, which takes up the antidote after the process of digestion is completed, and receives from the remedies employed, that which is required to nourish the human frame, and carry on life free from those diseases which hereditary predisposition has implanted? True it is that the exciting causes of asthma are various; and that the disease, although in the constitution, may not be brought to light until the individual so predisposed comes in contact with such causes. Now, in every case which I have recorded, I have been able distinctly to trace hereditary predisposition; and what, may I ask, does hereditary predisposition mean but a taint in the blood, or, in other words, a constitutional defect?

I think it must be admitted from the history and symptoms which I have given of dyspeptic asthma in particular, that this form of the disease, at least, is under the control of medical treatment. Nothing can be more conclusive of the fact than the evidence given by those who had been martyrs to the disease, and have had sufficient resolution to carry out the prescribed treatment in its full integrity. This being admitted, what does it prove? Surely that the beneficial change is derived from the purity imparted to the blood by means of a more perfect performance of the office of digestion of the wholesome food which is taken into the stomach for the purpose of nourishing the body, and here lies the secret of the treatment in an especial manner of all constitutional diseases. If this principle be admitted, then we have advanced far on the road in combating this distressing and formidable disease.

I have before stated that the next frequent exciting cause which brings the disease into action is atmospheric; these results are most mysterious, as, in some instances, an atmosphere apparently the most highly charged with impurities will act as a specific in the disease, whilst apparently the most pure air will, on the other hand, bring on the most distressing state of sufferings to the person predisposed to the disease of asthma, and *vice versa*; and how, I would ask, can these contradictory phenomena be accounted for, except by the supposition that some peculiar element in the atmosphere either adds to or takes from the healthy condition of the spring of life? In the atmosphere both the bane and the antidote are to be met with. And why should not the remedy be in our own hands, in these days of advance in science, both as regards microscopic discoveries and chemical research?

There are other exciting causes of the disease which, as yet, appear to baffle all conjecture as to how they act so instantaneously; I mean impressions on the mind, such as grief, fear, sudden passion, news of an exciting nature, and hysteria. When these cases do occur, I have almost invariably discovered, on strict inquiry, that hereditary predisposition to the disease prevails. There is, however, one remarkable feature in every form of asthma which is well worthy of notice, and it is this, that on the termination of every attack an expectoration is thrown off from the lungs varying in quantity and appearance. In some instances there is a thick heavy mucus; in others a large quantity of mucus of a light frothy appearance; whilst in the severest form of the disease there may be only a few small dark pellets coughed up before relief is obtained.

I will not venture further in my remarks which are the result of my own personal experience. I can only hope they may lead those who are more particularly occupied in scientific research to bear in mind the few observations which I have ventured to bring before my professional brethren.

OBSERVATIONS ON THE MORBID ANATOMY, PATHOLOGY, AND DETERMINING CAUSE OF EMPHYSEMA OF THE LUNGS.

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[*Read before the Royal Medical and Chirurgical Society.*]

Part II.

Two Principal Theories of the Determining Cause of the Disease. Views of Laennec. Theory of Dr. Gairdner. The Mechanism of Respiration. Effects produced by Collapse of the Lung. The Expiratory Theory. The Modus Operandi of Expiratory Efforts. The Anatomical Arrangement of the Walls of the Chest and the Disposition of the Lungs. Effects of Forced Expiration. The Case of M. Groux. Results of the Author's Observations. Infrequency of the Disease as a Sequel of Pleurisy and Pneumonia; its frequent occurrence in Tubercular Lungs. Cases recorded by M. Guillot. Hereditary Nature of the Disease.

Speaking generally with reference to the determining cause of emphysema, we may say that two principal theories of it have been entertained by pathologists: these are respectively called the inspiratory, and the expiratory, theory.

By those who hold the former view, it is supposed that dilatation and subsequent rupture of the air-sacs of the lung take place as the result of their over-distension during an inspiratory act; by those who entertain the latter view, that these results are brought about by expiratory efforts, more especially such as are produced by coughing.

Laennec, recognising the frequency of the disease as a sequence of pulmonary catarrh, supposed that it was occasioned by an overdistension of the air-cells, from an accumulation of air taking place in them, in consequence of the obstructed condition of the bronchial tubes. He says:—"The small bronchial tubes are distended by the viscid mucus, or by the swelling of the mucous membrane. Now, as the muscles which act in inspiration are strong and numerous, and as expiration, on the contrary, is only produced by the elasticity of the parts and the feeble contraction of the intercostal muscles, it must often happen that in inspiration, the air, after having overcome the resistance which was opposed to it by the mucus, or by the tumefaction of the mucous membrane, cannot overcome it during expiration, and remains imprisoned. The following inspirations add further to the dilatation of the cells to which the obliterated tube leads. Lastly, the distension by the heat of the lungs, of the air introduced cold into the chest, must contribute to this dilatation."

The theory thus advanced by Laennec is based on a view of the respiratory function which has been proved to be essentially incorrect; viz., that the inspiratory power is greater than the expiratory. The researches of Hutchinson and others have shown that the power of forced expiration considerably exceeds that of inspiration. This important physiological fact cannot be too constantly borne in mind in considering the nature of the affection we are examining.

But, further, it has been shown by the researches of Gairdner and others, that an accumulation of mucus in the bronchial tubes, such as Laennec thought would lead to a distension of the air-sacs, has an exactly opposite effect, and is, in fact, followed by a collapse, and not a dilatation of the pulmonary tissue. Dr. Gairdner has shown that the pathological condition of the lung, which has been known under the name of

lobular pneumonia, is nothing more than a collapse of the lung-tissue, which—unless the affection have existed for a long time, and have produced a truly atrophic condition—may be readily distended by inflation through the bronchial tube leading to it. He has found this state of the lung following bronchitis, and constantly associated with an obstructed condition of the bronchial tubes leading to the affected part. He accounts for the production of the collapse in the following manner:—

"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 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 occlude it. 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 a perfect collapse of the portion of lung usually fed with air by the obstructed bronchus."

In confirmation of the view, that obstruction of the bronchial tubes leads to collapse of the pulmonary tissue, we have the results of experiments performed by Mendelsohn and Traube. These experimenters introduced into the trachea of certain animals, small hard bodies, which they pushed down into the bronchial tubes as far as possible. The general result found on examining the lungs after death was, that the portion of lung connected with the tube which was obstructed by the introduced plug was red and void of air, in fact, in a state of collapse. In addition to these facts and observations, which may be adduced against the theory advanced by Laennec, it may be stated, that the seat of pulmonary emphysema, and that of obstructed bronchial tubes, is not the same; on the contrary, the two affections have altogether different localities. It has been shown by the researches of Gairdner, Lebert, and others, that pulmonary collapse and emphysema are frequently found existing together in the same lung; and the former author has so constantly seen the two affections associated together, that he has looked upon them as having the relation to each other of cause and effect. Hence he has sought for an explanation of the production of emphysema in the altered relation which the collapsed lung bears to the cavity in which it is placed, as compared with its relations in a state of health. His opinions may be summed up as follows:—Adopting the view that emphysema is produced by the force of the inspired air acting on the walls of the air-sacs, he considers the disease in the light of a complementary lesion, depending upon the fact that a portion of the lung has become *diminished in bulk and incapable of distension*. In consequence of this condition of the lung, which is found in pulmonary collapse, those portions of the organ which remain in a sound state, receive into them a larger quantity of air than usual, in order to fill the space previously occupied by the portion now collapsed: hence over-distension of the air-sacs and rupture.

Notwithstanding the very able manner in which the author of the above theory has supported his views, it appears to me that there is an error in the position he has assumed which tends to invalidate his conclusions; and that the exclusive doctrine of the production of emphysema, which he has advocated, will not bear the test of strict clinical investigation. That when one portion of a lung is collapsed, it necessarily follows that the sound portions of the organ will expand beyond their usual size, so as to fill the space previously occupied by the collapsed lung, and so to dilate the thoracic cavity to the same extent as before, appears to me to be opposed to what we know of the mechanism of respiration. During respiration, the chest expands to make room for the dilating lungs, and it will only expand to the extent required by the amount of air which enters the lungs. The air is drawn equally to all parts of the lungs; neither the muscles of the chest, nor the lungs themselves, have any power to *determine* the air towards any one part of the organs more than to another. No external force exists which can accomplish such a result; and therefore it seems difficult to understand how a diversity of currents could be produced in different portions of the lungs, so great as to lead to an over-distension of some parts whilst others remained normally dilated. That, when collapse of a portion of the lung takes place, so that no air can be received into it, the same quantity of air, as previously entered, finds its way into the chest so as to dilate it to the same extent as before, appears to me extremely doubtful; and such a view is, in my opinion, opposed to other pathological facts we witness in connexion with the lungs. But if we admit that this view is correct, and that in proportion as some parts of the lung collapse, others become more than normally dilated so that the chest reaches its previous state of expansion, then the air would become diffused throughout the whole of the lung remaining sound, and not driven or drawn to any particular locality. The consequence of this would be, that an increased small dilatation of every part would compensate for the want of action of the small collapsed portion. There would, under these circumstances, be no special strain on any particular part of the pulmonary tissue,—no rush of air to one part more than to another. But, further, if, as the result of pulmonary collapse, any portion of the lung becomes abnormally distended so as to lead to the production of emphysema, it appears to me that it ought to be those parts which lie in contiguity to the collapsed tissue. But we do not find this to be the case; on the contrary, the collapsed portions are most frequent in the posterior and lower parts of the lungs, the emphysematous at the apex and along the margins. It is true, that Dr. Gairdner has found in some cases patches of emphysema lying side by side with the collapsed tissue, and I have seen the same thing myself; but, as a rule, the two affections have, as I before stated, different seats.

From the views I have expressed above, it appears to me very difficult to account for the production of emphysema by a forcible distension of the lung as the result of an inspiratory act; and especially when we consider the amount of distension which the lungs will bear when in a healthy condition without any rupture of the air-sacs taking place, a distension, probably, far greater than they undergo in those cases of disease where one lung, or part of one lung, takes an increased action to compensate for the want of action of a disabled portion.

I pass now to consider briefly the expiratory theory of the disease. Are there circumstances in ordinary or forced expiration which have a tendency to produce a distension of any part of the lung, and consequently to lead to the production of emphysema? With reference to the act of ordinary expiration, we may safely say there are none; but in regard to forced expiration, it appears to me that such circumstances do exist.

It has been urged as an objection to the theory we are now considering, that the expiratory act is mechanically incapable of producing distension of any part of the lung, and that the air-sacs are emptied by an uniform pressure of the thoracic walls upon the whole pulmonary surface. This objection only applies to the ordinary act of expiration; and it is undoubtedly true that the lungs then undergo equable pressure on all parts, and that there is no tendency for the air to be forced towards, or retained in, any particular part of the pulmonary substance; but the argument loses its weight when we come to apply it to the act of forced expiration, such, for instance, as that of coughing. The effect which is produced on the lung by the act of coughing has been pointed out by Dr. Jenner, in a paper read before this society, and the remarks he has there made entirely accord with what I have myself observed. If we examine a person whose chest is exposed during the act of coughing, we see a distinct bulging produced above the level of the clavicle; in fact, it is clearly shown that the air is forced upwards by the respiratory act, and forcibly distends the upper part of the lung. Percussion of the tumour, formed as I have stated, yields a resonant sound, which becomes almost tympanitic if the lung be in an emphysematous state. Now, if we examine the anatomical arrangements of the walls of the chest, we have a ready explanation of the phenomena to which I have just alluded. The lateral and inferior walls of the thoracic cavity are strong and resisting, and by their muscular action and their elasticity they assist *actively* in expiration; further, the contraction of the abdominal muscles forces upwards the diaphragm, and this more especially in violent expiration. The part of the thoracic walls which is the weakest, and which

offers the least resistance, is that which separates the cavity of the chest from the region of the neck. We there find a fibrous structure, a strong fascia, in fact, connected externally with the first rib, and internally blending with the cervical fascia as it passes down into the chest. This plays no active part in the expiratory process, and offers no active resistance to the distension of the lung. From this peculiar arrangement of the walls of the chest, and from a consideration of the action of those muscles which are concerned in expiration, it appears to me that during violent expiratory efforts the lungs *must* be unequally compressed, and that air must be driven first to those parts of the lungs where the walls are least resisting; and secondly, to those portions which contain the least volume of air.

I have shown above that the apices of the lungs are the parts covered by the least resisting walls; and it will at once occur to all that the parts which contain the least volume of air, are the anterior bodies and the margins of the bases. These parts are not only the thinnest, but they are also out of the direct line of strongest pressure which the lungs undergo in expiration. Violent expiratory efforts are chiefly made with the abdominal muscles, and the most powerful agents are the recti; the contraction of these muscles forcing upwards the abdominal viscera and the diaphragm, produces the greatest amount of compression at the base of each lung; the air is consequently driven upwards in a strong current. There being no corresponding force acting at the upper part of the chest on the apex of the lung, this latter is not emptied; on the contrary, it becomes forcibly distended by the upward current. Further, the strong currents of air from the central and basic portions of the lungs overcome those from the thin portions, and thus these latter, instead of being emptied, become, like the apex, forcibly distended. Dr. Jenner supposes that the cartilaginous portions of the thoracic walls are somewhat yielding, and thus accounts for the production of emphysema along the borders of the lung. This explanation seems to me doubtful, and the one I have given as far more probable.

Again, I may refer to the phenomena which were witnessed in the case of M. Groux, who was over in this country some years ago, and made a tour of many of the metropolitan and provincial towns. In this case a fissure of the sternum existed which allowed of some of the movements of the heart being observed. Those who examined M. Groux will recollect that, during a violent expiratory act, the lung of one side came forward in the upper part of the fissure, and formed a distinct elongated tympanitic tumour; no such result taking place during inspiration. Whatever influence this fact may have on us with reference to the expiratory theory of emphysema, it tends, at any rate, to show that wherever there is a weak part, an absence of compressing power, in the thoracic walls, the lungs will there undergo distension during forced expiration.

The facts and arguments I have adduced seem to me to prove that the objection to the expiratory theory, on the ground that the expiratory act is mechanically incapable of producing distension of any part of the lung, is of an untenable character, and without expressing my own attachment to any exclusive theory of the production of emphysema, I may remark that my observations of a considerable number of cases, have led me to conclude that by far the most frequent cause of the disease is to be found in the cough which is attendant on pulmonary catarrh, or some other affection of the bronchial tubes, such as pertussis, etc. The fits of coughing caused by the repeated attacks of bronchial inflammation must, it appears to me, so react on the pulmonary vesicles which are most liable to distension as to produce after a time their dilatation and rupture, and I the more incline to this view of the production of the disease from knowing how difficult it is to produce artificial emphysema by inflation of the lungs, even after death. It may be objected to this view that emphysema should be more frequently found as a sequence of pleurisy or pneumonia. We know that in some cases of inflammation of the lungs partial emphysema *is* produced. I have seen this when the emphysema was apparently quite recent, and had, probably, been produced during the progress of the pneumonia. Such a result, however, only rarely follows, and I think we have an explanation of the fact in the character and short duration of the cough attendant on this disease as well as on that of pleurisy.

Again, it appears to me that the frequency with which we meet with emphysema in tubercular lungs favours the view I am taking. The deposition of tubercles in the pulmonary tissue filling up the air-sacs in the same way as a pneumonic exudation, with the subsequent and gradual contraction of the walls of the chest, presents no condition favourable to the formation of emphysema except the cough, which is usually so important a feature of the disease.

M. Guillot has collected a series of cases, to which I have alluded in a former part of this paper, illustrating the effect of long-continued spasmodic cough in producing emphysema. These cases are recorded, with some very excellent observations, in the *Archives Générales de Medecine* for 1853. They are fifteen in number; the subjects of them were infants who were for the most part affected with pertussis, but in all of whom long-continued spasmodic cough was a very prominent symptom. Death took place in all, and the *post-mortem* examination revealed the existence to a very considerable extent, of what M. Guillot describes as sub-pleural emphysema, with, in some instances, extravasation of air into the areolar tissue of the mediastinum, and even of the neck. No mention is made of pulmonary collapse in these cases. It appears to me that M. Guillot has satisfactorily established in the instances he has recorded, the connection between the violent spasmodic cough and the pathological results above alluded to; and that they cannot be considered as bearing to each other any other relation than that of cause and effect.

The existence of the various theories which have been advanced of this disease afford the strongest possible proof of the necessity of a more close investigation of its nature. I have stated my belief in a former part of this paper that in some cases a degeneration of the pulmonary tissue takes place, and leads to the formation of the disease. Cases of lobar emphysema are not unfrequently seen, where after death the whole of both lungs is found involved in the affection; but where there is no appearance whatever of collapse of the pulmonary tissue, or if any, only to a very partial extent. It may be said that in such cases the collapse was of the diffused kind, and that recovery from that condition has taken place. Such an explanation appears to me very doubtful, and it might fairly be asked, since every part of the lung is diseased, how the emphysema was produced in the collapsed portions.

Another circumstance which favours the view that there is some condition of the lung tissue, some degeneration, which predisposes to, and precedes the formation of, emphysema, is its hereditary nature. This subject has been investigated by Jackson, and he has obtained the following results:—

1. Of *twenty-eight* individuals attacked with pulmonary emphysema, *eighteen* had had their father or mother attacked with the same affection, and several of these had succumbed to it during its course. In some instances the brothers and sisters had also been attacked by the same affection.

2. Of *fifty* individuals exempt from emphysema *three* only had had their parents attacked with that disease.

Again, it has been shown by the researches of the same author that hereditary influence is much more marked in cases of emphysema which may be traced back to an early period of life, than in those where it has supervened at or after adult age. Of *fourteen* individuals suffering from the disease, and whose symptoms had existed from an early period, one or other of the parents of *all* had been *asthmatic*; whilst of fourteen others who had been attacked at a later period of life two only were born of parents who had died of a similar affection.

Transactions of Branches.

SOUTH-EASTERN BRANCH: ROCHESTER, MAIDSTONE, GRAVESEND, AND DARTFORD DISTRICT MEETINGS.

ON CERTAIN FORMS OF UTERINE HÆMORRHAGE OCCURRING AT OR NEAR THE FULL TERM OF GESTATION.

By John Armstrong, M.D., Gravesend.

[*Read October 26th*, 1860.]

There is no single part of obstetric practice which claims the attention of the practitioner more forcibly than uterine hæmorrhage in its varied forms; and upon a correct appreciation of its varieties, and the treatment suitable for each, must hang the life of his patient. Few men who have had large practices as accoucheurs have failed to witness those most distressing cases where their patient has died before their eyes, notwithstanding their utmost skill and attention. At other times, the practitioner has had, on reviewing his treatment, or from some subsequent light thrown on the case, to regret that he had not adopted a different course. It may indeed be stated, judging