THE

British Medical Journal.

THE JOURNAL OF THE BRITISH MEDICAL ASSOCIATION.

LONDON: SATURDAY, JULY 7TH, 1906.

The Cavendish Lecture ON SOME POINTS IN THE SURGERY OF THE LUNG.

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MR. PRESIDENT AND GENTLEMEN, — When invited to deliver the Cavendish Lecture, I perceived among the honoured names comprising the list of previous Cavendish Lecturers that the penultimate one was that of my old friend, Professor Mikulicz. It recalled the fact that, at what proved to be our last meeting, after busy days passed at his magnificent Klinik at Breslau, we were spending a quiet Sunday among the Riesen Gebirge, when he told me he had been asked to deliver the Cavendish Lecture. He foreshadowed its subject, and was looking forward to the London meeting with evident pleasure—partly from the love of his work, and partly because he perceived in the invitation an extension of international courtesy which he highly appreciated. As I had known Mikulicz since meeting him in Billroth's Klinik in Vienna, where he acted as second assistant, and subsequently in the many surgical positions he held throughout life, perhaps I may be allowed to say that I have ever admired the ceaseless and untiring energy with which he pursued the culture of surgery, and thereby so greatly contributed to the advancement of its science and its art.

In giving the subject of this lecture as Some Points in the Surgery of the Lung, I had hoped to be able to place before you a series of observations on the physical, physiological, and pathological phenomena met with in dealing with human lungs and their pleurae, and also to have contributed a series of cases illustrative of the modern aspects of lung surgery. It became apparent, however, that within the limited time at my disposal to-night, it was impossible to undertake both. Hence it has been considered expedient to confine attention principally to the former.

The admission of air to the pleura and its results has been, and according to some authors is still considered, the greatest barrier to the surgery of the lung. Thus we have an excellent surgeon and experimentalist stating in a recent paper that "the pleurae do not remain in contact when air is admitted into the pleural cavity, even by a pin-hole opening," and that "when openings large enough to admit the hand are made into the pleurae, the lung, in two or three expirations, collapses to its hilum and remains there stationary, without the slightest effort at expansion." Having been taught that the lung always collapsed on opening the pleural cavity when there were no organic adhesions present, the phenomena presented by some cases which came under my observation in the early Seventies caused me to consider the question and to doubt that collapse of the lung was always so absolute as it was supposed to be. Inquiry was also made as to whether there were modifying circumstances which could avert that collapse, or others which could rectify it when it did occur.

OBSERVATIONS FROM WOUNDS OPENING THE PLEURAL CAVITY.

One of these cases was that of a man who lived only for a short time, having been terribly injured by falling over a circular saw in motion. Besides receiving other mutilations, he had a slice taken off the outer aspect of the right side of his chest in a vertical direction, so as to form a flap consisting of soft tissues, ribs (which were cut as if by a keen-edged knife), and part of the parietal layer of the pleura. The visceral pleural layer was left exposed over an area as large as the palm of my hand. When first seen the patient lay on his back, and, though extremely weak and blanched from loss of blood, he breathed quite easily, and not like one who had collapse of the lung. After his other serious injuries had been attended to, the thoracic wound was examined. There was a mass of blood-clot protruding from and filling up the thoracic wound which precluded visual inspection of the deeper parts, but so easily did he breathe that the deflected flap of tissue was again inspected to verify the previous observation that the parietal pleura was really attached to it. Being fully satisfied on this point it was apparent that the visceral layer must be exposed, yet there were no signs of pneumothorax. As bleeding still continued under the clot it became necessary to remove it and ligature the vessels in the thoracic wall. The clot was carefully removed, first from the centre, until the visceral layer of the pleura was exposed. It was then seen that the lung was fully expanded, and that the pleural cavity was as yet sealed at the periphery of the thoracic wound. There, however, the exposed portion of the lung lay in contact with the atmospheric air, and there was no collapse. One of the intercostal vessels which was still oozing had receded somewhat under the margin of the parietal wound, and during the manipulation necessary to secure it—the blood-clot having been removed from the periphery of the wound—the two layers of the pleura were separated and air was admitted into the pleural sac. The lung receded from the thoracic wall, but only for a short distance, sufficient to enable the finger to be introduced. The bleeding vessel having been secured, the osteoplastic flap was cleansed and it was applied over the wound to see how it fitted. This was done with some pressure. When the flap was about to be removed for readjustment, it was found to have coalesced to the underlying parts, so as to offer considerable resistance to direct lifting. It was, however, easily slid aside, and when the wound was uncovered the lung was seen to have again expanded, the two serous layers having coalesced. Apparently the moist soft flap, during the pressure of its adjustment, had been pressed in along with the whole thoracic wall, until its parietal pleural layer had come in contact with the visceral layer. On relief of the pressure the flap had lifted the lung forward and outward—the coalescence of the two pleural layers remaining and the lung filling the aperture in the chest wall, as it did prior to the admission of air into the pleural sac.

The portion of lung still exposed by the wound was in contact with the chest wall while the patient lay on his back, but, on the patient tossing suddenly round on his opposite side, the air again entered the pleural sac and the lung receded markedly from the thoracic wall. When

turned gently on to his back and pressure was made at the same time on his thorax, the two pleural layers were again brought into contact. The wound was at once closed with the flap, pressure being meanwhile applied to the thorax, and the external parts were suitably fixed to prevent undue expansion of the thorax on that side. His breathing became again quite easy.

From the observation of this case, it appeared probable that there were modifying circumstances which might prevent the collapse of the lung when air was admitted between the two pleural surfaces. Should collapse of the lung occur, to a limited extent, that organ could be expanded again by the simple expedient of bringing together the two layers of the pleura and securing intimate contact between them. Even by putting a suitable moist substance in the gap in the chest wall and bringing it into intimate contact with the collapsed lung, coalescence between the two surfaces was sufficient to bring the lung into its expanded state.

The question arose as to whether the lung in a physiological state could not always be expanded after temporary collapse, as long as the two serous layers were normal and the chest wall was compressible to an extent sufficient to bring the parietal layer into contact with the visceral; also whether the blood effused between the pleural layers had an effect in increasing the capillarity of the parts.

The second case, which came under observation in 1873, was that of a boy, aged 12, who was stabbed in the back, on the right side and posterior part of the chest—a wound remaining half an inch in length, situated between the fifth and sixth ribs. He had haemoptysis, and from the distressing symptoms which he exhibited it was believed that a foreign body still remained embedded in the lung. The wound was examined by the probe, the patient being placed in various positions in order to facilitate its entrance. It was not until the lad had been placed in a stooping posture that the probe slipped through a slit in the muscles, entered the pleural cavity and glided against the smooth surface of the visceral pleura. As the probe entered the cavity it was accompanied by an audible rush of air causing a slight recession of the lung from the thoracic wall, which, while facilitating the examination of the lung, did not remove it from contact with the probe. The instrument was made to move lightly over the visceral layer of the pleura until it entered an irregularity or wound in the lung, the spongy, semi-elastic lung tissue being now felt instead of the smooth surface of the pleural covering along which it had previously glided. Next, embedded in the lung, a hard substance was detected, some portions of lung tissue intervening between it and the probe. By further manipulation the probe came into direct contact with a solid substance having a rough surface, but it was impossible to determine from the touch whether it was metal or earthenware. The outer border alone could be felt, and it seemed to be about $\frac{1}{2}$ in. in length and not more than $\frac{1}{4}$ in. in thickness. On introducing a long-handled aneurysm needle, the foreign body was found to extend into the lung and to have smooth sides. The parietal wound was therefore enlarged (allowing the air freely into the pleural cavity), so as to admit a pair of narrow-bladed dressing forceps, which, with considerable difficulty, reached, seized, and withdrew the foreign body from the lung. It proved to be part of the large blade of a pocket-knife, over 1 in. in length, $\frac{1}{2}$ in. in breadth, and about $\frac{1}{4}$ in. in thickness at the back part. There was a notch on the blade of the knife at the fractured part. The detached notched portion was found firmly embedded in the fifth rib, but projecting somewhat beyond the parietal pleura. This was likewise removed from the rib. After withdrawal of the blade from the lung, profuse haemoptysis occurred, and continued for a time. When it had ceased the wound was injected with an aqueous carbolic solution, the patient immediately afterwards expectorating, and saying that it gave him a "funny, nippy feeling" in his mouth. After the wound was dressed pressure was maintained on the chest wall. The patient made an uninterrupted recovery.

In this case, after the wound was opened, and the deeper parts exposed to visual observation, it was seen that though the air was freely admitted to the pleural cavity, and the lung had receded from the thoracic wall, it had only done so to a limited extent. In full inspira-

tion there was not more than half an inch between the two layers of the pleura. No doubt slight pressure was maintained on the outside of the thorax during the manipulation.

It was also seen that firm pressure on the thorax brought the parietal layer of the pleura into contact with the visceral, while this effected the extrusion of the air between the layers. When this was accomplished the two layers remained in contact and the expanded lung was brought to the level of the aperture in the thoracic wall, where it was in direct contact with the external atmosphere. As long as the lad lay on his back the lung maintained its normal position with the chest wall, but when turned on his face and towards the unwounded side, recession of the lung took place—unless pressure of the thoracic wall was maintained. As it greatly facilitated the extraction of the foreign body in the lung to have the pulmonary wound in close contact with the exterior, pressure was maintained on the thorax during the manipulation, and sufficed to keep the serous layers in contact.

No doubt here, as in the last case, the normal amount of lubricating fluid between the two layers of the pleura was augmented by the presence of a thin layer of liquid blood which may have aided in the coalescence of the two serous surfaces. The capillary attraction existing between the two moistened coated surfaces aided in the distribution of the serous fluid between the layers of the pleura, much in the same way as a drop of fluid spreads out under a cover-glass when placed upon a microscopic slide. The action of the biconcave meniscus was distinctly visible during the coalescence and the separation of the two serous layers. Though varied in detail, somewhat similar experience of the behaviour of the lungs when the pleural sac was opened, was many times repeated in penetrating wounds of the pleura, the lungs, and the mediastinum.

TRAUMATIC PNEUMOTHORAX.

In 1875 a case illustrating another aspect of the question came under observation. It was one of pneumothorax originating from fractured ribs due to direct violence. Immediately after the accident the patient fainted, and on recovery had become speedily cyanosed, and was in great distress from apnoea, the cardiac action being at the same time very feeble. It was evident that the pneumothorax was so great as to press the mediastinum and its contents toward the opposite lung, whose function was likewise embarrassed. Immediate aspiration was resorted to with marked benefit and relief. This was, however, of short duration. Ten minutes later the lividity and embarrassment was as great as before. Aspiration was again resorted to, very thoroughly this time, and though the relief was immediate it was as short-lived. On three other occasions the aspirations were each followed by a like result. It was evident that the pneumothorax was so great that it threatened life, being sufficiently powerful to cause collapse of the lung. If this compression went beyond a certain degree it would be impossible to bring the parietal layer of the pleura into contact with the visceral, and other means more difficult of application and more doubtful in effect would require to be resorted to to expand the lung. It is also believed that if a sufficient aperture were made through the thoracic wall in proximity with the wound in the lung the compression exercised by the confined air in the pleural cavity would at once cease.

My experience had not hitherto shown me an instance where such compression of the lung as this man was labouring from had been brought about by the entrance of air through an open wound in the thoracic wall. In such a case if the thoracic wall were opened one could deal with the lesion in the lung effectively. It was suggested that a trocar and cannula might have sufficed to draw off the air, the cannula being left *in situ*, but a direct opening in the chest wall, as nearly opposite the wound in the lung as possible, was preferred. An incision was made over the centre of the thoracic depression, a portion of the rib which had already been fractured was removed, and the pleural cavity opened freely, from which air, and afterwards blood, escaped. The lung was found to be much further collapsed than I had then seen in any of the cases with thoracic wounds. The thorax was vigorously compressed on to the contracted lung, and this induced the patient to hold his breath. The air was extruded through the aperture, and

the two serous surfaces were brought into contact. Pressure was maintained on the thoracic wall during the subsequent manipulations. It was now seen that the lung was torn, a portion of the visceral pleura having been invaginated into it. As the wound lay in the lung above the level of the rib which was removed, an inch of this upper rib was likewise taken away. The wound in the lung was fairly opposite the thoracic wound, and the air from the lung found a direct exit. Blood escaped at first freely from the pulmonary wound, and there was a considerable amount in the pleural cavity, compression of the pulmonary wound arrested the oozing, after which the invaginated and torn portion of the visceral pleura was taken out and secured across the gap by a single stitch. Blood-clot soon filled the gap in the lung. A light pad of antiseptic gauze, loosely applied, was inserted between the lips of the thoracic wound, which was thus kept open, and any air which might come from the lung would thus escape externally. Firm pressure was kept on the external thoracic wall above and below the wound in the thorax by means of adhesive plaster, as if for broken ribs, and a firm roller bandage was applied. The relief to the patient was immediate and lasting. The wound in the lung soon closed, and the tissues of the thoracic wall were then brought together.

On two other occasions when pneumothorax threatened life from collapse of the one lung and pressure of the mediastinum and its contents toward the opposite side, after repeated aspirations were found ineffectual, a similar opening in the wall was made, as nearly opposite the wound in the lung as possible, and the collapsed lung then expanded on bringing the two visceral layers intimately into contact.

On one of these occasions, however, considerable difficulty was experienced in effecting the restoration, as the lung had been reduced to such a small volume that it was impossible to bring the rather rigid thoracic walls into contact with the lung and arrangements were made for drawing the lung outwards. It was found, however, that the lung could be partially distended by the patient making expiratory efforts with his glottis shut, such as by straining, sneezing, or by trying to cough with the mouth and nose closed. By a combination of external pressure on the thorax and diaphragm, through the abdomen, and internal expiratory effort the two layers of the pleura were brought together, the lung expanding and remaining expanded, while light compression of the thoracic wall was kept up.

When pneumothorax causes such absolute collapse of the lung, and this condition is persistent, pulmonary consolidation might ensue. The longer air exists in the pleural sac the drier the serous surfaces become, and even when extra fluid is poured out the natural secretion is altered and its capillarity and cohesive power are defective.

In such cases, after repeated tapplings, if the pneumothorax persists to a marked extent and threatens pulmonary collapse, open the pleural cavity by removal of a portion of rib, if necessary, as near the seat of the pulmonary injury as possible, and close the wound in the lung by suturing the visceral layer of the pleura where permissible. Then place the two pleural layers together by bringing the parietal into contact with the visceral layer. This must be effected by firm pressure on the chest and diaphragm. Cohesion will again occur between the two layers of the pleura and the pneumothorax will disappear. Maintain the aperture in the costal pleura until the wound in the lung is healed, or at least until it has been firmly sealed by exudation.

Since 1875 this method of expanding the lung has been made use of by me in operations of the chest wall, in removal of tumours of the thoracic parietes, and in a few cases where the lung itself was involved in the growth, and it has answered the purpose as long as the pleural surfaces remained in an approximately normal condition.

During the thirty odd years which have elapsed I have regularly taught that molecular cohesion and capillarity were the principal forces at work in maintaining the expansion of the lung, and also that when collapse of the lung did take place, these forces could be brought into play so as to produce re-expansion of the lung. Though much has been written on the physiology and surgery of the lung, which has increased our knowledge and improved our practice, yet the old theory of the maintain-

ance of the expansion of the lung by atmospheric pressure has been maintained, with few exceptions, by physiologists and surgeons alike. The current belief, even now, is that the moment air enters the pleural cavity, the lung not only collapses, but does so absolutely to its hilum, and that it remains permanently collapsed. Therefore the opening of the pleural cavity is considered so dangerous that it is eschewed. A notable exception to those views is found in an excellent paper by Dr. West, which has just been read by me, but which was published in 1887. He there advocates the existence of a cohesive force between the two pleural layers.

It is still held by most physiologists that the natural elasticity of the lungs would cause them at all times to contract away from the ribs "were it not that the contraction is resisted by atmospheric pressure, which bears only on the inner surface of the air tubes and air sacs. On the admission of air into the pleural sac atmospheric pressure bears alike on the inner and outer surfaces of the lung, and their elastic recoil is no longer prevented, and a considerable space is left between it and the chest wall."

THE CAUSE OF THE EXPANSION OF THE LUNG.

Whatever effect atmospheric pressure within the chest may have in aiding expansion of the lung, it is clear that it is not the only factor, if, indeed, it be the chief factor in maintaining such expansion.

Experience shows that the lung is maintained in full expansion by the molecular cohesion existing between the two serous surfaces of the pleura, and the capillary attraction exerted by a thin layer of serous fluid existing between the two moist membranes. Although this molecular force may not be considerable at any given point on the pleura, yet, when it is remembered that it is distributed over the entire surface of the periphery of the lungs, its cumulative effect is very great and amply sufficient for the purpose. When this molecular cohesion is removed collapse of the lung occurs, owing to the elasticity inherent in the pulmonary tissues. Doubtless, when the pleural sac is opened and air is allowed to enter between the layers of the pleura, the lung will recede, the molecular cohesion being in abeyance. Total collapse of the lung does not, however, occur from atmospheric pressure, and the degree of recedence which ensues depends upon a number of modifying circumstances. Not only so, but while the atmospheric air surrounds the partially collapsed lung, the molecular cohesion, aided by the capillary attraction, may be brought into play to the exclusion of the intervening air, with the restoration of the lung to its expanded state.

The resultant collapse of the lung depends somewhat on the mode adopted, and on the part of the thorax selected for opening the pleura. When a choice is possible one ought to select a point as far from the hilum of the lung as possible. The outer convex aspect of the lung affords a broad surface which is suitable for opening the pleura, minimizing the collapse, and at this part the molecular cohesion can much more easily be brought into play, so as to restore the expansion of the lung.

Molecular cohesion is usually illustrated by the phenomena shown when two sheets of bright polished plate glass are brought into intimate contact; they cohere, and the truer the surfaces, the more perfect the cohesion. Not only do they hold one another together against the action of gravity, but in addition they sustain a considerable weight. This must be attributed to a reciprocal action between the two surfaces, and can have nothing to do with atmospheric pressure, as the experiment succeeds in a vacuum. When, however, the surfaces of the glass are moistened and they are pressed together, the cohesion becomes more perfect, and the resistance to separation much greater. If, instead of using water, the surfaces were covered with some fluid whose surface tension was better adapted for the purpose, the cohesion would become still greater. It is well known that there are differences in the surface tension of different liquids, and even of the same liquid in varying conditions. Serous fluid, such as is found in the normal pleura, is admirably adapted for the purpose which it serves. It is at once sufficiently fluid to flow easily and to lubricate freely the surfaces of the pleura, and to exhibit the phenomena of capillarity when one of the two pleural layers is wounded. Besides this, the fluid has sufficient viscosity to make it

valuable in hermetically sealing the lips of an incision in the parietal pleura, as when one tries to separate the two surfaces a concave meniscus is at once formed at either lip of the solution of continuity. In this way the serous pleural fluid is of value in preserving the normal relations of the two pleural surfaces, and consequently in preventing pneumothorax when the parietal layer is wounded. The two serous surfaces of the pleura, being lubricated by this viscid serous fluid, are capable of gliding over one another as in respiration. In surgery this freedom of movement may be made use of when the parietal layer of the pleura has been incised by sliding the parietal over the visceral layer of the pleura. One of the most striking illustrations of the power of capillarity and molecular cohesion is seen in the hip-joint. When the head of the normal femur has been detached from its socket, and all the ligaments and muscles which unite it to the body have been severed, if the head of the femur be again placed in the acetabulum while it is still moist with synovia, it will become so fixed that not only will it support unaided the weight of the whole lower extremity, but one finds it difficult to displace the head of the bone by forcible traction on the limb. Under these circumstances, in order to remove the head of the bone from the joint, it is sometimes necessary to take advantage of the long lever which the femur presents, in order to prise the head of the bone out of its socket. Those who seek to perform amputation of the hip-joint by first detaching the soft parts from the head and neck of the femur, and turning the head of the bone completely out of the acetabulum, and then proceed to sever the muscles at a lower level of the limb, find that they must take the precaution of introducing a portion of gauze or other suitable material between the head of the bone and the acetabulum, otherwise the head of the femur would slip again into position in the acetabular cavity, where cohesion will fix it in such a way as to render it difficult to remove the head of the bone at the critical moment when the soft parts and the vessels are severed. Even a notch made in the acetabulum does not destroy the molecular cohesion, unless large. Experience shows that the pleura, while in a physiological state, acts in a similar way, and that the molecular cohesion enables the two layers to be held together even when the parietal layer has been penetrated and a considerable area of the lung has been exposed.

The effect on the respiration of the admission of air into one of the pleural sacs will depend first on the condition of the other lung and its pleura. Provided the opposite lung is normal and in full functional activity, and its pleural and mediastinal connexions are intact, the opening of one pleura will produce less serious effect on the respiratory and circulatory function as a whole. Should the one lung be limited in its respiratory cavity, the opening of the pleura on the opposite side will clearly be attended by more serious consequences. The effect of admission of air into the sac will be modified somewhat by the position in which the patient is placed at the time of operation. If the pleura be opened while the patient is lying on the sound side, the compression of that side is greatly increased by the action of gravity, which throws the weight of the now loosened mediastinum and its contents, including the heart and blood vessels, upon the sound side, and prevents the mediastinum from aiding in the fixation and expansion of the sound lung. On the other hand, if the patient be turned on the affected side, the action of gravity favours the apposition of the lung to the chest wall on that side, thus limiting the amount of air which enters the pleural sac, and aiding in fixing the mediastinum and in expanding the sound lung and enabling it to perform its increased function with the least impairment.

Doubtless, after the pleura is opened and the lung collapsed or removed, the steadying of the mediastinal septum is of service in restoring the power of respiration of the other lung, and in allowing the heart to act free from the jumble produced by the mediastinal flapping. This is best produced by the cohesion of the two pleural surfaces after the pleural cavity has been opened. This cohesion of the visceral to the parietal layer draws out the lung and fixes the mediastinum. It may also be aided by temporarily closing the aperture in the thoracic wall with moist lint. The action of gravity further assists both the expansion of the collapsed lung and the fixing of the

mediastinum—therefore, by placing the patient on the affected side before opening the pleura, the difficulties of breathing are greatly obviated. The lung is kept fairly expanded and the mediastinum fixed. Drawing a portion of the lung out of the aperture in the chest wall and suturing it, so as to act as a plug, has not been performed by me, but has been found also to cause the cessation of the dyspnoea by aiding in the expansion of the lung.

PRESERVE THE PHYSIOLOGICAL, LUBRICATING, PLEURAL FLUID.

The possibility of the visceral and parietal pleurae becoming dry must be considered, though this can rarely happen while the two layers are in a physiological condition. A lubricating fluid between the pleural surfaces is necessary for the maintenance of capillary action which aids molecular cohesion. Blood while in small quantity and mixed with the normal pleuritic fluid does not seem to diminish the capillarity of the fluid, and when it is in sufficient quantity to coagulate round the orifice of the pleural wound it aids in sealing the pleural cavity. Sterilized glycerine and water was used on one occasion, painted over the pleural surfaces of the lung when it had become dry from exposure during operation, and it seemed to aid the coalescence, but did not act so well as the normal lubricating fluid. Sterilized water was often used and answered the purpose sufficiently.

THE PLEURAE UNDER PATHOLOGICAL CONDITIONS.

The foregoing remarks apply to the pleura in a healthy or physiological condition; but when pathological changes have supervened, then the behaviour of the pleura may be modified accordingly. When the pleural surfaces are covered by plastic exudation or layers of fibrinous patches the power of molecular cohesion between the surfaces is greatly lessened or lost. Instead of the smooth, glistening surface of the healthy serous membrane, with its interposed lubricating fluid, there is an unevenness of surface which prevents the exact and intimate coaptation which is so necessary for the purpose of securing perfect results, and coupled with this there is an absence of the normal lubricating fluid. When such a pleural sac is opened, unless organic adhesions have already formed, fixing the lung to the parietes, there is apt to be a degree of recedence of the lung, sometimes considerable, though this contraction does not increase much on freely exposing the cavity, emptying the contents and maintaining the patency of the external opening. Here the consistence of the pulmonary tissue is altered. There is less elasticity and less recoil, and therefore there is less tendency to collapse and less capacity of expansion. Bringing the costal walls into contact with the pulmonary pleura is not in this case followed by immediate obliteration of the pleural cavity, as it was when the pleura was in a normal condition. Hence the necessity, in many such cases, of removing a sufficient portion of the osseous walls to permit the thoracic wall to come in contact with the lung. Obviously the recedence of the lung from loss of molecular cohesion may lead to the physical production of hydrothorax, and its restoration will quickly cause reabsorption of the fluid.

THE DETERMINATION OF PNEUMOTHORAX AND EMPHYSEMA IN CASES OF FRACTURED RIBS.

Many authors comment on the well-known fact that with simple fracture of the ribs emphysema is very common and pneumothorax is rare, even when the pleural surfaces are free from pathological adhesions. To account for this, some assert that the air does enter the pleural cavity, but becomes so rapidly absorbed therefrom that pneumothorax is prevented. Others believe that the resistance offered by the coalescence of the two pleural layers prevents the admission of air, as it is greater than the resistance offered by the subcutaneous cellular tissue, and therefore the air issuing from the lung causes emphysema.

Regarding the former theory, one would require to assume that the air in the pleural cavity was absorbed as rapidly as it issued from the lung, otherwise pneumothorax would form. There are no known facts which support the belief in such rapid absorption of air from the pleural sac. With regard to the second theory, it is possible that the subcutaneous tissue may offer less resistance than that presented by the cohesion of the two pleural surfaces, but

even if this were granted it would not alone account for the facts. In looking for an explanation, it is necessary to take into consideration the conditions in which the various layers constituting the wounded parts have been left by the lesion, and in this will be found the principal determining factors between the formation of emphysema and pneumothorax. If a spiculum of rib, while penetrating the lung, merely punctures the parietal pleura, the pleura being caught firmly round the base of the spicule so as to prevent air passing into the subcutaneous cellular tissue, then pneumothorax would be likely to result. On the other hand, if the parietal pleura be freely torn as well as the subcutaneous cellular tissue, and a direct exit for the air from the lung into the lacerated cellular tissue be provided, doubtless emphysema would follow. The lung is also occasionally found bound to the rent in the parietal pleura by a spiculum of rib which has been transfixed and which holds it in position, the air passing directly from the lung into the lacerated cellular tissue. On several occasions where emphysema had been marked, it was found, *post mortem*, that there were rents in the parietal pleura, and a probe could be passed from the lacerated and emphysematous cellular tissue directly into the aperture in the lung.

It will be obvious also that extravasated and coagulated blood may act as a determining factor. If one of the intercostal vessels poured its blood into the lacerated cellular tissue and coagulation of the blood ensued, emphysema would be likely to occur. If blood became effused between the two layers of the pleura, and it coagulated round the periphery of the pleural wound, the air from the torn lung would be apt to be diverted into the lacerated cellular tissue and emphysema would result.

If blood was extravasated into the pulmonary tissue at the periphery of the wound in the lung, it might, by expansion, bring the sides together; or if a clot formed in the pulmonary wound itself; in either case neither emphysema nor pneumothorax would result. So that in cases of fractured ribs, with penetration of the lung, emphysema occurs more frequently than pneumothorax on account of the fact that the costal pleura and subcutaneous cellular tissue are usually sufficiently torn to permit the air from the damaged lung to escape directly into the cellular tissue, and of course cohesion of the pleurae prevents the cavity from being invaded. On one occasion a case was seen by me where rupture of the lung had occurred from direct violence applied through the chest without fracture of the ribs ensuing. In such cases pneumothorax would be likely to result.

COMPRESSION ADHESION BETWEEN THE TWO LAYERS OF THE PLEURA.

Immediate adhesion between the two layers of the pleura takes place from sudden and violent impact in the same way as it does between other serous surfaces, such as those of the pia and dura mater, but perhaps more frequently seen between the visceral and parietal layers of the peritoneum and the omentum or mesentery. I have seen intestinal obstruction following injury occasioned in this way.

This I have called compression adhesion. It is usually followed by pathological changes which may lead to organic union between the two parts originally held by compression. How often it occurs between the two layers of the pleura there are at present no data to guide one, and from the nature of the parts such will be difficult to obtain, but that it does occur between the pleural layers the following cases show: On two occasions of immediately fatal injuries this was seen to have occurred, the adhesions in the pleural surfaces corresponded in form to that of the external body with which the thorax had come into contact. There had been no time for pathological changes to have taken place, and yet the compression adhesions were as firm as if the two pleural layers had been stamped together. There was slight extravasation of blood into the pulmonary tissue corresponding to the position of the adherent pleurae. This extravasation would doubtless have been much greater had time permitted. In one of these the woman had fallen from a height on to a kerbstone, the thorax striking the stone longitudinally from the third rib in front of the diaphragm below. An ecchymosis corresponding to this strip appeared in the subpleural pulmonary tissue. In such

cases neither emphysema nor pneumothorax could occur even with fractured ribs.

THE CAUSATION OF THE PRIMARY SHOCK IN ADMISSION OF AIR TO THE PLEURAL SAC.

The primary shock which ensues on the admission of air to the pleurae, and which follows the collapse of the lung, has been attributed to the effect which the air produces on the pleura, or to the effect on the pneumogastric filaments occasioned by the sudden contraction of the lung tissue. Neither of these theories are sufficient to account for the intensity of the phenomena produced. Doubtless the compression of the lung and impairment of the respiratory exchange of gases, as well as the displacement of the mediastinum, lessening the powers of expansion of the lung, are contributory to the production of primary shock. My observation leads me to the opinion that primary shock is really due to the effect upon the heart. Although the lungs are respiratory organs, they are so intimately connected with the heart that one cannot consider them apart, especially during surgical procedure. The heart and the thoracic great vessels, along with the mediastinal structures, are practically suspended or buoyed up by the soft elastic lung, as long as the lung remains fixed to the thoracic wall. When one lung collapses the heart and great vessels lose much of their support, and though still partially supported by the pulmonary tissue on the other side are yet apt to suffer. With the thoracic cavity of one side filled with fluid or air, the heart though pressed on unduly, is steadied, and is not allowed to swing about as it does when the thorax on one side is fully opened, the lung collapsed and the mediastinum flops toward the empty space.

One patient, who was in *extremis* from disintegration of the left lung and pressure within the left side of the thorax, was relieved at once by thoracotomy, yet the flopping of the heart was so great within the empty cavity as to cause him to have an intense angina-like spasm on being turned ever so gently from one side to the other. The recurrence of this cardiac spasm was prevented by a soft gauze packing inserted inside the thorax, fixing the pericardium and steadying the mediastinal contents. In the subsequent course of the case it was interesting to observe how Nature intervened by fixing the pericardium by means of fibrous adhesions to the anterior thoracic wall and by an extra band to the diaphragm.

When air is admitted into the pleural cavity an important factor, which must not be overlooked, is the immediate and direct effect which the shrinkage of the lung produces on the heart. The pulmonary circulation is carried on by a series of vessels possessing great capacity for extension under linear traction, and having a remarkable power of recoil. These vessels are in contact with the heart at one extremity and with the periphery of the lung on the other, and in their whole course between the heart and the pulmonary periphery there are no anastomoses. In the periphery of the lung, where the veins lie beneath the pleura, and the subdivision of the vessels is very fine, anastomosis does occur, but between this point and the left auricle there are no anastomoses. Consequently, the influence produced by the peripheral arrest of pulmonary circulation would be instantly transmitted to the heart. This will be further evident when it is borne in mind that the four main pulmonary veins are so intimately connected with the heart that they may be regarded as diverticula of the left auricle sharing in its rhythmical contractions, having even incorporated into their texture striated cardiac fibres. The pulmonary veins have no valves anywhere in their course; so that the full force of any sudden stoppage of peripheral pulmonary circulation will be borne upon the heart directly. The result of the entrance of air into the pleura, when it produces contraction of the lung, is felt most at the periphery of the lung which shrinks, and that peripheral shrinkage in the calibre of these minute vessels greatly impedes, and may even arrest, the flow of the blood through them, and consequently through both the pulmonary arteries and veins. As this occurs suddenly, arresting the flow of blood into the lung from the right ventricle and at the same time gorging the right auricle, it produces an immediate staggering effect upon the heart, which falters in action and results in dyspnoea and lividity of the cutaneous surface. If it were bilateral it would probably lead to a fatal issue; but, when

unilateral, and if means be adopted to minimize the action, the cardiac function is soon restored, even when the one lung remains contracted.

THE PLEURA IN THE ANTERIOR MEDIASTINUM.

In the anterior mediastinum the layers of the parietal pleurae from both lungs meet, and as at this point they are very thin, when air has entered one pleural sac, and when rapid displacement or flopping of the mediastinum takes place, it is possible for rupture of the opposite pleura to occur, leading to embarrassment of function or collapse of the opposite lung. The possibility of a potential or patent opening existing between the two pleural layers in the anterior mediastinum ought to be borne in mind. Though such an opening has not been demonstrated as occurring in man, it is sometimes found in the dog, and commonly in some of the lower animals, such as the solipeds. If such an opening pre-existed admissions of air into one pleural sac would fill both pleurae, and tend to produce compression of both lungs. I have not had such an experience, and have not heard of any reported case of the kind. The mediastinal pleura is attached to all the important mediastinal structures, such as the pericardium, the trachea, oesophagus, and the vagus and phrenic nerves. It is difficult to separate the mediastinal pleura without endangering these important structures, besides possibly opening the neighbouring pleural sac.

The Hilum of the Lung.—In attempting removal of one lung, injury to the mediastinal contents is one of the chief dangers. The hilum of the lung in the mediastinal space is close to the right auricle, the vena cava and the trunks of the pulmonary artery and veins. In attempting removal of the lung, it is better to imitate Nature by leaving the hilum as is seen in separation of the slough in gangrene of the lung, severing the tissues several inches to the outer side of the hilum when the phrenic and vagus nerves are left uninjured. The pleural covering of the mediastinum ought to be left intact if possible.

The Apical Pleura.—The pleura at the apex of the lung is attached to the innominate vein, and further out to the subclavian artery, and sometimes even to the brachial plexus. Therefore it is better to leave intact the parietal, and even the visceral, layer of the pleura if it be adherent at the apical part.

PRESERVE THE ATTACHMENTS OF THE DIAPHRAGM.

When one makes extensive operations on the chest wall, it is important to preserve the ribs to which the diaphragm is attached and also the attachment of the diaphragm to the ribs. By removing the whole of the ribs on one side of the chest to which the diaphragm is attached, the breathing on both sides of the chest becomes laboured or is made with an effort, the diaphragm no longer performing its function properly. Fresh attachments are, however, soon formed by the diaphragm if there are fixed osseous points remaining. When one remembers that the diaphragm is the most powerful respiratory muscle, the importance of retaining its attachments will be obvious.

In the few remaining minutes which are still at my disposal, instead of giving a hasty clinical summary of personal experience, I have thought it better to give an illustration of one of the gravest forms of tuberculous destruction of the lung, in the hope of showing that whatever opinion we may form concerning surgical intervention in the earlier stages of tuberculous invasion of that organ, yet when the other extreme has been reached, and the patient has sunk into what has commonly been regarded as an incurable condition from lung disintegration, that surgery may be of service not only in alleviating distress, but also in furthering actual cure.

EXTRACT FROM CASE OF R. W.

When admitted in Professor Macewen's wards patient was in the following condition:

Patient was much emaciated, and sat in bed. He had rapid breathing and an aspect of distress, and was covered with perspiration, and complained of pain below the left shoulder-blade and in the left mid-axillary line, toward the lower border of the costal margin. When he attempted to get out of bed he was very short of breath, and experienced what he described as a crushed feeling round the body. He could lie for a short time on his back or on the left

side, but could not do so on his right side for more than a few minutes without becoming cyanosed and having great discomfort and a sensation of asphyxia.

Physical Examination.—There was a great bulging and fixation of the left side of the chest. Expansion movements were lost on the left side. The respirations numbered 24 per minute. There was a diffuse pulsation in the third, fourth, and fifth interspaces on the right side. The pulsation could be seen in these spaces extending from the vertical nipple line to about 3 in. to the left of the nipple. There was dullness to percussion over this area, and the sounds of the heart could be distinctly heard. The sounds were evidently due to the displacement of the heart to the right side. There was absolute dullness over the left lung both in front and behind. This dullness was continuous with the splenic dullness below, and in the middle line extended for 2½ in. below the costal margin. On the left side the respiratory murmur, the vocal fremitus, and resonance were absent. Over the right lung the respiratory murmur was free, with the exception of the right mammary region, where some subcrepitant râles were detected, and there were coarse râles at the base. The temperature has only been about 100° F. in the evening, and in the morning has been normal. He has almost no expectoration, the patient stating "he only wished he had," as when he was expectorating mouthfuls of pus he had been easy, but since it ceased he had become gradually more and more distressed, and now found he could not get breath. The history of the case pointed to:

1. A disseminated tuberculous invasion of the left lung, commencing nearly three years previously.
2. Probable extensive tuberculous disintegration upon which a secondary pyogenic infection had taken place.
3. The formation of an abscess cavity or cavities in the left lung.
4. The formation of a pneumothorax from perforation or the formation of a very large cavity in the lung representing total pulmonary disintegration, and giving signs simulating those of pneumothorax or possibly both combined.
5. The pulmonary cavity emptied itself regularly for eleven months by free expectoration, through the bronchi, trachea, and mouth, till shortly before his readmission into the infirmary, when it ceased entirely, soon after which there was increased pain and distress in the chest, and probably from the accumulation of pus. The cessation of purulent expectoration was possibly due to the soldering of the bronchi by granulation tissue, which no doubt took place gradually, but the last patent bronchus became filled with a plug of granulation tissue probably projecting from the margin of this bronchus when the expectoration suddenly ceased.
6. The right lung, it was feared, was already affected with tubercle, judging from the physical signs in the right mammary region, and also from the liability of both lungs being affected when such an extensive lesion existed in one of them. His condition was such as to imperatively demand relief.

Operation, April 24th, 1895.—The patient was placed slightly under the influence of an anaesthetic, as, owing to the great embarrassment in breathing and the general lividity, the full anaesthetic effect was not considered safe. An incision was made over the seventh and eighth ribs in the line of the posterior axillary border. The periosteum was raised and a couple of inches of each rib were removed. The wound was then dusted with iodoform and boracic acid, which was rubbed into the soft tissues and the cut ends of the ribs, after which the periosteum and thickened pleurae and layer of lung tissue were penetrated by a pair of dressing forceps, which were then withdrawn while the blades were fully expanded.

From this aperture 160 oz. of pus, along with sloughs of the lung and caseated debris, were removed. The pus contained tubercle bacilli in enormous numbers and individually of large size; each drop, taken indiscriminately from the receiver containing the pus, swarmed with them. It is rare to find tubercle bacilli in such quantities in pus even from lung abscess. The fluid contained in this cavity seemed to have acted as a culture medium for pyogenic organisms, both streptococci and staphylococci being present. By means of the finger and the long probe introduced into the

cavity a dense layer of lung tissue with shreds of disintegrated lung were found still adherent to the chest wall. After thorough evacuation this cavity was washed out with an antiseptic solution, and gauze was introduced as a drain.

This operation afforded great relief, except that the patient would not dare to turn from one side to the other, and preferred to lie on the sound side. He said that when he tried to turn, something gave him a violent wrench, as if "his heart were being pulled out." This was attended by a distressing angina-like spasm. The reason for this sensation, which the patient experienced, was not understood at the time, though it was afterwards fully explained. After four weeks it was evident that the great space on the left side of the chest was not closing, and besides there was a persistent and free purulent discharge from this large cavity, which, if it were allowed to continue, would soon have exhausted the small amount of strength which the patient still possessed.

Under these circumstances it was deemed advisable to perform thoracoplasty, both with the view of thoroughly exploring the cavity and removing the shreds of lung tissue still remaining, and also with the hope of reducing the dimensions of the cavity by permitting the external wall to be involuted.

Excision of portions of the fourth, fifth, sixth, and seventh ribs was performed, each portion extending from the angle of the rib to the costal cartilage. It had been intended to remove three other ribs, but the patient's condition was such that their removal had to be deferred. The view obtained of the interior of the left side of the thorax by this free opening was such as to enable one to see that there was no vestige of the left lung remaining, save shreds adhering to the walls of the chest, a portion at the apex and a stump representing the larger bronchi and the corresponding blood vessels, somewhat fused together, and covered by granulation tissue. Behind this stump the descending aorta was seen and felt, and in front of the stump the pericardium lay freely exposed and non-adherent by pathological adhesions. The absence of the pericardial pathological adhesions was so marked and the normal attachments so relaxed from over-stretching of the diaphragm downwards and pressure of the heart toward the right side, that when the patient lay on his sound side the heart rested against the anterior mediastinum, and when he turned on to his back the heart flopped backwards over the stump of the bronchi, and the lower portion of it within the pericardium swung free in the thoracic cavity. The pericardium was somewhat thickened, but not so much as one would have expected, owing to the great destruction of the lung tissue occurring in its immediate vicinity.

The free movement and flopping of the heart inside its pericardium explained the strong objections which the patient expressed, previous to this second operation, to being turned from one side to the other, as he said each time he was turned it gave his heart a wrench. This had not been experienced by him before the evacuation of the fluid from the chest, but had been markedly felt in the interval between the first and second operation. There were several shreds of disintegrated lung tissue still adherent to the diaphragm, to the outer walls of the chest and to the apical part of the thorax. The former were removed, the latter cut short, their proximity to the large subclavian vessels rendering it undesirable to attempt complete removal. In order to prevent displacement of the heart, and to hasten adhesions between the pericardium, the mediastinum, and the diaphragm, the large cavity was packed with gauze, which was changed at frequent intervals. Subsequently adhesions quickly formed between the pericardium and the diaphragm, one of these first appearing as a comparatively thin band which stretched somewhat during the dressing. It gradually became thicker until it attained the diameter of a finger. These adhesions were then able to hold the over-stretched pericardium in its place, and thus to prevent the swinging of that organ, which it was previously subject to when the patient was turned from one side to the other. Soon after these adhesions had formed, the pericardium became attached to some of the structures in the anterior mediastinum and later to the anterior wall of the chest and to the stump of the bronchi behind. Gradually all the structures presenting on the left side of the mediastinum became covered with granulation tissue and fused

together so that their individual outlines were lost, a continuous wall having been formed over the left side of the mediastinum from the anterior to the posterior portion of the thorax. The right lung likewise expanded to the right side. Portions of the third, eighth, and ninth ribs were removed at a later date to further facilitate contraction. The osteoplastic resection thus extended from the third to the ninth rib inclusive. After the interior had become aseptic and a layer of healthy granulation tissue had filled the various recesses, the skin was laid over the now thickened pericardium and diaphragm and stump of lung, and soon became adherent and filled up the cavity, a small sinus at the upper end persisted, coming from a point where shreds of the apical portion of lung remained. On several occasions, while the cavity of the thorax was still open, these were attempted to be detached, but on each the adhesions were found too firm to permit of easy separation. The adhesion of the apical portions to the subclavian vein precluded further detachment. The loose portions were trimmed. His general health meanwhile improved, the râles disappeared from his right lung and he gained strength and weight, and was able to walk about and enjoy life.

It is now eleven years since this man was operated on, and since his convalescence he has been able to engage in light work, supporting his wife and family thereby. He can walk about easily while taking time, and generally enjoys good health.

Examination of his lung at present time:

On June 3rd, 1906, this patient was examined by three physicians, who reported that there were no breath sounds on left side of the chest, except the transmission of bronchial sounds heard under the clavicle on the left side. The right lung appeared normal. The heart sounds were good. The patient states that he has been regularly at work as a light porter; he can go upstairs without trouble. He mounted the eighty odd steps of the infirmary stairs before he was examined, and did so without pause and with very little inconvenience. He states that since his operation he has walked ten miles without fatigue, but he does so at what he calls an "ordinary easy rate." Since the operation he has never been ill and has regularly been at work.

Gentlemen, allow me to thank you for the honour you have done me by inviting me to deliver the Cavendish Lecture for 1906, and also for your patient and courteous attention during its delivery.

Clinical Lectures

ON THE

BORDERLAND OF EPILEPSY:

VERTIGO.

DELIVERED AT THE NATIONAL HOSPITAL FOR THE PARALYSED AND EPILEPTIC.

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LECTURE I.

GENTLEMEN,—Epilepsy is a many-sided malady. The prominent symptoms that attend its various forms differ widely in character, and some of them are common to it and diseases that are of different nature. Some of these maladies resemble epilepsy closely, and their distinction from it may be difficult. It seems sometimes that there is a gradation from the one to the other, and occasionally that an actual transition may occur. It is not surprising that this should be so with such diseases, considering how complex are the functions of the brain, and how close are their interrelations. Functional diseases are often less defined than our definitions. Thus, epilepsy has a borderland of disorders which resemble it in some of its special features, and their distinction from it may be a matter of considerable difficulty. The difficulty may be real, or may disappear with knowledge and consideration. It is important, therefore, to know the features of these borderland cases, the symptoms they present in common with epilepsy, and their distinctive characteristics. They fall into groups according to their nature and dominant