THE BRADSHAW LECTURE ON PNEUMOTHORAX. ✓

Delivered at the Royal College of Physicians on August 18th, 1887.

By SAMUEL WEST, M.D., F.R.C.P.,

Assistant Physician to St. Bartholomew's Hospital; Physician to the City of London Hospital for Diseases of the Chest, Victoria Park; and Physician to the Royal Free Hospital.

By appointing me, Sir, to deliver this lecture, you have paid me an honour which I am proud to acknowledge; and although, for the moment, the feeling of honourable distinction is lost in one of responsibility, still the confidence which your judgment always inspires relieves some of the anxiety I feel lest my ability should not prove equal to the task you have imposed upon me.

I have chosen Pneumothorax as the subject of my lecture—a subject so large, however, that I cannot undertake to deal with the whole of it within the time placed at my disposal, and I intend to limit my remarks to those parts which present especial interest or which call for further elucidation.

Of the causes to which pneumothorax is due, phthisis is the chief. This is the primary lesion found in at least 90 per cent. of all cases. When from the remaining 10 per cent. are excluded those cases in which pneumothorax is the result of gangrene or acute inflammatory destruction of the lung, or the consequence of injury to the lung or walls of the thorax, the percentage of the other assigned causes becomes very small; 2 or 3 per cent. perhaps will include them all. It would be unprofitable to spend time in considering most of these groups, and I wish to refer only to two: first, that in which pneumothorax has occurred in apparently healthy persons; and, secondly, that in which the pleural cavity has been laid open by injury.

Many cases have been recorded, especially in recent years, of pneumothorax in the apparently healthy; but, as in these recorded cases recovery seems always to have taken place, there has been no opportunity of proving or disproving conclusively the existence of a pulmonary lesion. The question is thus raised whether it is possible to rupture the healthy lung by any force which respiration can bring to bear upon it. To this question an affirmative answer must be given. The violent paroxysms of whooping-cough and the straining of parturition afford clinical evidence that the lung may be ruptured by expiratory efforts. The condition, however, which is commonly produced is not that of pneumothorax, but that of subcutaneous emphysema.

A careful experimental study of this subject has been made by Dr. Champneys (*Med. Chir. Trans.*, vol. lxx, and *Artificial Respiration in Stillborn Children*, 1887) upon the lungs of infants. When the lung is over-distended and gives way, the air first makes its way beneath the pulmonary pleura, stripping it easily off for some distance from the surface of the lung. It then passes along the root of the lung to the mediastinum, and following thence the course of the cervical fascia it reaches the subcutaneous tissue of the neck, whence it may spread over the whole body. If the pleura give way, the place of rupture is to be found usually near the root of the lung, but although pneumothorax did now and then arise in this way, the usual result was that already described, namely, emphysema of the mediastinal tissue and of the neck. Sometimes by artificial respiration after tracheotomy the air has taken the reverse direction, and, tracking downwards from the tracheotomy incision along the deep cervical tissue, has reached the mediastinum, or even the subpleural tissue, and occasionally the pleura has been ruptured and pneumothorax produced.

These experimental observations are confirmed by clinical experience. Dr. Champneys records 27 necropsies (*loc. cit.*) after tracheotomy. Mediastinal emphysema was found in 3, and in two of these pneumothorax was present. The pneumothorax in one of them was double, and the cause of death. In 82 necropsies (*loc. cit.*) at the Children's Hospital mediastinal emphysema was found in 5, but pneumothorax in none. Wilks and Moxon (*Lect. on Path. Anat.*, 2nd Ed., 1875, p. 308) mention 2 cases of pneumothorax after tracheotomy, and connect it with mediastinal emphysema. Dr. Angel Money (*Med. Chir. Trans.*, lxxvii, 101), examining 28 similar cases with especial reference

to this point, found mediastinal emphysema in no less than 16 and pneumothorax in 2, but in both of these the pneumothorax was associated with mediastinal emphysema. In most of these cases artificial respiration had been performed.

It seemed desirable, considering the delicate texture of the infantile lung, to repeat these observations upon the adult, and I did this, with the result of confirming in the main the conclusions at which Dr. Champneys had previously arrived. I noticed also in my experiments the same important and curious fact to which Dr. Champneys has referred, namely, the difficulty of obtaining bursting pressure by means of water, owing to the permeability of the lung to water while it is still air-tight. The lung seemed almost like a sponge, so that the water flowed from it with such readiness when distended, that the pressure could not easily be maintained.

The pressures at which rupture took place varied much in different cases. In four cases it reached two and a half inches of mercury, in one two, and in another four; so that what Dr. Champneys found in infants is true also in adults—that the normal resistance of the lung is widely different in different persons. My own observations were made upon the lungs after removal from the body, and the bursting pressures thus obtained are no doubt much below those which would be required when the lungs are *in situ*. In the only case in which I was able to attempt this experiment upon the dead body, the air did not appear in the neck until a pressure of nearly eight inches of mercury was registered, and pneumothorax was subsequently found on both sides. This experiment agrees with the statement made by Hutchinson, that the cells of the lung can resist a pressure of three to nine inches of mercury when expanded as in health.¹

I append the actual notes made at the time of experiment, and I wish to acknowledge with thanks the assistance I have received from Mr. Willoughby in this work.

1. Left lung; adult man, about 50. Septicæmia after osteo-myelitis of femur. Lower lobe good deal collapsed, especially on posterior and lower part; no consolidation, no old adhesions. Lung expanded readily in all parts except where there was collapse. The pressure was raised to two and a half inches of mercury, and the lung then burst at the posterior and middle part of upper lobe, about two inches from root of lung. When rupture occurred the pleura was suddenly stripped off over a space about the size of a five-shilling piece, and then it gave way, the hole measuring about one-eighth of an inch. The tube was next tied into the bronchus of the lower lobe; as the pressure rose, some of the collapsed portions expanded but not all. The lung finally ruptured suddenly on the posterior border, where the lung lies at the side of the spinal column. Here the pleura was suddenly stripped off the lung for the whole length of the lobe (six inches), and this gave way with a rent about half an inch long. On opening the raised part of the pleura, the lung was found to be ruptured in several places; for the most part the holes were minute, but in one, which corresponded with a small portion of collapse, the hole was nearly one-eighth of an inch in diameter.

2. Left lung; adult man, about 50. Lung good deal collapsed; small fibrous nodules felt through the pleura, scattered throughout the lung; an old fibroid cicatrix at the apex. The lung distended readily (even the collapsed portions), and resisted a pressure up to four inches of mercury. At this pressure it gave way in the posterior and middle portion of the upper lobe; the pleura stripped off for a space about the size of a shilling, and then burst by a small aperture. The tube was fixed in the lower lobe, and the pressure was now raised to nearly five inches of mercury before any considerable rupture occurred. This took place at the posterior border, close to the root of the lung, but the pleura did not strip off as in the last case. Before this pressure was reached, many small patches of interstitial emphysema developed all over the surface of the lobe, but none of them burst. On section a fibrous nodule was found in the apex, and small nodules (so-called "peri-bronchitis") scattered throughout both lobes.

3. Right lung of a boy. The cannula was tied in the bronchus and the lung blown up; at a pressure of two and a half inches of mercury it burst. The air came out of minute apertures around the inferior bronchial vein and along the posterior and mediastinal borders; some air seemed to come from the vein itself. The inferior lobe burst first, but there was a large slit accidentally made in the upper lobe before the experiments began. Where the lung burst, except close to the root, the pleura was first raised. It was not stripped off in large pieces, but in pieces varying from a large pea downwards in size.

4. Left lung of male, about 55. Died after gastrostomy for stricture of œsophagus. Upper lobe particularly emphysematous, lower lobe not so much so; otherwise healthy. Air was forced into the lower lobe only as the upper one was cut. At a pressure of two inches of mercury the pleura suddenly was slightly raised in two or three places near the vein, and air came up also through the vessels. The holes were very minute in the lung-substance, and the pleura raised was about the size of a pea in each place.

5. Left lung of male, aged 66, who died after cancer; stomach, emphysematous, but otherwise healthy. At a pressure of two and a half inches of mercury the pleura was suddenly raised up over an oval space, longest diameter two inches, shortest three-quarters of an inch. The part raised was the part covering the lower lobe where it was opposite the upper (that is, between the two lobes), and near the root.

6. Left lung of male, about 50, who died suddenly from bursting of aneurysm of aorta, situated where thoracic becomes abdominal aorta. The aneurysm burst into the right pleura. Air was forced in, and at a pressure of two and a half inches, a part of the pleura, one inch and a half long and one inch wide, was suddenly raised from the lower lobe close to the root. At the same time air came up through both artery and vein. The hole in the lung itself could not be seen; the hole in the pleura, which was raised, was a quarter of an inch in diameter.

¹ The causes of death in the cases from which the lungs are taken were not pulmonary, but in some the lungs had undergone a good deal of post-mortem change.

A pressure of eight or nine inches of mercury is far beyond anything that even an adult can produce by expiratory effort, and it is clear, therefore, that it can only be in cases where the normal resistance of the lung is greatly reduced, that in the absence of any disease of the lung pneumothorax can be produced. The lung is therefore capable of resisting in most cases the highest pressure that the most violent expiratory efforts can bring to bear upon it, and the question may well arise whether in those cases in which the lung has given way, the absence of a local lesion can be, without hesitation, assumed. However this may be, it is clear that when rupture of the lung occurs the result is usually subpleural, mediastinal, and cervical emphysema, and only very rarely pneumothorax. This conclusion clinical observation abundantly confirms. Although every text-book speaks of emphysema among the complications of whooping-cough, pneumothorax is by most writers not even mentioned, and so far as parturition is concerned, Dr. Matthews Duncan allows me to state that although emphysema is by no means uncommon, he has never yet seen a single case of pneumothorax produced during parturition.

If, now, we turn to the cases recorded in which pneumothorax has occurred in the apparently healthy, it is remarkable that in so many there is a complete absence of any history of forcible respiratory effort. In some the pneumothorax developed actually during sleep; in others when the patient was sitting still or walking quietly. In a few it occurred during effort, but in many of them the efforts could not for a moment be compared in violence to those of parturition or whooping-cough. It is therefore probable that in these cases the lungs were not really sound.

This conclusion raises another question of importance. If the lungs were not sound is it not remarkable that recovery should so often occur? In the first place, it is well known that lesions may exist in the lung, which no skill in physical diagnosis may be able to detect, and that such a lesion is capable of producing pneumothorax the following case provides proof:—

A man, aged 56, was admitted into St. Bartholomew's Hospital with some shortness of breath; and, on examination, he was found to have pneumothorax of the right side. He gradually sank and died; and, on the *post-mortem* examination, the only lesion in the lung to be found was a small cavity with caseous walls, the size of a hazel-nut, near the root of the lung. This had ruptured into the pleural cavity.

But it might be objected that pneumothorax due to the rupture of a phthisical cavity would hardly recover. Such an assumption is not warranted by clinical experience.

Martha L., aged 19, was admitted into the Chest Hospital, Victoria Park, with phthisis of the right apex. About a fortnight later pneumothorax was discovered on that side. She had had an indefinite attack of sickness, to which no importance had been attached a few days before I saw her. The signs of pneumothorax were characteristic. No fluid developed; but, after a few days, the air began slowly to disappear, and in about nine weeks had completely vanished. The lung had re-expanded, and the breathing was audible as before over the whole side.

While the pneumothorax on the right side was getting well, the mischief in the opposite lung was rapidly advancing, and the patient was sinking of consumption. She was removed from the hospital against advice, and I heard that she died a few days later. The signs of pneumothorax had completely disappeared for more than a fortnight before her departure from the hospital. In such a case there can be no reasonable doubt of the phthisical nature of the lesion, and yet recovery was complete.

In another case in which complete recovery without effusion took place (*Clin. Soc. Trans.*, xvii, 56), I have repeatedly examined the chest with great care, and have always failed hitherto to obtain any definite signs of local lesion. The patient has, however, had three or four attacks of slight hæmoptysis, and I think the probabilities are in favour of a phthisical lesion. It is now four or five years since the patient had pneumothorax, and he continues in his usual health.

From these facts, therefore, that lesions may exist in the lung, which cannot be detected, and that recovery from pneumothorax may be complete even when the lesion is presumably phthisical, supported as it is by the experimental and clinical evidence already brought forward, we are led to the conclusion that in many, if not all, of the cases occurring in the apparently healthy, a lesion, which may even be of a phthisical nature, has really been the cause of the affection.

I turn now to the second class of cases to which I wish to refer, namely, to that in which the pleural cavity has been laid open by injury. The mechanism of pneumothorax in these cases is thus described: "The elastic pulmonary tissue is always to a certain extent

on the stretch. It is always, so to speak, striving to pull asunder the pulmonary from the parietal pleura, but this it cannot do, because the air can have no access to the pleural cavity. When, however, the chest ceases to be air-tight, and air is introduced into the pleural chamber, the elasticity of the lungs pulls the pulmonary away from the parietal pleura, and the lungs collapse" (Michael Foster, *Phys.*).

This quotation expresses concisely the physiological doctrine of the day. It is not, however, completely correct, for the chest may cease to be air-tight, and yet air may not enter into the pleural cavity. If, however, air does penetrate between the layers of the pleura, the lungs collapse, as they are stated to do, by virtue of their own elasticity.

Experiment upon animals, as well as observation of disease and injury in man, establish the truth of this latter statement, but there are not a few instances in which, in spite of the pleura being opened, collapse of the lung does not occur, and air does not therefore enter the pleural chamber. In considering the whole question, these instances must not be disregarded.

There are two great classes of such cases; first, that in which the pleural cavity has been laid open by an external wound; secondly, that in which, without any external wound, a communication between the lung or some other air-containing viscus and the pleura has been made by an internal lesion.

In the first class of cases, where the side of the chest may have been laid open by a tear or cut, the lungs are sometimes seen torn and bleeding in the floor of the wound, but still in close apposition to the walls of the thorax.

A very striking case of this kind is recorded in the *St. Bartholomew's Hospital Reports* for 1876 (Surgical Appendix to the Statistical Tables). A wood-turner, aged 33, stabbed himself in the head, chest, and abdomen with a chisel. The wounds of the head and abdomen were not very severe, but the wound in the chest, situated on the left side between the fifth and sixth ribs, was three inches long. Through it the lung could be easily seen wounded and bleeding. This wound in the chest was closed with strapping and oiled lint. Two days later, there was emphysema, but this soon cleared up, and in four weeks the patient recovered completely without any further trouble.

The second class of case, in which, without any external wound of the thorax, the lung is torn and the pleural cavity opened, is still more interesting, as it is also much more frequent. Simple fracture of the ribs is one of the commonest of surgical injuries. Pneumothorax, however, in these cases is extremely rare, and yet it is clear that both layers of the pleura are torn across and the lung opened, from the fact that air has made its way into the subcutaneous tissue, producing surgical emphysema.

It is hardly necessary to bring forward proof of such a well-known fact, but it may be useful to quote one case in point, the most remarkable I have myself met with. I made the *post-mortem* examination upon the body of a woman who had been crushed by a waggon. There was no external wound beyond bruising, but several ribs were fractured, and a considerable amount of subcutaneous emphysema developed. The lungs had been severely injured, it was evident, for the patient had expectorated a good deal of blood. On the necropsy, several ribs were found fractured, and the lung extensively torn over an irregular area about two inches in diameter; but in spite of this, the lung was everywhere in apposition with the chest-walls, and there was a complete absence of pneumothorax. The pleural cavity was free from adhesions. The case puzzled me much, and having no better explanation to offer, I assumed that the lungs must have become hitched over the protruding and jagged ends of the bones, and have been in this way prevented from collapsing.

Without bringing further evidence forward, I am expressing, I believe, only the common experience when I say that with simple fracture of the ribs pneumothorax is very rare, and surgical emphysema very common. This is a great difficulty, and various solutions have been proposed. It has been asserted that in such cases the pleural cavity has either been completely obliterated by previous disease or that adhesions rapidly formed round the wound have sealed the pleural cavity. This theory is *a priori* improbable. It has no clinical facts to support it, and it is disproved by *post-mortem* examination. It is by others asserted that air does enter the pleura, but that it is rapidly absorbed again, so that its absence after death is accounted for. When so careful a clinical observer as the late Dr. Hilton Fagge (*Med. i*, 938) endorses this view, the need of reconsideration of the whole question is evident. These explanations are unsupported by clinical observation, and are one and all disproved by *post-mortem* examination. The fact, therefore, that the pleural cavity may be laid open, and that yet collapse of the lung with consequent pneumothorax may not occur, must be accepted and an explanation sought.

The normal elasticity of the lung was calculated by Donders (Henle, in Pfeiffer's *Zeitschr.*, iii, 1853, 293) to be equivalent to 7 millimètres of mercury. Michael Foster rates it somewhat lower—namely, at 5 millimètres (*Cycl. Anat.*, v, 1058). Hutchinson agreed with the higher estimate, as the result of direct determination upon the lungs of two criminals immediately after death. If, for the present purpose, the higher estimate be accepted, it follows that, when the lung does not contract after the pleura is opened, it must be kept on the stretch by some force greater than 7 millimètres of mercury; and there seems to be no place for such a force to exist, unless it be in the pleura itself; and, if here, it is probably to be found in the cohesion between the two serous surfaces. A theory of this kind seemed to admit of easy investigation, and it was put to the test in the following manner.²

Two discs made of mahogany were taken. They measured 2 inches (5 cubic centimètres) in diameter, were smooth on the surface, and had a groove cut round the margin. Over these pieces of stomach were tightly stretched, with the peritoneal surfaces outwards, care being taken to select pieces which would lie as smooth and even as possible. These two discs, so covered, moved very freely over one another; but when they were pulled straight asunder, considerable force was required to separate them.

In order to estimate this force one of the discs was connected with the arm of a balance, and, while the other was fixed fast, weights were placed in the scale-pan until the two were pulled apart. I found in some of my experiments that about 325 grammes were required. It was thought that the force which kept these discs together might have been atmospheric pressure, and in order to eliminate this two similar discs were taken, perforated in the centre by a hole a quarter of an inch in diameter. Corresponding with this hole in the lower disc a cut half an inch long was made completely through the membrane. The retraction of the membrane converted this slit into an oval hole, and in this way, when the discs were placed in apposition, a free communication was provided between the air and the space between them. I found, however, that the same weight was still required, namely, 325 grammes, to separate them. A similar slit was made next in the upper disc and membrane as well, but this, too, made but a few grammes difference.

The result of these observations is to prove that there is some force other than atmospheric pressure³ by which these two smooth surfaces are held together, and without using the term in too technical a sense, I may speak of it as cohesion.

The next point to be determined was this—Was this cohesion greater or less than the elasticity of the lungs? Accepting, for the sake of the calculation, the higher estimate made by Donders, namely, 7 millimètres of mercury, it was found that a column of mercury of this height, with a circular base of 5 cubic centimètres in diameter weighed 185.6 grammes.⁴ The weight required to separate the discs was 325 grammes, so that there was an excess of about 140 grammes in favour of the force which kept the discs together. If this weight be reckoned out in terms of mercury, it is equivalent to 12.5 millimètres as opposed to 7 millimètres, giving thus an excess of 5.5 millimètres.

Having thus roughly established the correctness of the theory, I endeavoured next so to modify the experiment as to imitate somewhat more closely the conditions which exist in nature. I took a bell-jar, the mouth of which was about 3 inches in diameter. The top was bored so as to provide for the insertion of a perforated cork, into which a glass tube could be fitted and communication made in this way between the external air and the interior of the jar. Over the mouth two pieces of stomach were stretched, with the peritoneal surfaces in contact. By exhausting the air the membranes were made to bulge inwards. They remained, of course, in contact. A slit was now carefully cut through the outer membrane, forming an oval hole about half an inch long and a quarter of an inch wide. This hole provided for the access of air between the membranes, just as in the case of the discs. When the air was now exhausted, the two membranes still remained in contact at first, and it was only after considerable bulging that they separated and air passed between them. A manometer was now connected with the interior of the jar, so that the pressure could be measured. By means of a special modifica-

tion of the mercury manometer, the reading could be converted at once into its equivalent in inches of water.

7 millimètres of mercury = 3.8 inches of water.

12.5 " " " = 6.8 " " "

The jar was now exhausted until the negative pressure indicated was 3.8 inches of water. At this pressure, the equivalent of the elastic tension of the lung, the membranes remained in contact. The exhaustion was continued until a pressure of 6.8 inches of water was reached, but still the surfaces remained in contact. The pressure could even be slowly still further reduced to 10 or 12 inches of water before the surfaces separated and air passed between them. When the pressure was relieved, they again passed into contact, often so completely that the original experiment could be repeated. More often, however, unless the membranes had been very carefully stretched, a few small bubbles of air remained, which had to be pressed out with the finger to make the contact perfect.

It is not to be wondered at that, with the bell-jar experiments, higher values were obtained for the force of cohesion than with the discs. It is clear that, although I had discs of 5 centimètres in diameter, covered with membrane, the actual surface of contact was something less than this, and the results obtained were therefore below the real value. While this value was, in all probability, too low, I accepted for the purpose of comparison the highest estimate of the elasticity of the lungs, so that on both accounts the difference arrived at was less than the real difference.

With the bell-jar experiment certain precautions are necessary. The two pieces of membrane must be such as will stretch uniformly. This makes the parietal pleura itself not a very suitable membrane for experiment. It is much thinner in the parts corresponding with the ribs than it is in the parts corresponding with the intercostal spaces. The thinner parts yield most on exhaustion of the jar, so that the surface is thrown into furrows, and the tension on bulging differs in consequence in different parts. This affects greatly the results. The stomach is more suitable, but, even with this, care must be taken in the selection of the parts used, for when stretched the large blood-vessels act as bands, and if they be included folds are also produced.

The membranes also must be so stretched as to have as nearly as possible the same tension in each. If this be done the surfaces may remain in contact at much greater negative pressures; it may even be at 2 inches of mercury before separation is effected. This result was so much in excess of what I anticipated that I sought for some explanation of it. While the membranes were on the stretch, therefore, at this pressure, I gently raised the edges of the slit with a blunt probe, so as to admit a little air between the membranes here, and I found then that the two layers separated at once. I repeated the experiment at different pressures. As the pressure was reduced more and more, the ease with which air passed between the layers when the edges were raised became less and less, until when the pressure was below 12.5 millimètres of mercury, that is, below the pressure at which the force of cohesion had been estimated, in spite of the edges being freely separated by the probe, the membranes did not separate, but directly the outer layer which had been lifted up was allowed to fall, it passed again into close contact with the other, and the air which had been admitted was forced out. This experiment appears to me to be conclusive.

The figures given above are actual numbers obtained in particular experiments. The numbers differ a good deal in different observations, their variations depending upon differences in the stretching of the membranes, and in the condition of their surfaces at the time of experiment. Without laying, therefore, any special stress upon the actual values obtained, these observations establish, I consider, the existence of a force between the pleural surfaces much in excess of the elasticity of the lungs, and sufficient therefore to maintain the lungs in apposition with the walls of the thorax, even when air has free access to the pleural cavity.

Pneumothorax can, therefore, be no longer regarded as a condition to which there is an inherent tendency in the healthy body, but, on the contrary, as a condition brought about by the forcible separation of the pleural surfaces, and in this respect exactly analogous to the distension of the subcutaneous tissue, which occurs in surgical emphysema; and, considering the frequency of subcutaneous emphysema and the rarity of pneumothorax in simple fracture of the ribs, to which reference has been made, it would seem to follow as a corollary that the force required to distend the subcutaneous tissue must be less than that required to separate the layers of the pleura. Pneumothorax, therefore, so far from being, as it is commonly regarded, a passive process, and inspiratory in origin, is really expiratory in its initial stage, and requires an active force to produce it; and surgical

² These experiments were described in a paper communicated to the Royal Society on May 23rd, 1887.

³ Cohesion between two smooth glass plates is independent of atmospheric pressure.—*Draper's Medical Physics*.

⁴ A disc of 5 centimètres in diameter has an area of $5^2 \times 0.7854 = 19.635$ square centimètres. Seven millimètres of mercury upon the square centimètre = 9.45 grammes. A pressure of 7 millimètres of mercury upon a disc of 5 centimètres = $19.635 \times 9.45 = 185.6$ grammes.

emphysema, so far from implying, as Fagge asserts, the necessary antecedence of pneumothorax, may in reality be a protection against it, the air making its way in the direction of least resistance, beneath the skin rather than between the pleural surfaces.

It is evident, further, that where the pleura is laid freely open, so that the air passes away without hindrance on expiration, the liability to pneumothorax will be still further reduced, for the expiratory pressure requisite to overcome the cohesion of the pleural surfaces will be absent.

By means of the discs I have been able to investigate some of the conditions which modify the force of this cohesion. In the first place, the surfaces must be absolutely smooth, for any roughness prevents close contact. This is one of the reasons why stomach well stretched over the discs is more suitable than pleura, the mucous membrane forming a cushion into which any little unevenness will sink. I have frequently found that the same pieces of pleura which would not give good results when spread directly on the discs answered admirably as soon as they were stretched with a piece of stomach beneath them.

The surfaces in contact must be neither dry nor wet, but simply moist, as are the normal surfaces of the pleura. If dry, they will not cohere at all; if wet, the force of cohesion is greatly reduced. The cohesion is, however, not between the membrane and the surface of the fluid, for a disc floated on water, blood, or serum requires but little force to raise it out. What is essential appears to be an extremely fine film of fluid, a capillary film, if I may call it so, between the two surfaces.

It may be that the cohesion varies with the nature of the fluid; but, so far as I have gone at present, I do not find, with my rough methods of investigation, any perceptible difference between serum, blood, or water. The ready abolition of this cohesion by the presence of even a small excess of fluid is important, for there will be, on this account, less resistance to the entrance of fluid than of air into the pleura.

This force of cohesion has a general bearing in physiology, for it will exist wherever serous surfaces are in close contact; in the peritoneum therefore, and in the pericardium as well as in the pleura. In the peritoneum its tendency will be to keep the intestines partly distended, and in this way it will diminish resistance to the passage of its contents along the tube. It will be, perhaps, in the joints that one of the most interesting illustrations of its usefulness will be found, for it is compatible with the freest movement possible of the surfaces upon each other. It may, perhaps, prove to be one of the most important means by which the head of the femur is kept in the acetabulum, but it will come also into play even in the hinge-joints. Since the force appears to be directly proportional to the extent of surface of contact, it will continue to be the same so long as the extent of surface of contact does not vary, and in the hinge-joints therefore, so long as this is the case, the same amount of force will be exerted, whatever be the position of the joint, to keep the ends of the bones in place.

The effect of excess of fluid in diminishing or even abolishing this force is well illustrated by the looseness which develops in the joints when effusion takes place into them.

But to return to the pleura, with which we are now more especially concerned. The existence of this force of cohesion is sufficient to explain satisfactorily many of the clinical difficulties which present themselves, and which led me to institute these investigations.

Among the physical signs of pneumothorax, the chief interest centres in the variations of pleural pressure and their consequences. If the observations to which I have just referred be correct, it is clear that pneumothorax, in its initial stage, must be an active process. Some force will be required to overcome the normal cohesion between the two layers of pleura, and to separate them. This must be obtained by expiration; and pneumothorax, therefore, in its initial stage, is an expiratory process, and not essentially different in its production from surgical emphysema. As soon, however, as separation has been effected, the elasticity of the lung will come into play, and air will enter the pleura until its retractorility is completely satisfied, and, according to the observations of Donders (*loc. cit.*) this will continue until the lung occupies, in its collapsed state, not more than one-eighth of its original volume. Air, therefore, may enter or fluid collect in quantity enough to occupy the space thus left in the pleura without the development of any increased intrapleural pressure.

In considering the pleural pressures in pneumothorax, a sharp distinction must be drawn between the pressure during inspiration and that during expiration. The expiratory pressure will, of course, be always positive. Only when there is a large patent opening, so

that air can pass freely out, will the expiratory pressure approach to zero. It will, however, differ widely in different cases, depending upon the force of expiratory effort.

With the inspiratory pressure the case is quite different. Air will continue to enter through the opening into the pleura until the pressure within, in the position of the deepest inspiration, is equal to that of the atmosphere. When this point is reached, no more air can enter; and if, as usually is the case, the opening be valvular, so that no air can escape, the pressure on inspiration will remain at zero. If the inspiratory pressure now rises, some other cause must be at work, and in most cases this is found in the development of effusion, either serum or pus, and a positive inspiratory pressure may therefore be taken as indicative of the presence of fluid. The converse, however, is not true, for fluid may be present in considerable quantity as already stated, and yet the inspiratory pressure be not raised above zero. Under one condition only could the inspiratory pressure rise above zero without fluid—namely, when, after the closure of the opening, the body temperature had been raised several degrees, but as such a rise in temperature is nearly always due to the development of fluid effusion, a difficulty is not likely to arise.

The next point of interest is the respiratory oscillation—that is, the difference between the pressures on inspiration and expiration, measured, of course, during ordinary quiet, not forced, respiration. This has been variously estimated in health. [By Donders at $1\frac{1}{2}$ millimètre of mercury, by Hutchinson at 3 millimètres, by Adamkiewitz from 3 to 5 millimètres, by Hegel and Spehl at 3.6 millimètres (quoted Heynsius, xxix, 265; Pfliiger's *Archiv f. Physiol.*)] The majority of observers value it at about 3 millimètres of mercury, or about an inch and a half of water. This is found to be the respiratory oscillation in many cases of pneumothorax. It is, however, often in excess of this, especially when there is dyspnoea, the excess being due then chiefly to the rise of pressure on expiration.

There are two conditions under which the respiratory oscillation in pneumothorax is observed to be even less than normal. In the first the opening is widely patent, and air passes without any hindrance in and out of the pleura; in the other the opening is closed, and the inspiratory pressure is high. What is observed in this latter case is this: on first introducing the trocar, before air or fluid has been removed, the respiratory oscillation is small, perhaps not more than half an inch of water; but after relief of the tension, as the inspiratory pressure falls, the respiratory oscillation increases often much beyond the normal. The explanation of this is, I think, to be found in the effect produced by the high pressure upon the opposite lung. This lung, by the raised inspiratory pressure, has been partly collapsed, and the walls of the thorax have been allowed to expand, so that they are in the position of deep inspiration. The high expiratory pressure prevents the normal elastic recoil of the lung on that—that is, the unaffected—side. As soon, however, as the pressure is relieved, the elastic recoil comes once more into play, and the respiratory oscillation increases.

The following selected cases of pneumothorax with effusion illustrate the statements which I have just made.

CASE I.—Male, aged 22. Hydro-pneumothorax. Recovery.

First Paracentesis: Inspiratory Pressure = + $6\frac{1}{2}$ inches of water.
 Expiratory " = + $13\frac{1}{2}$ " "
 Respiratory Oscillation = $6\frac{1}{2}$ " "

The patient was an apparently healthy young man, of considerable muscular power. The high respiratory oscillation was due to dyspnoea, and depended chiefly upon the very high expiratory pressure. Forty ounces of serum were removed.

Second Paracentesis: Inspiratory Pressure = + $6\frac{1}{2}$ inches of water.
 Expiratory " = + 8 " "
 Respiratory Oscillation = $1\frac{1}{2}$ " "

The fall is observed chiefly in the expiratory pressure. Twenty ounces of serum were removed. From this time the patient rapidly improved, and left the hospital perfectly well. He has been frequently seen since, and he has been in active work for nearly three years. He appears in perfect health, and it is now almost impossible to say which was the side affected.

CASE II.—Male, aged 21. Hydro- (later pyo-) pneumothorax; death from rupture of an aneurysm of the pulmonary artery.

First Paracentesis: Inspiratory Pressure = + 1 inch of water.
 Expiratory " = + 5 " "
 Respiratory Oscillation = 4 " "

Twenty-eight ounces of serum were removed.

Second Paracentesis: Inspiratory Pressure = + $\frac{1}{2}$ inch of water.
 Expiratory " = + $2\frac{1}{2}$ " "
 Respiratory Oscillation = $1\frac{1}{2}$ " "

Thirty-six ounces of serum were removed.

Subsequently an abscess formed in the seat of puncture, which was found later to communicate with the pleura. A free incision became necessary. Some days after this the patient suffered from severe hæmoptysis, and blood passed from the incision as well. Death resulted from exhaustion consequent on the hæmorrhage, and, on *post-mortem* examination, a ruptured aneurysm of the pulmonary artery was found.

CASE III.—Male, aged 22. Hydro- (later pyo-) pneumothorax. Death. A large opening into the lung was found.

First Paracentesis: Inspiratory Pressure = +3½ inches of water.
 Expiratory " = +8½ " "
 Respiratory Oscillation = 4½ " "

Thirty-six ounces of serum were removed.

Second Paracentesis: Inspiratory Pressure = +1 inch of water.
 Expiratory " = +6 " "
 Respiratory Oscillation = 5 " "

Hardly any fluid was obtained.

Third Paracentesis: Inspiratory Pressure = - ¼ inch of water.
 Expiratory " = + ¼ " "
 Respiratory Oscillation = ½ " "
 Fluid not reached.

Fourth Paracentesis: Pressures the same.

Forty ounces of pus were removed.

The conditions were clearly entirely changed at the time of the last two paracenteses. The conclusion was drawn, from the small respiratory oscillation of a quarter of an inch only on each side of zero, that a large opening must have developed in the lung. The patient died shortly afterwards, and this was found to be the case.

CASE IV.—Male, aged 24. Pneumothorax with sero-purulent fluid. Death.

Inspiratory Pressure = +6½ inches of water.
 Expiratory " = +8½ " "
 Respiratory Oscillation = 1½ " "

Thirty-four ounces of sero-purulent fluid were removed.

CASE V.—Male, aged 30. Pyo-pneumothorax. Death.

Inspiratory Pressure = +1½ inches of water.
 Expiratory " = +2½ " "
 Respiratory Oscillation = 1 " "

Eighteen ounces of pus were removed.

Subsequently pus discharged itself at the seat of puncture, and an incision was made. The patient died shortly after.

CASE VI.—Male, aged 46. Hydro-pneumothorax.

First Paracentesis: Maximum Pressure = +9 inches of water.
 Seventy-four ounces of serum were removed.

Second Paracentesis:

Pressure on Ordinary Inspiration = +1½ inches of water.
 " Deep " = -1 " "
 Expiratory Pressure = +4 " "
 Respiratory Oscillation = 2½ or 4½ " "

No fluid was obtained.

The variations in inspiratory pressure showed that the opening from the lung was closed to inspiration as well as to expiration. The patient left the hospital greatly improved. He returned, however, about two months later with a discharging empyema. The side had fallen in greatly, so that the ribs were close together, and signs of phthisis had developed at both apices.

All the preceding cases confirm the statement made that, when the inspiratory pressure is positive, the presence of fluid may be inferred. As already stated, however, the converse is not true—namely, that, if the inspiratory pressure be not raised, fluid is absent. The following case illustrates this fact.

CASE VII.—Male, aged 31. Pneumothorax with effusion. On paracentesis the inspiratory pressure was zero, but 74 ounces of serum were removed. After this the fluid was gradually absorbed, and the patient left the hospital feeling well, although the fluid had not quite disappeared.

Certain results follow from the alterations in pleural pressure just described, the most important and characteristic being, of course, the displacement of organs, especially of the heart. This displacement, it is known since Dr. Douglas Powell's experiments, is not due to pressure, but to the retractile power of the lungs themselves, and therefore is not necessarily associated with a rise in intrapleural pressure; but it is clear that, when the pressure is positive, the displacement will be increased.

The heart, when displaced, is not, as was once supposed, rotated, but simply drawn or pushed bodily over. I append a drawing, for which I am indebted to my friend Mr. E. O. Bark, of Birmingham, a former resident medical officer at the Chest Hospital, Victoria Park.

In this it will be seen that the relation of parts is the same as when the heart is in its normal place; the only twisting or rotation which takes place—and it is but very little—is that necessitated by the curving of the ribs. The displacement is the same in kind, whether it be to the right or to the left side, and whether by air or by fluid.

E. M., female, aged 17. Phthisis, left pneumothorax. The chest was opened by an incision down the middle line of the sternum, which was sawn through, and the costal cartilages on the right side having been divided, the front wall of the right pleural cavity was removed, leaving the left side unopened. At the upper angle of this opening was a large, rounded, tense tumour. This was the left pleural sac distended with gas, and pushed considerably beyond the right border of the sternum, nearly reaching the end of the second rib; directly below this was the pericardium, on opening which the heart was seen to be entirely on the right side of the chest, the apex being behind the right border of the sternum; the front of the heart consisting of the right ventricle, with a narrow border of left; scarcely any twisting on the axis, but the mediastinal structures, bronchi and great vessels, all pushed over to the right side, the trachea passing nearly behind the right sterno-clavicular articulation. The right lung was only visible at the lower external angle of the opening, where the anterior border slightly overlapped the pericardium. On opening the left side of the chest a considerable gush of air took place, and the tumour of the left pleura became flaccid, but did not entirely recede behind the sternum; the heart returned more than an inch towards the left side. The left lung was adherent to the chest-wall posteriorly down to the third rib; anteriorly and laterally the lung was free, except that the small tongue of the upper lobe, which lies ordinarily in front of the heart, had a firm band passing from its apex to the pericardium. There were two openings in the pulmonary pleura communicating with cavities in the lung. The pleural cavity contained a considerable quantity of pus, and the costal pleura was thickened, and covered in many places with pyogenic membrane.

In spite of the extreme displacement of the heart which occurs, cardiac murmurs due to displacement are, I believe, very rare. I have also been unable to satisfy myself of the actual existence of the sharp bending in the vena cava, which the late Dr. Moxon described in displacement, and to which he referred some of the cases of sudden death in large pleuritic effusions.

When the intrapleural pressure is high, the veins running upon the inside of the thorax may be compressed, and the interference to the circulation through them will show itself by distension of the superficial veins of that side, and also by distension of the veins of the arm and neck.

It might be supposed that when air or fluid are present together in the pleura, the air would probably be readily absorbed as the pressure rises. This, however, is by no means the case. I shall return to this point later.

One result attributed by many writers to great distension in pneumothorax my own observation does not confirm. It is stated that by the high pressure the percussion note may lose its tympanitic character, and become muffled or even dull. I have never been able to obtain the slightest evidence at the bed-side to support this assertion, and a few experiments I have made go far, in my opinion, to disprove it.

The access of pneumothorax is usually sudden, and marked by characteristic severe symptoms, but in some cases these initial symptoms are slight, or even, it may be, entirely absent. These cases are described as latent pneumothorax. They are met with usually in the course of advanced phthisis, for to a bed-ridden patient it matters little whether a lung be useless on account of phthisis or because of the collapse which results from pneumothorax. Latent pneumothorax is, however, sometimes met with in phthisical patients who are not in bed, and even occasionally, though of course much more rarely, in the apparently healthy; that is to say, pneumothorax is sometimes discovered in these cases where there is nothing, or very little, in the clinical history to fix the date of its access. I know of no satisfactory explanation of the absence of initial symptoms in these cases, but I have myself met with it a few times.

The prognosis of pneumothorax is, of course, unfavourable. When it occurs in the ordinary course of phthisis, the mortality is very high. My own statistics show that about 90 per cent. of these cases die within the first month. (*Lancet*, May 3rd, 1884.) Weil's mortality-rate is lower—namely, about 50 per cent. (*Deutsch. Arch. f. Klin. Med.*, xxix, 364). It is difficult to attach much statistical importance to such small numbers, and the truth probably lies somewhere between these two extremes. Each case must, however, be judged upon its own merits. In any particular case the prognosis will be determined chiefly by the cause of the affection, and by its effect upon the patient, and next by the character of the effusion which may develop.

I wish, however, now only to speak of the cases in which recovery occurs. This may take place in one of two ways; either by the gradual conversion of the pneumothorax, through the development of fluid and the disappearance of the air, into a case of fluid effusion, which heals in the usual way, or by the simple disappearance of the air without the development of any fluid at all.

In the first group of cases the exudation is, of course, usually serous, but not invariably. In the case of a man, aged 22, suffering from pneumothorax, on the first paracentesis, pus was withdrawn. On the second paracentesis, about a fortnight later, the fluid was found to be sero-purulent, and finally it became serous. Recovery was ultimately complete.

The second class of case, namely, that in which recovery takes place without any effusion, is of course much less common, but still not altogether rare. I described, in the *Clinical Society's Transactions*, vol. xvii, p. 56, one such case, and I collected records of twenty-four other cases from literature, although the list did not pretend to be complete. Since that paper was written I have seen two or three other instances myself, and a few more have been reported, so that the number of recorded cases of this kind is now not very small, and as attention has been directed to them, it is probable that this number will be soon further increased.

This rapid removal of air is familiar in experimentally produced pneumothorax. It is in fact, I believe, difficult to maintain an artificial pneumothorax long in a healthy animal, because the air so quickly disappears. Fluid effusion may also in like manner be rapidly removed, and in both cases alike the lung may very rapidly re-expand. The common explanation given of the re-expansion of the lung after fluid effusion, is not altogether satisfactory. The walls of the chest fall in; adhesions, it is assumed, have formed across the pleural cavity, and as these contract, the lung is pulled out so as to be again in contact with the parietes of the thorax. This may be the correct explanation, but it seems improbable, for adhesions are quite as likely to form over the lung and bind it down, and in fact the existence of adhesions in this position is often assumed to explain cases in which the lung has not expanded. This theory assumes,

moreover, that the pleural cavity must, as a matter of course after fluid effusion, be obliterated, and I do not think that this is always the case in the pleura any more than it is the necessary and invariable consequence of effusion into the joints. Whether this explanation be sufficient or not for the cases of long standing pleuritic effusion, it is clearly inadequate to explain the cases of rapid absorption of fluid referred to. Cure by the formation and gradual contraction of adhesions is slow, and requires many weeks or months to complete itself, but in the cases we are now considering, recovery is often accomplished in as many days.

What is the mechanism, then, by which the lungs in such cases are re-expanded? They collapse, it is known, by virtue of their own elasticity, with a force equivalent to about 7 millimètres of mercury. If they are to be re-expanded, some force is required for the purpose which shall be greater than this. When they are brought out so that the two layers of the pleura are in close contact, cohesion comes into play, as I have shown, to keep them out, but to get them so far a force greater than 7 millimètres of mercury will be necessary. Where is this force to be found?

The question perhaps is simpler in the case of fluid than it is in the case of air. The fluid may be removed in many ways—by absorption, by transudation, or by the pump-like action of the respiratory movements upon the lymphatics of the pleura. The existence of this last process as a factor in the removal of the fluid depends upon the demonstration of stomata in the parietal and visceral layers of the pleura. Recklinghausen first demonstrated stomata on the peritoneal surface of the diaphragm; Dybkowski in the costal pleura; and, although their real existence has been denied recently by Sappey, Klein, in his work on the *Lymphatics of the Lung*, has no doubt of their existence, and thus describes the part they take in absorption. "It is quite clear," he says, "that the respiratory movement of the lungs is a factor that plays a most important part in the process (that is, of absorption by the lymphatics). When the lungs expand, as during inspiration, it will necessarily have the effect that the stomata of the surface are opened widely, and that the inter-muscular lymph spaces of the lung (of the guinea-pig) as well as the proper lymphatic vessels of the pulmonary p'ura, become distended. Under these conditions the lymphatic system will become filled with material that happens to be in the pleural cavity, and that is able to pass through the stomata. During expiration, on the other hand, the stomata as well as the lymphatic spaces and proper lymphatic vessels will become compressed, so that their contents will be discharged into the efferent trunks." He further states, elsewhere, that the existence of stomata is actually demonstrated in inflammation by the bands of coagulated fibrin, which are found plugging them. In the case of pathological effusions into the pleura this arrangement of the lymphatics cannot fail to be of the highest importance. It is often observed that a large effusion remains stationary for a long time when left alone, but that removal of a part of the effusion by paracentesis is followed by a rapid disappearance of the remainder. Paracentesis is followed, as I have shown above, by a return of the respiratory movements, which had been more or less impaired by the large effusion, and the consequence of this will be that the lymphatic pump will be restored to action. It is evident that in this process we have a power sufficient of itself to produce the force requisite for the re-expansion of the lung.

In considering the removal of fluid from the pleura, that peculiar property of the lung must not be forgotten, of which mention has been made. I refer to the ready permeability of a still air-tight lung to fluid under pressure, so that it is possible that a part of the exudation may be actually squeezed through the lung as through a sponge, and, when it has reached the bronchi, may be got rid of partly by expectoration and partly by absorption. Perhaps this is an explanation of the profuse watery so-called bronchitic expectoration occasionally met with in large pleuritic effusions, and which is usually attributed to pulmonary congestion. I think this theory might be tested by chemical examination of the sputum, for such a transudation would probably be more richly albuminous than that of ordinary bronchitis. It is not likely, however, that this process can be of much importance in ordinary cases, on account of the frequent absence of any considerable pleural pressure, and because of the collapsed condition of the lung on the affected side. It may, however, play its part in relieving tension to some extent, and aiding in this way the lymphatic pump to get once more to work.

Lastly, we have to consider the removal of fluid by absorption, with special reference to the consideration whether it may be capable of producing the force requisite for the re-expansion of the lung. I do not know of any experiments bearing directly upon this question, that is, the question of the force developed by absorption within the body.

Exudation (which, I suppose, may fairly be regarded as the counterpart of absorption) may take place under considerable pressure, for proof of which we need not go beyond the pleura itself, as the pleural pressures in many cases of effusion demonstrate. We have only to imagine the process reversed, and more than the required force will be available.

It is possible that, so far as mere exudation goes, there may be no essential difference between dropsy and inflammation, and that in both the exudation may be due to obstruction, with this only difference, that in dropsy the obstruction has usually a more or less distant origin, and in inflammation a local cause in the inflamed part. If this be so, the cessation of inflammation will remove the obstruction, no more exudation will occur, and the effusion will be ready for absorption. If, as Draper assumes (*Medical Physics*, 1885, 303) in opposition to the commonly accepted doctrine, the interchange of gases in the blood in the capillaries is sufficient, as I must confess it appears to me to be, to produce a current onwards in the blood, we may have in this a force of some magnitude to aid in absorption, a kind of capillary aspiration, if it may be so termed.

At any rate, in the absence of direct experiment, we may, I suppose, assume that we may have the same force available in disease to remove pathological fluid from the pleura, which, in health, is active to keep the pleura empty.

The removal of gas from the pleura is a still more complicated problem. The conditions we have to deal with are these, two gas-holding cavities—first, the lungs containing gas of a tolerably constant composition; secondly, the pleura containing gas which differs, in many respects, from that in the lungs;—these two cavities being separated by a complex membrane richly supplied with blood-vessels.

We know that the gas is quickly and completely removed from the pleura, and under such conditions that, at the last, not less than 7 millimètres of negative mercury pressure is produced. The process by which the gases may be removed are these—diffusion, simple absorption, and absorption aided by chemical combination.

The percentage composition of the air in the vesicles of the lungs may be taken to be the following (Michael Foster's *Physiol.*, 1878, p. 265):—N = 79.557; CO_2 = 4.380; O = 16.033, and this probably remains fairly constant, the exchange between it and the atmospheric air in the larger tubes being provided for to a great extent by diffusion.

The air in the pleura varies in composition. If the air enters from without through a wound in the thoracic walls, it is of course to begin with simply atmospheric air. If the air enters through a perforation in the lung, it will have the composition of the air in the vesicles as given above. If the openings remain patent, the composition of the pleural gas may change but little; if, however, the openings be closed, changes in its composition at once take place, and these changes have been used to determine the question of the patency of the perforation. The earliest analysis of the gas in the pleura in pneumothorax was made by Dr. Davy (*Philosophical Transactions*, 1820), who found that the O varied from 2.5 to 5.5, the CO_2 from 6.0 to 8.0, and the N from 88 to 90 per cent. More recent analyses (Ewald, *Reich. u. Dubois Reyn. Arch.*, 1875, 167) show that after remaining for some time in the pleura, the oxygen almost completely disappears, while the carbonic acid and nitrogen increase, the former very considerably. An analysis of Demarquay's will illustrate this (Demarquay, *Gaz. Méd.*, 1865, 496) O = 0.49; CO_2 = 11.16; N = 88.35.

There are, so far as I know, no systematic analyses of the gases made during the stages of disappearance of a pneumothorax, so that the subsequent changes can be only a matter of inference. In the end, however, all the gas, whatever be its composition, is removed.

It is evident, however, that diffusion simple is quite inadequate to explain even those changes which are known to occur; for although at a given time, owing to unequal rapidity of diffusion of the different gases, the relative composition of a mixture might be changed, diffusion could in the end only lead to uniform composition of the two exposed mixtures, and the gases in the pleura could not therefore by diffusion be made to disappear. Grébaud (*Gaz. Méd. de Paris*, 1877, No. 49; 1878, Nos. 13 and 15) has further shown by experiment that when the circulation is intact, little, if any, interchange of gases takes place through the pleura. If his observations be correct, it follows that, in the removal of gas from the pleura, we have to deal only with the same processes as are concerned in the exchange of gases in the lungs; and that what we have to consider is the action of circulating blood upon the gases contained in the pleura. In these processes, diffusion enters only so far as to provide for the passage of the gas into the blood-plasma, where the other processes, namely, absorption and chemical combination, come into play. The

only difficulty here is that the blood is exposed on two sides to gases of different composition; and it might be supposed that, when the tension of gases in the pleura became less than that of the same gases in the vesicles of the lungs, no further removal of these gases from the pleura would occur.

Although the blood absorbs oxygen, carbonic acid, and nitrogen, still these gases were not all found to obey in the blood the laws which govern the absorption of gases in fluid, and it was inferred that the gases entered into loose chemical combination in the blood. This has been proved in the case of oxygen and carbonic acid, and is possible also, though to a much less degree, in the case of nitrogen.

The complete disappearance of oxygen from the pleura after pneumothorax is illustrated by two series of physiological observations. Analysis of the gases contained in the lungs after suffocation by obstruction of the trachea or bronchi shows that the oxygen is reduced to almost exactly the same amount as that in the analysis given of the air in pneumothorax, namely, about 0.4 per cent. Again, in experiments on respiration in a closed space, analysis shows a complete absorption of oxygen from the air of the chamber in which the animal is confined. There is, therefore, no difficulty in accounting for the complete disappearance of the oxygen from the pleura.

The carbonic acid, though partly absorbed, exists similarly in loose chemical combination, both in the serum and in the cells of the blood; but there is this difficulty with carbonic acid, that while oxygen is naturally absorbed from the air in respiration, carbonic acid is naturally returned to it; and the analysis of the gas in pneumothorax seems to show that the carbonic acid does at first actually increase in amount, not only relatively to the nitrogen, but absolutely, so as to even reach 12 per cent., an amount considerably in excess of the carbonic acid tension in venous blood. These facts admit, however, of another and, I think, a more probable interpretation. If, as we suppose, the changes between the blood and the gas in the pleura are the same as those between the blood and the air in the lungs, there will be for every volume of oxygen absorbed an almost equal volume of carbonic acid discharged; so that in the end, when all the oxygen has disappeared, its place should have been taken entirely by carbonic acid. There ought on this assumption to be about twenty volumes of carbonic acid, the nitrogen remaining the same, but none of the analyses given show more than 12 per cent. The conclusion, therefore, may be drawn that the difference, namely, eight volumes, has been already absorbed. This interpretation has the advantage of removing the difficulty previously referred to, of the apparent increase at first of the carbonic acid.

A similar apparent accumulation of carbonic acid takes place, it is stated, in the vesicles of the lung, when the bronchus leading to them has become plugged, the lung in that part, on the absorption of the carbonic acid, becoming atelectatic. It is, however, in the end all removed. As experimental evidence of this is quoted the complete atelectasis which is produced by suspending a lung in water after it has been filled with carbonic acid (Landois and Stirling, *Phys.*, 287).

The nitrogen exists in the blood, it is believed, chiefly in simple solution, although the possibility of weak chemical combination cannot be absolutely excluded. The ease with which the nitrogen may be removed from the blood is demonstrated by the experiment of allowing an animal to breathe an innocuous gas containing oxygen, but free from nitrogen. Under these circumstances the whole nitrogen is discharged from the blood. According to Reynault and Rieset, there is naturally found in the expired air of health a slight excess of nitrogen; but it is also shown by other observers that, under certain conditions, nitrogen may be absorbed to some extent during respiration.

Now, although I do not know of any experiments which actually demonstrate the force which may be developed by the absorption and chemical combination of these gases in the blood, still it is extremely probable that in both of these processes there may be a far greater force developed than that which we require for the re-expansion of the lung. Into this problem the question of time hardly enters; for, if these processes be active at all, whatever influence time may have will be all in their favour. If, however, we may measure the force of interchange by its rapidity, it is clear that we must have great forces to deal with; for we find that in the twenty-four hours no less than 900 grammes of carbonic acid are excreted and 744 grammes of oxygen absorbed; and, in spite of this, there are contained in the entire blood of the body at any given time a total quantity of only 4 grammes of carbonic acid and oxygen combined (Landois and Stirling, *Physiol.*, p. 281). Such a statement shows the extreme rapidity with which the exchange of gases is carried on; and we may, I think, fairly argue from the rapidity to the force of exchange.

The preceding speculations are necessarily somewhat crude, still the

facts which have to be explained are well established, namely, the rapid disappearance of the air and fluid, and the consequent rapid re-expansion of the lung. Incomplete as is our present knowledge of the actual processes concerned, it is evident that in many of them there are sources of power sufficient to provide the force of which we are in search.

I wish that I could have spoken of the treatment to be adopted in the different forms of pneumothorax. There is much to be said upon this question. I must, however, leave this and many other interesting parts of the subject untouched, for the time at my disposal has reached its limit.

When I selected pneumothorax as the subject of this lecture, I did so because it had long been with me a favourite clinical study. I was aware that much had been already done, but I did not realise how much still remained for investigation. I can hardly hope that in this short lecture I have been able to throw much light on the many complicated questions which have arisen, but I shall be content if I have rendered more visible some of the shadows which still rest upon this field of knowledge.

AN ADDRESS

DELIVERED AT THE OPENING OF

THE SECTION OF OBSTETRIC MEDICINE,

At the Annual Meeting of the British Medical Association held in Dublin, August, 1887.

By ARTHUR V. MACAN, M.B.,

Master of the Rotunda Lying-in Hospital, Dublin; President of the Section.

GENTLEMEN,—The first pleasing duty I have to perform on taking the chair is to return my sincere thanks to the members of the British Medical Association for the great honour they have done me in electing me President of this Section. My appreciation of this honour is increased rather than otherwise by the consciousness that your choice was determined more by the fact of my being at present the Master of the Rotunda Lying-in Hospital than on any more personal grounds. This evidence of the respect paid to and the interest shown by the members of this Association in the institution of which I am the temporary head, has emboldened me to make some points in our theories and practice the subject of my opening address, more especially as it gives me the opportunity of inviting any members of the Association who desire it to visit the hospital, and judge of such theories and methods by seeing them put to the test of actual practice.

The points in which I think the teaching of the Rotunda Hospital differs most, either in kind or in degree, from that at the student's command in most other teaching centres, are, first, the systematic use of abdominal palpation in every case of labour, together with the practice of external version when considered necessary; secondly, our time-honoured, and now almost universally adopted, method of conducting the third stage of labour; thirdly, the application of antiseptics to midwifery. This, I, of course, know has been done with success in many other places, and as it will be embraced by one of the subjects fixed on for discussion, I need only here mention it. With regard to gynaecology our practice differs from that in most other teaching centres in this kingdom in the systematic employment of the bimanual examination, the patient lying on her back; the washing out and curetting of the uterus for endometritis, without previous dilatation of the cervix; and lastly, in the theories held as to the normal position of the uterus, and the treatment of displacements of that organ which naturally follow. This last question will be embraced by the second subject fixed on for discussion, namely, the proper treatment of anterior and posterior displacement of the uterus.

To begin, then, with the systematic external palpation of the abdomen in every case of labour. There are probably few among the present company who have not experienced a feeling of despair during their early career at the difficulties which had to be encountered and overcome before the child's position, even in the least complicated cases, could be determined by a vaginal examination, and when the labour was prolonged and a caput succedaneum formed, the diagnosis became almost impossible. Hence my belief that a large number of even experienced practitioners used to content themselves with dia-

gnosing whether the head presented or not, and never troubled themselves as to the mere position. The relative positions, too, of the patient lying on her left side, the practitioner standing behind her, and the child placed upside down in the woman's abdomen, made the mental process very complicated; so much so, that students were recommended in some of the text-books to cut little figures and an imaginary pelvis out of paper to assist them in the process. All this is now changed, and, except in very difficult cases, any pupil of ordinary ability who is not absolutely devoid of the sense of touch can, after a very short apprenticeship, make out the exact position of the child through the abdominal walls, and thus, if called on to deliver artificially, can do so at least intelligently. Not only, however, can we make out the position of the child, but we may generally determine whether the child be large or small, what the quantity of liquor amnii is, and whether the birth is plural or not, and, perhaps most important of all, we can estimate with considerable accuracy the amount of disproportion between the head and the pelvis, by the projection of the head over the pubes, or the extent to which it can be made to enter the brim by firm pressure applied externally. Another point is that the auscultation of the fetal heart follows as a natural corollary, whereas few take the trouble to expose the abdomen merely to examine for the fetal heart. And, finally, there is no such school for learning the palpation of abdominal or other tumours as the palpation of the pregnant uterus. To give some idea of the accuracy of this method, I may mention that we can often feel the arm of the child growing out from the shoulder, and get the fingers into the hollow of the groin, while occasionally the elbow may be made out. In one case a hydrocephalic head was readily made out; in another, the probability of the fetus being anencephalic was foretold; while, in some rare cases, the cord has been felt crossing the child's back; and quite lately we have thought that we have made out the placental site by the fingers being lifted off the child, and the outlines of its body being rendered indistinct. In these latter cases, we have also made out a low continuous murmur, like the uterine *souffle*, but not nearly so loud, over the supposed placental site. Of course, cases in which the placenta can be palpated will always be rare; for to make this possible the placenta must be implanted on the anterior abdominal wall, and the conditions of the abdominal walls and of uterine tension, etc., must be favourable to palpation.

To resume briefly. The points that can be determined by the external examination are:—1, the fact of pregnancy; 2, the period of pregnancy; 3, the size of the child; 4, the condition of the bladder; 5, the axis of the uterus; 6, the condition of the abdominal walls; 7, abnormalities of the ovum, such as twins, excessive liquor amnii, hydrocephalus, anencephalus, hydatid mole, and frequently the life or death of the fetus. Further, it directly leads to the practice of external turning, renders the finding of the fetal heart simple, and generally enables an estimate whether the pelvis be contracted or not.

The step from the accurate diagnosis of the position and posture of the child to external turning is an almost imperceptible one. In a premature case, or in some multipare, even near the termination of the first stage of labour, when the child is not fixed, we can sometimes turn the child by the pressure of a couple of fingers of one hand on the head. Of course, such cases are exceptional, and the difficulty gradually increases with the tension and thickness of the abdominal walls, the fixation of the presenting part in the pelvis, and the passiveness of the patient, till we come to cases where we must first mobilise the presenting part by drawing it out of the pelvis from the abdomen, or pushing it upwards by the fingers in the vagina; and, finally, to such cases where external turning, even under chloroform, is impossible. The cases in which we have found external turning of most importance, besides the mere rectification of a breech or transverse-presentation, are cases of funis-presentation, where, by turning, the funis may be carried up from the os with the part presenting, and cases of head-presentation or cases of antero-posterior contraction of the pelvis, when we wish to extract by the feet. I think, too, that the habit of external palpation also naturally tends to the use of pressure over the fundus in cases of delay, the head being on the perineum. The amount of power we can thus bring into play is very great; we can, in fact, get a large proportion of our own weight to act on the uterus through the abdominal walls; but let us suppose we can apply only 40 lbs. in this manner, and that a considerable portion of this is lost from maldirection. Still, if even 10 lbs. remains as an aid to labour, it will very materially lessen the number of cases in which we will have to apply the forceps to the head at the outlet.

There is one point about such cases which is apt to be overlooked, and that is, that the greatest triumph of the method is when the least amount of pressure has to be exercised. Let me explain this.

If the combined expulsion force is exactly equal to, or the slightest