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· THE · BRADSHAW · LECTURE · 1908 ·

ROYAL COLLEGE OF PHYSICIANS

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The Bradshaw Lecture on

MASSIVE COLLAPSE OF THE LUNG.

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London:—J. Bale, Sons and Danielsson, Ltd., Oxford House. Great Titchfield Street, W.

(1908?)

MASSIVE COLLAPSE OF THE LUNG.

Mr. President,—My first duty is to express to you my deep appreciation of the honour you have done me in asking me to deliver the Bradshaw Lecture. When I glance through the names of the distinguished Fellows of this College, who have preceded me in this office, and when I recall some of the admirable discourses they have pronounced in this place, I am almost painfully conscious of the great responsibility which your kindness has placed on my shoulders, and of my unfitness to do proper justice to the occasion. In one circumstance, at least, I esteem myself fortunate, that the subject I have chosen falls within the department of medicine with which your name, Sir, will always be associated, and in which you have made important contributions to our knowledge.

Massive collapse of the lung may at first sight appear a somewhat well-worn theme to bring before you on an occasion such as this,

but the particular variety of the condition I desire to bring to your notice, that, namely, which is related to great deficiency or absence of respiratory muscular effort, is rarely mentioned in medical literature, and does not appear to be generally recognized. Moreover, a subject of this nature cannot be altogether inappropriate at a time when the mechanism of respiration is receiving much attention and our knowledge of its processes is undergoing a gradual transformation, after having remained almost unchanged for nearly half a century.

Much of this recent advance is due to the work of anatomists and physiologists, but the most important factor has undoubtedly been the introduction of X-ray methods of investigation. So far clinicians have contributed but little to it. One reason for this is, without doubt, the inherent difficulty of drawing exact conclusions as to the state of the lungs by physical examination alone, and another, that hitherto the difficulty of using radiography in the examination of patients suffering from acute diseases has been very great. But these difficulties have already been to some extent overcome, and are being surmounted so rapidly that we may confidently look forward, in the near future, to obtaining accurate skiagrams in most cases. An experience of nearly twelve

months work with a portable modification of the orthodiagraph in the wards convinces me that the method will prove of the greatest value, precisely where our present means are inadequate, namely, in determining the exact position and extent of lesions. I hope to show this afternoon that clinical observations, strengthened by the testimony of the *post-mortem* room, throw an important light on some of the views recently advanced on the mechanism of respiration, and to bring forward evidence in support of the view that disturbance of respiration is a factor of considerable importance in the causation of post-operative lung complications.

Among the anatomists, no one has of late years done more to arouse interest and stimulate enquiry into the processes of respiration than Dr. Arthur Keith, and even though we may hesitate to accept every one of the views he has put forward, there can be no two opinions about the great value of his contributions to this subject. The time at my disposal will only permit me to refer to a few of the points to which he has directed attention.

He holds that the elasticity of the lungs and the elastic recoil of the ribs play a far less important part in respiration than is usually assigned to them in current teaching, which

regards expiration mainly as a non-muscular act. "That the elasticity of the lung is concerned in expiration, and that there is a certain degree of aid obtained from the torsion of the cartilages when the thorax has been placed in an extreme position of inspiration, are well-ascertained facts, but it is also certain that the application and action of that elasticity are controlled by the expiratory muscles. The elasticity of the lung is soon injured if the lung, after removal from the body, be repeatedly inflated, even if only to a moderate degree; the elastic recoil from the torsion of the cartilages must be slight, for if the muscles be stripped from the thorax of a perfectly fresh subject, it will be found that on holding the specimen head downwards the thorax assumes a position of full inspiration; when held feet downwards, the ribs sink into an expiratory position . . . . It is most improbable that the law of reciprocal innervation of antagonistic muscles, which Sherrington has shown to be valid for so many groups of muscles, should not also hold good for the muscles of respiration. . . . The most difficult cases to explain, if the theory that expiration is always under muscular control be true, are those cases where the spinal cord is severed below the exit of the phrenic nerve;

and where the diaphragm alone keeps up respiration.[1]

Some of the opinions just quoted are somewhat at variance with current views, and as they are to some extent based on observations made under abnormal conditions, will require confirmation. As regards the law of reciprocal innervation, the muscular mechanism of respiration is of such a peculiar character that it seems to me at least conceivable that it may prove to be an exception to it. On the other hand, everyone will agree that the rate of expiration in the breathing of effort, as in speaking and singing, is and must be controlled by muscular agency ; but that does not involve the admission that pulmonary elasticity has no appreciable share in the process. It is, to my thinking, as difficult to imagine expiration taking place without pulmonary elasticity as without muscular action. The two agencies, the one automatic, the other largely under voluntary control, are both necessary. To regard pulmonary elasticity as taking "only a passive part in expiration" seems to imply an emptying of the lung by muscular compression, which is not admissible. In ordinary quiet breathing, where elastic recoil of the ribs, according to

[1] *British Medical Journal*, August 29, 1908.

Keith, should be negligible, the rib movement that takes place under muscular action enables the elasticity of the lung to have free play, but is not in itself the direct cause of the contraction of the organ. In coughing, sneezing, &c., the case is obviously different; here a definite muscular compression of the thorax takes place.

I shall have occasion, later on, to quote cases in which respiration was carried on by the unaided diaphragm, which have a direct bearing on this question. In singing, the importance of the muscles in expiration is well recognized, for the education of this muscular control is not the least important part of the training of singers and public speakers.

The current teaching on respiration does not always lay sufficient stress on the fact that the lungs do not expand equally in all directions. It is important to bear in mind that in inspiration the lung cannot expand upwards, backwards or inwards; in these directions expansion can only take place indirectly, that is, by enlargement of the opposite wall. The apex of the lung, for example, is expanded mainly by the descent of the diaphragm. Of the three surfaces of direct expansion the diaphragmatic is by far the most important. In women with a well-marked costal type of breathing, it is

generally held that the anterior or ventral wall is of more importance in expanding the lung than the diaphragmatic. It is to be noted, however, in this connection, that the examination of healthy women with the orthodiascope shows that in quiet breathing the inspiratory excursion of the diaphragm is commonly 12 mm. or more, which by itself is probably more than sufficient to supply the requisite amount of air to the lungs. The third surface of direct expansion, the lateral, is generally held to be the least important. It is said that in many people, especially men with a purely abdominal type of respiration, there is no outward movement of the thorax. This is no doubt true, but I have been told by well-known teachers of elocution that the pupils who breathe best from the point of view of voice control and facility of training are those who naturally expand the chest laterally in respiration, and that the teaching of this form of breathing is usually necessary in the training of those who do not habitually use it. The reason of this is not obvious; it is not unlikely that lateral expansion of the lower ribs enables the abdominal muscles to control expiration more effectually.

Keith properly insists on the importance of recognizing the division of the thoracic walls,

on physiological grounds, into three zones—the dorsal, lateral, and ventral. The dorsal or spinal segment of a costal arc is that part to which the spinal muscles are attached. The angle of the rib marks the junction of the spinal with the lateral segments. These spinal segments are really lateral levers of the spine, and only indirectly concerned with respiration. They represent the axis round which a costal arc rotates. The muscles attached to them are essentially muscles for spinal movements.

The lateral segments are nearly on the same plane when the thorax is viewed from above. They form the peculiar flattened area which is obvious on examining any articulated thorax. Beginning at the 4th rib, they increase both in length and obliquity as far as the 8th; below this they diminish in length and have disappeared at the 12th rib.

The characteristic of the ventral segments of the costal arcs is that each succeeding segment from above downwards is part of a greater circle than the one above, in contrast to the lateral segments. The cartilages are included in the ventral segments; beyond the 8th rib these segments cease to exist.

The part of the lungs lying beneath the ventral segments is the costal surface of the upper and middle lobes of the right lung and

the upper lobe of the left lung. The elevation of the ventral segments causes a direct enlargement forwards of the underlying area of lung, and an indirect expansion of the lateral surface. The lateral segments, by their outward movement in inspiration, cause direct expansion of the lateral surface, and by increasing the antero-posterior diameter from the 4th to the 8th rib, contribute indirectly to the expansion forward of the ventral surface of the lung.

We are indebted to the same observer for the information that there are certain structural differences between the 2nd, 3rd, 4th and 5th pairs of ribs on the one hand, and the 7th, 8th, 9th and 10th pairs on the other, whilst the 6th pair is always intermediate in structure. This distinction is found to hold good in the thorax of all orthograde primates. "In the upper set of ribs, when the thorax is viewed in profile, each rib is seen to be bent, with its lower margin showing a convexity from the angle to the costo-chondral junction; the convexities are directed downwards to the 6th rib. In the lower set the upper border is convex, the convexity being directed upwards to the 6th rib. The upper set of ribs is seen to be raised by a "bucket-handle" movement, thus expanding the chest laterally. The lower set undergo no such movement; they are long levers articulated

at their vertebral ends; when their ventral ends are raised they lead to a great lateral enlargement of the abdominal wall, but only to a very partial enlargement of the real pulmonary space. That is to say, while the upper set are direct lateral distenders of the lung, the lower set are made accessory to the diaphragm. They provide a fulcrum for the diaphragm: their lateral movement is designed to make room for the abdominal viscera displaced by the diaphragm, rather than cause a direct enlargement of the lung."[1]

Keith further assigns important respiratory functions to the abdominal muscles. Following the division just outlined, they also fall into dorsal, lateral, and ventral groups. "The rectus and the triangularis sterni are the respiratory opponents of the interchondrals—the intercostals of the ventral segments. The external oblique is the respiratory opponent of the external intercostals, the chief muscles of the lateral wall of the thorax. The internal oblique is a functional complement to the internal intercostals—they are parts of the same physiological sheet, and are expiratory.

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[1] "Contribution to the Human Mechanism of Respiration," *Proc. Anat. Soc. of Great Britain and Ireland*, May, 1903.

All the muscles of the dorsal segment, quadratus lumborum, spinal segment of the external intercostals, levatores costarum, accessorius, cervicalis ascendens, are essentially spinal muscles; the only part they play in respiration is as fixing or synergic muscles; they steady and restrain the spinal segments of the ribs round which the lateral or real respiratory segments of the ribs turn."

The anatomical views which I have just put before you bring out certain points regarding the movements of the lungs that are worthy of notice. The upper lobes, we have seen, are directly influenced by the movements of the ribs, their direction of expansion being mainly forwards and upwards, whereas the expanding force of the diaphragm tells more immediately on the bases, although it also exerts its influence with diminishing intensity as high as the apex of the lung. The direction of this expansion is downwards and forwards. It may therefore be stated in very general terms that the expansion of the lungs takes place under the influence of two forces, and that the overlapping between their respective spheres of action roughly corresponds to the position of the great fissure: the costal force acting mainly above and the diaphragmatic force mainly below it. It will be seen directly that this

view is supported both by clinical and pathological observations.

The surfaces of direct expansion and the action of the respiratory forces on the lungs are clearly shown in figs. 1, 2 and 3, which I owe to the kindness of Dr. Keith.

I turn now to the influence of failure of the muscular apparatus of respiration on the state of the lungs.

In his treatise on diseases of the lungs and pleura, Wilson Fox classifies the causes of collapse of the lungs under the following heads :—

(1) Congenital non-expansion.

(2) Imperfect power of expanding the thorax.

(3) Obstruction of the bronchial tubes and air passages.

(4) Pressure on the lung by fluids or solids.

(5) Pneumothorax, with a free opening in the chest wall or in the lung.

The variety of massive collapse with which we are concerned falls under the second head—imperfect power of expansion—and the greater part of our knowledge concerning it has been derived from the study of cases of paralysis of the diaphragm.

The history of the growth of our knowledge of the functions of the diaphragm is not without

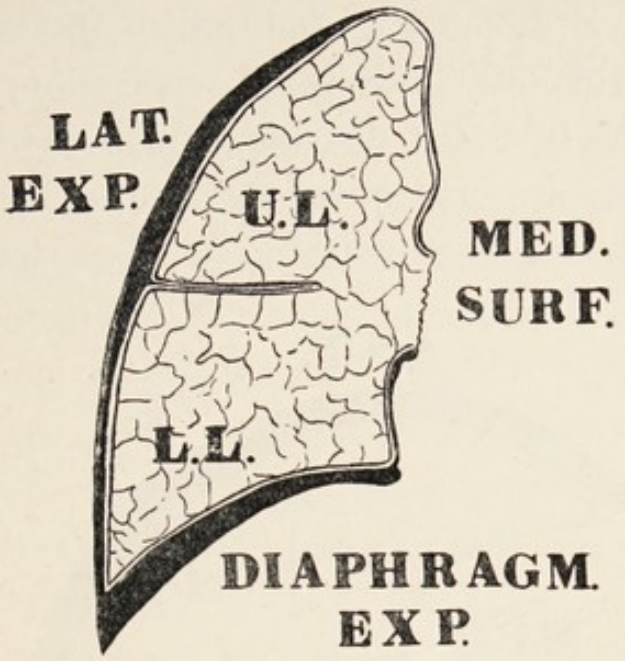

FIG. 1.—Vertical section in a transverse plane of the right lung (after Keith). The blackened areas represent the amount and direction of expansion due to inspiration.

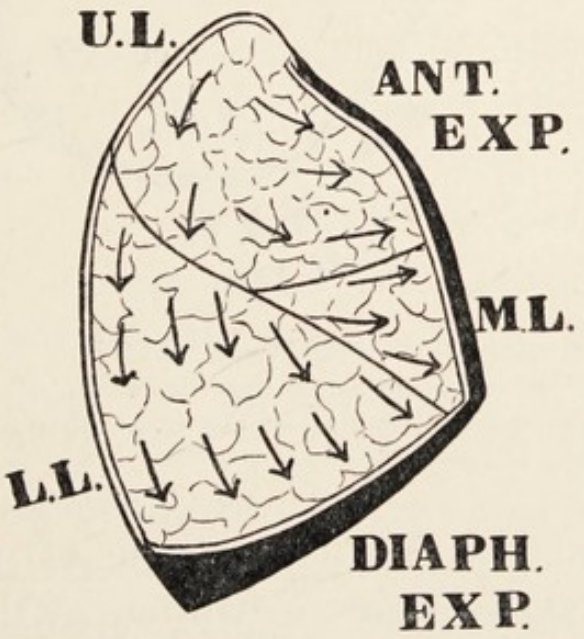

FIG. 2.—The right lung viewed from the side (after Keith). The blackened areas represent the expansion of inspiration. The arrows show the direction in which the expanding movements are effected.

interest. The early anatomists thought that it was the only muscle of respiration. Galen was the first to discover the existence of other respiratory muscles, as he was also the first to demonstrate by experiment on

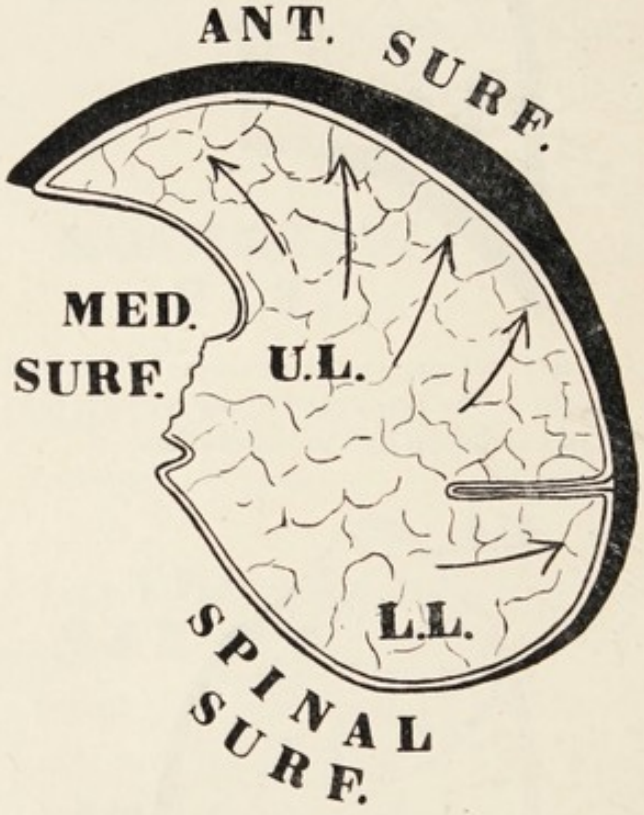

FIG. 3.—Horizontal section of the right lung (after Keith). The blackened area indicates the expansion of inspiration. The arrows show the direction of the inspiratory movement of the pulmonary tissue.

living animals that contraction of the diaphragm alone causes expansion of the lower thoracic zone. Vesalius explained the uplifting and expansion observed by Galen by assuming that during contraction the dia-

phragm rises upwards into the thorax, drawing up the ribs to which it is attached. This view does not appear to have gained many adherents. Throughout the seventeenth and eighteenth centuries the question appears to have attracted but little attention. In 1833 Magendie again enunciated the elevating action of the diaphragm, pointing out for the first time that this action on the lower ribs must be dependent on support from below, that is, on the upward pressure of the abdominal viscera. But it was reserved to Duchenne to prove the correctness of this hypothesis some twenty years later. In a remarkable series of observations on men and animals, both during life and immediately after death, before the extinction of electrical excitability, he was able to demonstrate by stimulation of the phrenic nerves that the outward movement of the lower ribs is determined by the pressure of the abdominal contents on the under surface of the diaphragm. He also explained how the upward movement of the ribs is accounted for by the fact that the hollow dome of the diaphragm being entirely occupied by the more or less resisting mass of the abdominal viscera, its muscular bundles are maintained for the most part in an almost vertical position, so that

their contraction causes movement in an upward direction. He showed, further, that in an eviscerated animal electrical stimulation of the phrenics causes contraction instead of expansion of the lower thoracic zone. A clinical application of these facts is afforded by Wenckebach's[1] observation that the respiratory troubles of the subjects of marked enteroptosis can readily be explained by the interference with proper diaphragmatic action which is caused by removal of the visceral support.

The last great advance in our knowledge of the processes of respiration has been due to the advent of radioscopy. Since the introduction of the orthodiagraph, which eliminates the error due to magnification of the shadow, it has become possible to obtain accurate records of the size and position of the thoracic viscera in the different phases of respiration. With the aid of this instrument, Dr. J. F. Halls Dally[2] has carried out a very comprehensive and important series of observations on the respiratory movements of healthy adults of

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[1] "Sammlung klinischer Vorträge," neue Folge, Leipzig, 1906.

[2] *Proc. Roy. Soc.*, B, vol. lxxx., 1908, and *Proc. Anat. Soc. of Great Britain and Ireland*, October, 1908.

both sexes. He finds that in quiet respiration the total average movement of the diaphragm is 12·5 mm. on the right side and 12 mm. on the left; whilst in deep breathing the total average range is 30 mm. on the right side and 28 mm. on the left. During the course of the last twelve months I have had the opportunity of examining a considerable number of cases with Mr. Cecil Lyster, and our results agree closely with those of Dr. Halls Dally. There are also strong reasons for believing that the central tendon of the diaphragm, which was formerly regarded as immovable, moves freely in respiration; and Halls Dally, among others, has drawn attention to the fact that the curvature of the diaphragm undergoes very little change in its descent. Supported below by the abdominal viscera, it acts somewhat like a piston, pushing them forwards and downwards towards the epigastrium, at the same time expanding the lower ribs. If the descent of the viscera is prevented by contraction of the abdominal muscles, the effort of the diaphragm is expended chiefly on the lower ribs, raising them and pushing them outwards.

The expanding force of the diaphragm, then, is exerted mainly on the portion of the lungs lying below the great fissures, that is, on the

lower lobes, and a consideration of the cases in which the diaphragm has become paralyzed shows very clearly what happens to the lungs when this expanding force is in abeyance.

Respiratory paralysis is a comparatively frequent complication of diphtheria, and when the muscles of respiration are involved in this disease it is rare for the diaphragm to escape. In a series of sixty-four cases of diphtheritic paralysis observed by myself,[1] the diaphragm was affected in no less than twenty-eight, of which fifteen ended fatally, twelve recovered, and one was lost sight of. An autopsy was performed in eight cases. The following is a very brief summary of the condition of the lungs found after death.

H. H., aged 4, male. The diaphragm had been paralyzed for six or seven days. Right lung: Lower lobe of a deep blue colour, entirely devoid of air. The lung does not crepitate, and is soft and unduly wet. On section a few small patches of broncho-pneumonia are exposed; there is recent pneumonia at the back of the right upper lobe. The left lower lobe is affected like the right, though in a less degree. The remainder of the lungs is crepitant and œdematous.

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[1] *Cf. Clinical Transactions*, vol. xxviii.

W. M., aged 2½, male. Admitted with some signs of paralysis of the diaphragm : died four days later. Clinically this case resembled broncho-pneumonia, and the signs of diaphragmatic paralysis were not well marked. There were patchy areas of collapse over both lungs, with slight general dilatation of the bronchi, which exuded pus on pressure.

A. T., aged 3½, female. The diaphragm became paralyzed eight days before death. Right lung : Lower lobe deep purple in colour and quite airless ; sinks entire in water. Left lung : Scattered areas of collapse ; some of considerable size, some broncho-pneumonia.

M. V., aged 4, female. Paralysis of diaphragm of ten days duration. Both lungs very œdematous. Lower lobe of right lung of a deep purple colour, tough and airless ; sinks in water. Lower lobe of left lung partly collapsed. Emphysema of upper lobes.

A. E., aged 5½, female. Diaphragm paralyzed four days. Right lung : Lower lobe completely collapsed and of a uniform deep purple colour. Upper lobe also totally deflated, with the exception of the extreme apex. Left lung : Lower lobe extensively, but not entirely, collapsed.

E. B., aged 6, male. Admitted during a cardio-pulmonary crisis ; no signs of respiratory

paralysis. There were well-marked signs of asphyxia, but no pulmonary collapse.

D. E., aged 4, male. There was some paralysis of the diaphragm two days before death, but the muscle had recovered some power during the last twenty-four hours of life. There were emphysema of the upper lobes and some small patches of collapse over the backs of both lungs.

A. E., aged 2½, male. Admitted with paralysis of the diaphragm seven days before death. Right lung: Extensive collapse of the whole of the posterior portion of the lung, the lower lobe being affected almost in its entirety. Pieces of collapsed portions sink in water. The left lung is in a precisely similar condition.

In three of these cases there was no massive collapse. In one of them the evidence of paralysis was indefinite, in the second there was no paralysis of the diaphragm, and in the third the signs of paralysis had disappeared at least twenty-four hours before death. In the remaining five cases the diaphragm had been paralyzed for several days before death. In each of these the lungs were profoundly affected. The part chiefly involved was the lower lobe, which was rendered totally airless in four out of the five cases; and it will be noted

that the right base always suffered more than the left.

The evidence from these cases is conclusive as to what happens to the lungs when the diaphragm ceases to contract, and they lend strong support to the view that the expanding force of the muscle is exerted mainly on the lower lobes. The greater vulnerability of the right base is probably accounted for by its anatomical relations.

The clinical evidence of massive collapse is not quite so definite. It is proverbially difficult, in many cases of disease within the chest, to decide whether the physical signs presented are due to compression, consolidation, or collapse of the lung. When the state of the patient permits of a careful examination, the signs observed in massive collapse are loss of percussion resonance, sometimes amounting to actual dulness, with weak breathing, which is often bronchial or tubular. Œdema râles may also be present, or crepitations when there is associated pneumonia.

Curiously enough, the cases in which physical signs have been most marked have recovered, so that in them the final proof is wanting. But these were, for the most part, instances of affection of the upper lobes from paralysis of the intercostals. The cases fall roughly into

two groups, according as the costal or the diaphragmatic respiratory force is put out of action. In the former event the signs are mainly apical, in the latter they are limited for the most part to the bases. More rarely a combination of intercostal and diaphragmatic paralysis may give rise to physical signs over the whole of one lung. Two examples will suffice to illustrate the physical signs of massive collapse.

I venture to quote here a case reported in 1876 by Pearson-Irvine,[1] as it appears to me to afford convincing evidence of massive collapse on clinical grounds alone, and is, as far as I know, the first reference ever made to this condition. A girl, aged 6, had had diphtheria a few weeks previously. Her present symptoms began rather suddenly with short, frequent cough and considerable dyspnœa. She was feverish the first two or three days, but had suffered no pain. When first seen she was dull and listless of aspect, with a queer look about the eyes, and a drooping head. The temperature was normal, the respirations (14) shallow, slow, but not laborious. She had a continuous cough, as though to clear the throat. There was complete flattening of both

[1] *Clinical Transactions*, vol. ix.

infraclavicular regions, with almost complete absence of movement. The flattening was extensive and uniform, and it appeared as though the upper part of the chest had fallen in. The bases expanded unusually fully, and there was a strong action of the diaphragm. There was marked loss of resonance above and under the clavicles as low as the 3rd ribs, and on each side toward the sternum it terminated at the level of the 2nd ribs in absolute dulness, in what appeared to be an increased area of cardiac dulness. In the supraspinous fossa there was what appeared to be absolute dulness, but the rest of the back was resonant, the bases, indeed, being abnormally so. Every variety of bronchitic râle could be heard. At the apices there were loud sonorous râles, not concealing what seemed to be true tubular breathing. There was clear whispering pectoriloquy at both apices.

Four days later she was again brought to the hospital, having been worse in the interval. The chest signs were unchanged, the temperature remained normal. Pulse 60, respirations 14, no distress. The head drooped more, and there was a decided left strabismus. The paralysis of the trunk and limbs was more marked. The flattening of the chest was pointed out to the mother, who at once stated

that the child had been remarkably full-chested before the present illness. Ten days after this there was a striking change. The head was all but erect, the squint had all but disappeared, the walk was no longer ataxic, and the child was quite cheerful. The improvement occurred quite suddenly. There was now an increase of resonance below the clavicles, and a distinct diminution of dulness around the cardiac area. The breathing was tubular at the right apex, weak elsewhere, with a complete absence of râles. The upper regions of the thorax still moved imperfectly. A fortnight later she was to all appearance quite cured. The subclavicular regions still moved imperfectly, but had become more prominent. Tubular breathing persisted for some time longer. Pearson-Irvine rightly considered that the physical phenomena observed at the upper part of the thorax were due to paralysis of the muscles concerned in the elevation and expansion of those parts.

J. H., a boy, aged about 4, was under my care in 1886 for diphtheritic paralysis of the trunk and limbs. There was no respiratory paralysis at first, but it is stated in the notes that the movements of the ribs appeared to grow less day by day. On the seventeenth day after admission respiration suddenly became embarrassed.

Examination of the chest within a few hours of the onset of these symptoms showed that, with the exception of the two or three upper ribs, the whole of the right half of the chest was practically immobile. The percussion note was much impaired over the right front and right base. The breath-sounds were loud and tubular over the upper lobe, weak over the middle lobe, and almost inaudible at the base. Behind, above the level of the angle of the scapula, the breathing was bronchial. The temperature was raised one degree. On the following day there was exaggerated breathing on the left side and weak tubular breath-sounds all over the front of the right lung. The movements at the epigastrium were reversed. During the next two days the temperature rose to 102° F. and the condition of the patient became very serious ; but, quite unexpectedly, on the twenty-first day there was a sudden improvement, which coincided with a return of movement in the right chest and the rapid disappearance of the signs of diaphragmatic paralysis. Examination of the back revealed tubular breathing over the apex and upper part of the lower lobe, and distant weak breath-sounds at the extreme base. Four days after this the two sides of the chest moved equally, and on the twenty-eighth day there was no tubular breathing on the right side.

There is nothing distinctive in the physical signs themselves, and it is often only by considering a case in all its bearings that a diagnosis can be made. In the two cases I have just related, the clinical picture is so striking and suggestive that there is no room for doubt; but when the affection is more limited, or less obtrusive, or when the patient is too ill to stand a thorough examination, the diagnosis may present considerable difficulty. The most important diagnostic indication is the association of paralysis of the muscles of respiration, for massive collapse apparently never takes place in its absence.

The signs of paralysis of the diaphragm are too well known to need recapitulation here, but I should like to refer to one symptom which does not appear to have attracted notice. Exaggerated action of the lower ribs occurs, in my experience, in the majority of cases and is one of the early indications of diaphragmatic failure. It is generally most obvious when the paralysis is well marked, and invariably disappears when function is restored. The phenomenon is not easy to account for, and is moreover to some extent paradoxical, as one action of the diaphragm is to raise the lower ribs.

I am not at present able to offer a satis

factory explanation of this phenomenon. It would seem to suggest one of two things: either that the removal of diaphragmatic influence, which may be to some extent antagonistic to the intercostals, confers greater freedom of movement on the lower ribs, or that it is a genuine attempt on the part of the intercostals to compensate the lungs for the loss of diaphragmatic expansion.[1]

In paralysis of the intercostals no sign is of greater value than flattening of the chest wall. This is unilateral or bilateral, according to circumstances, but is always present when paralysis is profound. It was a striking feature in Pearson-Irvine's case, and no doubt was present also in mine, although the notes are silent on the point. Were it necessary I could quote many other cases in support of this statement.

As the lungs are normally maintained in a state of expansion by the action of muscular force on the thoracic cage, it follows that massive collapse can only take place when this force has been reduced to such an extent that the elasticity of the lungs is enabled to "take

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[1] *Cf.* "Respiratory Paralysis after Diphtheria," by W. Pasteur, *American Journal of the Medical Sciences*, September, 1890.

charge." Consequently the condition is only met with in cases of profound paralysis, and for the most part in cases of paralysis of rapid onset. Whenever the loss of respiratory muscular power falls short of this point, there is a deficiency of lung expansion exactly proportionate to the lessening of muscular power. This condition is quite familiar to everyone, and is often loosely described as partial collapse.

I had expected to find instances of massive collapse in the *post-mortem* records of cases of myasthenia gravis, but a careful search has only been rewarded by the discovery of a single case, that of a patient who had some severe choking fits and urgent dyspnœa before death. "The lungs contained air throughout, except the base of the left lower lobe, which was plum-coloured and sank in water." [1]

Another clinical feature of some value is the rapid onset of symptoms, and the equally prompt improvement which occurs on the restoration of function. The case of the boy J. H., previously mentioned, illustrates this point. The symptoms he presented might well have been mistaken for those of pneumonia,

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[1] Quoted by E. F. Buzzard, *Brain*, vol. xxviii., 1905, p. 458.

but for the rapidity with which they became fully developed, in the absence of any marked febrile reaction. It is probable, however, that the subsequent aggravation of the constitutional symptoms was due to the onset of secondary broncho-pneumonia.

It follows from the data I have placed before you that the mechanism of massive collapse differs materially from that of patchy or lobular collapse. The latter condition, even though it lead to the deflation of a whole lobe, as in a case recently reported by Samuel West,[1] is probably always caused by obstruction in some part of the air-passages. The difference between them lies in the fact that in massive collapse due to muscular weakness all the evidence goes to show that it occurs in the absence of any bronchial obstruction. It goes without saying that the two conditions may, and often do, occur side by side ; indeed, any degree of deflation of the lung is likely to promote the occurrence of lobular collapse in the presence of any source of obstruction in the air-way ; but they differ in their mode of origin. Massive collapse is essentially an active process, lobular collapse a passive one, for the former is brought about by the active

[1] *Lancet*, February 15, 1908, p. 489.

exercise of the elastic property of the lung, whilst the latter is generally believed to be caused by a passive removal of the air from portions of lung which have been cut off by bronchial occlusion from communication with the external air.

We have been concerned hitherto chiefly with the effects of paralysis of the diaphragm, and it will be useful to contrast with them the characters of respiration when this is mainly or entirely carried on by that muscle. I have had the opportunity of examining two cases of this nature within the last few months.

A man, aged 20, was admitted to hospital with acute ascending myelitis. There was complete paralysis of the legs and trunk muscles, and total loss of sphincter control. The front of the chest was abnormally flattened, the interspaces depressed and not filling up in inspiration. There was no sign of intercostal action, but the scalenes seemed to harden very slightly during inspiration, respiration being almost entirely diaphragmatic. The abdominal muscles were paralyzed. The heart was uncovered, its impulse being visible over a large area, reaching up to the second interspace. In inspiration there was no visible movement of the manubrium, and the upper part of the chest, instead of expanding, actually

diminished in circumference by $\frac{1}{8}$ in. This contraction was noticeable as far as the 5th rib, but below this level there was a slight but definite inspiratory expansion of from $\frac{1}{8}$ in. to $\frac{1}{4}$ in., and slight elevation of the lower six ribs. The movements at the epigastrium were normal, but of very limited extent. The abdomen was rather full, and the prominence of the epigastric region in particular was unusual. The breathing was rather hurried, but regular.

The second case was that of a sturdy boy, aged 7, suffering from diphtheritic paralysis. On admission to hospital there was slight inspiratory expansion of the upper ribs, but on the third day paralysis of the intercostals was complete. The upper six ribs were motionless, there was no forward movement of the manubrium, the front of the chest was very flat and the interspaces depressed. In inspiration the lower six ribs were visibly raised, and the circumference in the meso-metasternal plane increased by $\frac{7}{8}$ in. The movements at the epigastrium were well marked and normal in kind, The state of the upper half of the thorax offered a striking contrast. At the level of the 2nd and 3rd costal cartilages expansion was replaced by an inspiratory lessening in girth of from $\frac{1}{2}$ in. to $\frac{5}{8}$ in. This reversal of the normal movement was gradually lost at the 5th rib

cartilage, the normal movements being clearly marked from the 7th rib downwards. The abdominal muscles were healthy. The upper part of the abdomen was full and prominent. An orthodiagram of this patient is shown in fig. 4.

These are the only two cases in which I have, so far, had the opportunity of observing

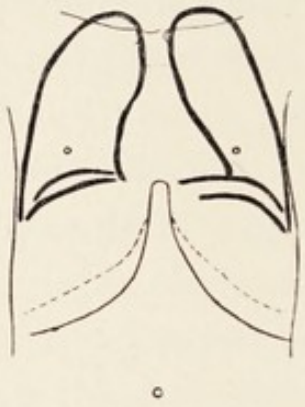

FIG. 4.—The horizontal dark lines show the position of the diaphragm in inspiration and expiration in quiet breathing. The vertical movement of the diaphragm, measured at a point midway between the edge of the transradiancy of the lung and the median line, is 9 mm. on the right and 13 mm. on the left. The dotted line shows the amount of elevation of the lower costal border in inspiration.

reversal of the usual respiratory movements of the upper set of ribs in paralysis of the intercostals; but I expect it will be met with, if looked for, whenever paralysis is profound, and it may prove to be a diagnostic sign of some value. In a sense it is the exact counterpart

of what occurs at the epigastrium when the diaphragm is paralyzed. It follows from what has been already said that in cases of this kind, when paralysis of the intercostals is complete, there can be no direct expansion of those portions of the lungs which lie beneath them. The ventral segments of the ribs which expand them are inert; the costal respiratory force is in abeyance. Under these circumstances the upper lobes are in a state of more or less complete collapse, and the upper six ribs in the position of expiration; meanwhile the bases are expanding and contracting under the influence of the diaphragm alone. But why should the upper ribs fall in still further in inspiration?

It is not easy to account for this phenomenon. The only explanation that occurs to me involves the acceptance of the view that the root of the lung moves freely in respiration, and is not, as was thought until quite lately, practically fixed. A good deal of evidence has of late been accumulating in favour of this, and although it cannot yet be said that respiratory movement of the root of the lung in healthy individuals has been rigidly proved, it has reached a high degree of probability. Keith has given anatomical reasons of much weight in support of it, and he points out

with truth that as there is little or no direct expansion of the lungs upwards and backwards, if the root is fixed there can be no expansion, in the absence of rib movement, of the part of the lung lying between the root and the apex, and between the root and the posterior surface. He concludes from his observations that "with an ordinary inspiration the base of the heart and the roots of the lungs move forwards and downwards about half an inch; with pure diaphragmatic breathing, the movement is almost entirely in a downward direction; in costal breathing the movement is a forward one."[1]

Assuming, then, the movement of the root of the lung, I conceive the explanation to be as follows. During inspiration, the root of the lung, by carrying down with it the unexpanded apex, develops a certain amount of negative pressure in the upper part of the thorax; and as it is evident from the facts observed that the apex does not expand, the ribs fall in still further to satisfy it. In other words, the amount of negative pressure created under these circumstances is insufficient to enable the intrapulmonary pressure to overcome the

[1] Discussion at Medical Society, London Hospital, October, 1903. See also "Hunterian Lectures on the Nature and Anatomy of Enteroptosis" (Keith).—*Lancet*, March, 1903.

elasticity of the lung. A consideration of the several cases I have brought to your notice justifies another conclusion which bears on this point, namely, that when one of the two chief respiratory forces is completely destroyed, the other is unable to prevent that portion of the lung which has thus been deprived of its direct expanding power from collapsing. Thus in complete paralysis of the upper ribs the expanding power of the diaphragm is not sufficient to prevent collapse of the upper lobes (as in Pearson-Irvine's case), whilst the condition of the bases in the fatal cases of diaphragmatic paralysis testifies to the inability of unaided costal breathing to maintain the expansion of the lower lobes. One cannot fail to be struck by the close correspondence of the difference in function between the upper and lower sets of ribs exhibited by these two patients with the difference in structure to which allusion has already been made.

NOTE.—The evidence in favour of the movement of the root of the lung cannot be more succinctly stated than it is in the letter which Dr. Halls Dally kindly sent me in reply to my request for his views on this subject: "On X-ray examination of a subject in the lateral-oblique position two transradiant triangles are seen, retrosternal and retrocardiac respectively, between which is a shadow somewhat

triangular or wedge-shaped, the base of the wedge resting on the central tendon of the diaphragm. This shadow is a composite one, being made up of shadows on heart, pericardium, great vessels, root of lung, and other mediastinal contents, and on inspiration the mediastinum as a whole is seen to be pulled downwards and forwards, the triangles at the same time increasing in size and clearness, since the vertebral column also executes a backward movement. With a tube giving good definition, one can to some extent single out the structures which go to form the main shadow, although never with great clearness, because most X-ray shadows are superimposed. This movement of the mediastinum is produced by the crura of the diaphragm pulling downwards and forwards upon the posterior part of the central tendon. The supplementary evidence is anatomical. As long ago as 1860-62, John Hilton, in his classical lectures on Rest and Pain, pointed out that the pericardium may be regarded as the "fascial insertion of the diaphragm." Each of the two layers of the pericardium has its several function, the external layer being composed of several laminæ of white and yellow fibres, mainly longitudinal and inelastic. The base of this layer is united firmly to the upper surface of the diaphragm, and inseparably with the central leaflet of the central tendon at the circumference of the caval aperture, also in lesser part with the adjacent muscular surface, especially on the left side, being strengthened all round the periphery. This stout aponeurosis is continued upwards over the heart into the sheaths of the great vessels, becoming

blended with the adventitia of the two pulmonary veins and pulmonary artery on each side, and with the superior vena cava, aorta, and obliterated ductus arteriosus above. This piece of anatomy I have often verified. Until quite recently it was always thought that the central tendon on inspiration remained unaltered in position, and if this were the case no movement downward of the lung-root would be possible; but from X-ray appearances, and from watching the diaphragm contract in the living rabbit, as I have done, one finds that the crura (the right crus especially) of the diaphragm on contraction pull down the whole of the mediastinal structures, and in the cadaver, if one throws boiling water on the under surface of the diaphragm, as Dr. Keith has done, or by exercising traction downwards on the crus, the structures above are pulled downwards, and to such an extent that one can see the skin of the neck often indrawn."

Confirmation of the movement of the root of the lung may also be obtained by recording under X-rays the behaviour of shadows situated at or near the root of the lung. The shadow of a cretaceous gland, for instance, is usually well defined, and can be easily seen and measured. Mr. Lyster has recently examined such a case for me, taking instantaneous skiagrams in full inspiration and in forced expiration. The patient in question, a young and healthy-looking man, had had an attack of slight hæmoptysis with cough five months before, and had just completed an open-air cure. There were no physical signs of disease in the lungs, but on X-ray examination there was a small shadow

(probably a tubercular patch) at the right apex, two deeper shadows near the root of the right lung, and some loss of transradiancy at the left base. The darker of the two shadows at the root lay at the level of the fourth interspace, about 1½ inches from the median plane and ⅜ inch nearer the anterior than the posterior wall of the chest.

The measurements show the respiratory excursion of this shadow to have been 3 mm., whilst that of the fourth rib was 2 mm. Identical measurements were obtained from a second set of skiagrams.

It goes without saying that observations on cases of this kind only afford evidence of the movement of the root of the lung ; they give us no indication of what the extent of that movement is in healthy persons, though we may reasonably expect it to be greater than in individuals whose lungs are diseased and possibly adherent to the parietes.

The case of the reversed epigastric movements in paralysis of the diaphragm is not susceptible of quite the same explanation. For here the descent of the root of the lung —which is determined by diaphragmatic contraction—does not take place ; the pulmonary bases are more or less collapsed, and respiration is carried on mainly by the upper lobes under costal action ; further, the abdominal cavity is enlarged owing to the higher level of the diaphragm. The inspiratory recession observed is in part a negative effect, owing

to the fact that there is no inspiratory descent of the contents of the upper abdomen, whilst the lower ribs are being raised to a variable extent by the action of the intercostals. Similarly the expiratory filling up at the epigastrium is largely due to a return of the parts to their previous position. Nevertheless it is quite possible that, in those cases where rib movement is excessive, some negative pressure is developed in the lower part of the thorax during inspiration, which the collapsed lung cannot expand to meet, so that the flaccid diaphragm is pushed further up into the chest. Holzknecht[1] quotes a case of Kienbock's in which such a "paradoxical" movement of the diaphragm was observed under the X-rays. He attributes it to the fact that "the paralyzed muscle offers no resistance to the inspiratory action of the thorax, being elevated, by aspiration, like a membrane." Clearly this is a point which will be settled by radioscopy. In the only two cases I have had the opportunity of examining the paralyzed diaphragm was motionless,

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[1] "Die röntgenologische Diagnostik der Erkrankungen der Brusteingeweide ('Archiv und Atlas der normalen und pathologischen Anatomie in typischen Röntgenbildern')," *Fortschritte auf dem Gebiete der Röntgenstrahlen*, Ergänzungsheft 6, p. 198.

but I should add that in both there was only slight movement of the lower ribs.

There is one more feature of these two cases to which I would draw your attention, namely, the upward movement of the lower ribs during inspiration. This is not due to intercostal action—for the intercostal muscles were paralyzed—but illustrates an important action of the diaphragm. The diaphragm may be compared to a lever of the first order, of which the fulcrum is the underlying liver and abdominal viscera. When the vertebral portion of the muscle is pulled downwards by the crura, the costal attachments are drawn upwards over the liver, at the same time elevating the ribs. So that the movement observed in these cases is the clinical counterpart of Duchenne's electrical stimulation of the phrenic nerves immediately after death.

A case that was recently under my care throws an interesting light on this action of the diaphragm.

A man, aged 48, was admitted to hospital for mitral regurgitation with right heart failure. There was marked orthopnœa with paroxysmal attacks of "bad breathing," during which cyanosis became profound. The right heart was enlarged, and the apex beat outside the

mammary line with an apical systolic bruit. There were no signs of kidney disease. The breathing was laborious, and on uncovering the chest it was noticed that, while the upper ribs and manubrium moved about normally, there was excessive epigastric protrusion with inspiration. On placing the hand over the epigastrium the forcible downward push of the diaphragm on the viscera could be clearly appreciated. At the same time there was a visible contraction of the lower ribs during inspiration, which caused a well-marked Harrison's sulcus to appear. There was no percussion dulness over the lungs; the breath-sounds were obscured by loud rhonchi. There was no discoverable cause, during life, for the non-entry of air to the bases. The larynx was not diseased, and skiagrams did not reveal the presence of any source of pressure on the bronchi, nor could the condition be put down to the enlargement of the heart. The dyspnœa was temporarily relieved by blood-letting, but the patient died of asphyxia eighteen days after admission. The following is a transcript of Dr. C. E. Lakin's notes of the autopsy:—

"On opening the thorax the heart was seen to be considerably enlarged and the lungs rather small. On closer examination this was seen to be largely due to a considerable loss

of bulk of the lower lobes, so that when the lungs had been removed from the body the conical form of the lungs was no longer apparent; the base of the cone which is normally formed by the lower lobes being even narrower than the parts above. Although both lower lobes showed partial collapse, this was most marked on the left side. . . . The lung substance was congested, and crepitation was much less marked in the lower lobes than in the upper. There was but slight emphysema, and no bullæ were seen; there were no definite signs of bronchitis, and no bronchial obstruction. There were no pleural adhesions. The liver was congested and showed a nutmeg appearance on section. Anchoring the liver to the under surface of the diaphragm were a number of adhesions, which were tough and had to be divided with the knife. They were situated on the antero-superior aspect of the right lobe, extending in a horizontal line from the falciform ligament to the lateral aspect of the right lobe. There were also a few adhesions of the extremity of the left lobe of the liver, connecting the latter with the diaphragm. Adhesions also existed between the lateral aspect of the right lobe of the liver and the body wall. The aortic valves showed extensive disease . . . the

mitral valve admitted three fingers, the tricuspid four. The heart muscle was soft. The kidneys appeared to be healthy."

It is proverbially unwise to draw conclusions from insufficient premisses, but it is difficult to resist the inference that the non-expansion of the bases in this case was due to the interference of the adhesions with the proper action of the diaphragm. The fixation of the liver to the diaphragm must have greatly hampered, if it did not entirely prevent, the contraction of the crura from drawing up the costal attachments of the muscle, an action to which we have seen that the elevation and expansion of the lower ribs are in part due. At the same time the presence of adhesions would not appreciably interfere with the downward displacement of the abdominal viscera by the contracting muscle. So that, in effect, the contraction of the diaphragm in this case tended to deprive the lower ribs of the visceral support, the absence of which Duchenne has shown to cause contraction instead of expansion of the lower thoracic zone.

The observation that definite pulmonary lesions may be caused by failure of the diaphragm to expand the bases of the lungs led me to enquire whether this agency might

not in some cases be responsible for the chest complications which sometimes follow severe operations.

In considering this group of lesions two points must always be borne in mind : (1) that in a large majority of cases they follow operations on the abdominal cavity ; (2) that the bases of the lungs are peculiarly liable to be affected. The enormous increase in the number of abdominal operations during recent years has necessarily directed attention to these accidents, and many explanations have been put forward to account for them. Sepsis is a factor in many cases, so that it is not surprising to learn from surgeons that every fresh step towards securing more complete aseptic conditions at the time of operation has been attended by a diminution in the number of post-operative lung complications. That they are still very far from rare must be well known to every hospital physician. But septic infection alone, whether through the lymphatics or by aspiration through the lungs, will not explain the common situation of these lesions at the bases or along the posterior borders of the lungs. The reason for this is more likely to be discovered among circumstances which are peculiar to abdominal operations than in conditions which are common to

all operations. It is not likely, therefore, that the effect of the anæsthetic on the bronchial mucous membrane or the undue exposure of the patient are factors of special importance. There is, perhaps, more to be said of the harmful effect of long continuance of the Trendelenburg position, by causing visceral pressure on the diaphragm. Moynihan,[1] who has paid considerable attention to this matter, also suggests that the practice of giving large saline injections for shock may sometimes provoke acute œdema of the lungs. Bibergeil[2] bases the following opinions on an analysis of over 3,900 abdominal operations practised in Körte's clinic: "Careful study of the collected cases of post-operative pneumonia has led to the rejection of the views that this complication may be due to infection by way of the lymphatics, and to such causes as exposure to cold of the surface of the body, or of the peritoneal cavity to abdominal irrigation and to direct action of the anæsthetic. The lobular form, or broncho-pneumonia, which is frequently met with after laparotomy, is regarded as being usually the result of auto-infection, due to aspiration of secretions from the mouth and pharynx, whilst the patient is under the full

[1] "Abdominal Operations," second edition, 1906.

[2] Quoted by Moynihan, *loc. cit.*

influence of an anæsthetic. It is pointed out that the interference with free breathing and expectoration, resulting from pain at the seat of operation, and impeded movements of the incised abdominal wall, must favour very much the development of lung disease after laparotomy, whilst resistance to the inflammatory attack is in many cases much impaired in consequence of the enfeebled condition of the patient." Bibergeil clearly suspects that loss of functional activity of the lung is a factor in the causation of post-operative pneumonia, but whether this is due to the influence of wound pain in diminishing the movements of the abdominal muscles is an opinion which requires proof.

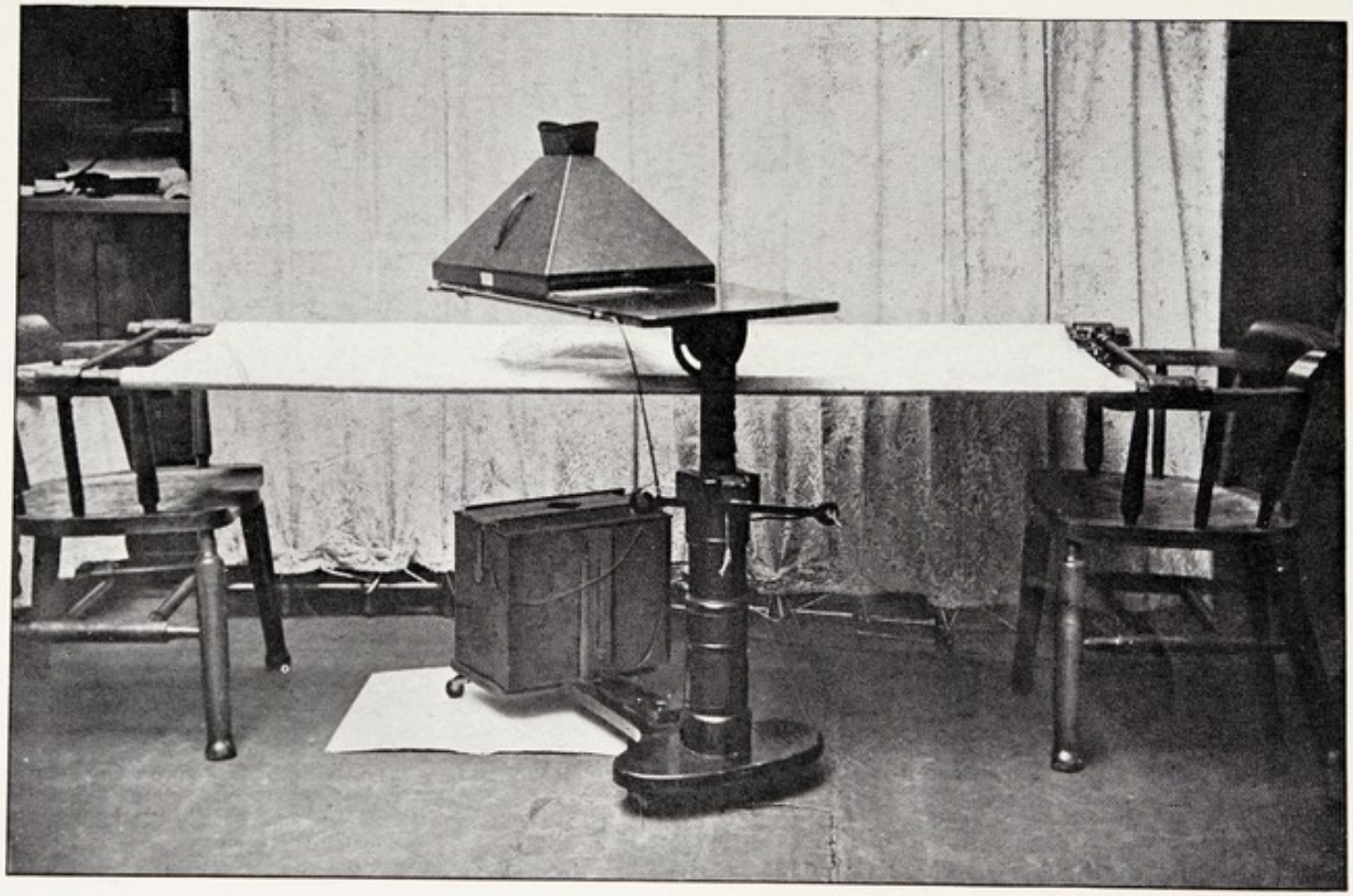
The facts ascertained with regard to the effect of paralysis of the diaphragm on the lungs, coupled with the knowledge that post-operative lesions are often situated in that portion of the lungs which is especially under diaphragmatic influence, led me to think that these lesions might sometimes be accounted for by interference with the proper action of that muscle. The probability of this hypothesis will depend on whether evidence can be adduced to show that the activity of the base of the lungs is diminished from this cause after surgical operations, and whether

there are any grounds for believing that lessened functional activity renders the lung more liable to infection. The evidence in favour of the latter contention is somewhat conflicting. The incidence of tubercle at the apex, and the tendency of terminal infections to be localized in the dorsal region of the lungs, may be cited in favour of it; but on the other hand, compression of the lung by a tuberculous pleural effusion does not predispose it to tubercular disease. I had hoped to be able to bring forward some experimental evidence on this point, but this work is not yet sufficiently advanced to permit of any conclusions being drawn.

With regard to the other point—whether there is any evidence of interference with the movements of the diaphragm after surgical operations on the abdominal cavity—I have failed to find any information. The nervous channels for reflex inhibition of movement are fully provided for in the distribution of the phrenic nerves to the diaphragm, and of the vagi to the adjacent organs; and also by the subpleural plexuses between the phrenic and intercostal nerves. In the hope of obtaining reliable information on this point, I have examined patients under the X-rays shortly after the performance of

gastro-enterostomy, but the number of cases placed at my disposal by the kindness of my surgical colleagues is as yet hardly sufficient to justify the drawing of positive conclusions.

The ingenious apparatus which was devised for this purpose by Mr. Cecil Lyster is in reality a rough-and-ready portable orthodiagraph (see plate). The slight loss of accuracy in the results obtained, as compared with those of the fixed orthodiagraph, is more than compensated by the great advantage of being able to use the instrument at the bedside, thereby largely extending the range of its usefulness. The instrument shown in this photograph was made from a bed-table. A piece has been cut out of the top of the table and a fixed point provided by means of two wires stretched across the opening. Vertically below the point at which the wires cross is placed the centre of the antikathode of the fluorescent tube, and below this again, so that it will write on the floor, an automatic pencil, which is worked from the side of the table. The whole apparatus is designed to move easily in any direction on castors. By using a fluoroscope, instead of an ordinary screen over the opening in the table, patients can be examined in ordinary daylight. The patient is placed on a canvas stretcher, which can be passed under him like



PORTABLE ORTHODIAGRAPH FOR WORK AT THE BED-SIDE.

a draw-sheet, with a minimum of disturbance, the poles of the stretcher being afterwards slid into position and tension secured by iron cross-pieces at the head and feet. The stretcher is then lifted into a suitable position, usually between two beds, a piece of cartridge paper

FIG. 5.—The upper tracing was taken before, and the lower one on the fourth day after gastro-enterostomy, during quiet breathing. The vertical movements of the diaphragm at a point midway between the edge of the lung transradiancy and the median line are respectively 17 mm. and 10 mm. on the right side and 18 mm. and 13 mm. on the left.

pinned to the floor, and the apparatus wheeled into position. The tracings are obtained by carrying the fixed point round the shadow margins of the organ, releasing the pencil at frequent intervals. The dotted figures obtained in this way may be transferred to

another sheet of paper and joined up into continuous lines, as in the diagrams I have placed on the screen.

The orthodiagrams shown in figs. 5, 6 and 7

FIG. 6.—Upper tracing taken before, lower tracing on fourth day after gastro-enterostomy. Vertical movements of diaphragm respectively 20 mm. and 23 mm. on the right side and 12 mm. and 18 mm. on the left. The dotted transverse lines show the position of the diaphragm in deep inspiration and forced expiration.

were taken from male patients. The upper tracing represents the excursion of the diaphragm in quiet breathing before operation; the lower one shows the same movement on

FIG. 7.—Upper tracing taken before, lower tracing on fourth day after gastro-enterostomy. Vertical movements of diaphragm respectively 16 mm. and 12 mm. on right side, and 24 mm. and 17 mm. on the left. The dotted transverse lines show the position of the diaphragm in deep inspiration and forced expiration.

FIG. 8.—Tracing taken on fourth day after gastro-enterostomy. There was no movement of the diaphragm in quiet breathing. The excursion shown, 7 mm. on either side, represents the greatest movement obtained in forced breathing.

the fourth day after gastro-enterostomy. It is clear that in these three patients, if the operation had any influence at all on the movements of the diaphragm, it must have been of short duration; but it is probable that it was unaffected throughout, as the movements of respiration were quite natural as soon as the patients had recovered from the anæsthetic. The orthodiagram shown in fig. 8 was also taken from a man on the fourth day after gastro-enterostomy. There was no movement whatever of the diaphragm in quiet breathing. The small excursion shown represents the maximum of movement developed when the patient was asked to take a deep inspiration. This man was in a rather weak state before operation, but it is stated in the notes of the case that the breathing was both abdominal and thoracic, and that air entered well into every part of the lungs. We may safely infer, therefore, that the diaphragm was acting normally before operation. His subsequent progress was rather slow for the first ten days, but there were no indications of lung trouble at any time.

These cases do not take us very far, but everything must have a beginning, and I am very hopeful that tracings obtained in this way from patients with post-operative lung

symptoms will yield important information. We have, at any rate, positive evidence in Case 4 that the function of the diaphragm may be seriously interfered with after laparotomy, without sepsis and in the absence of marked pain, or of any indication of lung trouble.

Further light is thrown on this subject by *post-mortem* records. In the six years 1901-06, in fifty-five autopsies after operations on the abdominal cavity at the Middlesex Hospital, collapse of the lung is mentioned in only eight cases. In two of these it was probably of the massive variety. One was a case of appendix abscess with general peritonitis, in which the right lower lobe was collapsed; the other was a case of general peritonitis after operation for gall-stones, in which both lower lobes are described as being deeply congested and containing but little air. During the last twelve months, on the other hand, I have met with no less than four well-marked instances of massive collapse in the *post-mortem* room. One of these occurred after laparotomy; the others were medical cases.

A woman, aged 49, underwent abdominal hysterectomy on May 5. There was repeated severe vomiting after the anæsthetic, but beyond this everything went well for the first

three days. On the fourth day the temperature rose suddenly to 103°, F. without rigor. Some friction and percussion dulness were made out low down in the right axilla. The diagnosis of pneumonia was made. When I first saw her, two days later, she was sitting up in bed, looking desperately ill. The face was dusky and sallow, with slight malar flush, the pulse small, 144, and the respirations 44. The temperature had remained over 103° since the onset of symptoms. The percussion note was much impaired all over the right lower lobe, and over the same area the breath-sounds were weak and tubular, without râles or pleural friction sounds. The voice sounds were unaltered and moderately well conducted. At the left base there was weak breathing with occasional fine inspiratory râles. There was very little movement of the chest, but the epigastrium appeared to move normally. Death occurred on the following day. There was general peritonitis reaching up to the lower border of the liver. The right pleural cavity contained 3 oz. of thin pus, and there was much lymph over the lower lobe, reaching up behind as high as the apex. The whole of the right lower lobe was in a state of massive collapse, with the exception of a small wedge at the anterior-inferior border. When this was

cut away the rest of the lobe sank in water. The posterior portions of the middle and upper lobes were also totally collapsed. The left base was almost as profoundly affected, the lower lobe only just floating, and feeling almost airless under the pressure of the fingers. There were no signs of pneumonia or broncho-pneumonia.

It will be observed that the extent and degree of collapse are as great as in any of the cases due to paralysis of the diaphragm, and that the area affected closely corresponds with the sphere of influence of that muscle. It is very difficult to account for such a state of the lungs after an illness of three days duration, except on the supposition that there was reflex inhibition of the movements of respiration of sufficient intensity and duration to cause massive collapse of the lungs. We have here, then, conclusive evidence that this condition may follow laparotomy, and that the symptoms presented may very closely resemble those of pneumonia. It is not unlikely, therefore, that some of the cases diagnosed as post-operative pneumonia are in reality instances of massive collapse.

The other three cases can be related in very few words.

A woman, aged 47, very sallow and

emaciated, was admitted for chronic gastric ulcer with agonizing gastralgia. She was too ill to justify operative interference. Two days before death she had a severe attack of breathlessness, accompanied by a rise of temperature to 101° F. On examination there was dulness, with well-marked tubular breathing, and a few crepitations over the left base. *Post mortem* there was a partial hour-glass contraction, with a chronic ulcer on the small curvature. The left lower lobe was airless, and sank in water, with the exception of the extreme apex. There were a few broncho-pneumonic areas in the collapsed portion. There was no pleurisy.

A woman, aged 48, the subject of biliary colic, died with symptoms of pyæmia. The liver weighed 151 oz. There were recent adhesions between it and the diaphragm. The upper surface showed a number of projecting abscesses, which burst as soon as the diaphragm was raised from the liver. There was a thin fibrinous exudate over the right lower lobe, which was collapsed and airless, except the extreme apex. The pleura contained half an ounce of bile-stained fluid.

A woman, aged 30, admitted in a state of profound collapse after taking hydrochloric acid, died within ten hours. The œsophagus,

the stomach and the duodenum were extensively damaged. The stomach was acutely inflamed and soft, but had not perforated. Both lungs were quite small and in a state of general incomplete collapse.

The striking feature about these cases is that in every one of them there was a powerful source of irritation in the near neighbourhood of the diaphragm, either within the stomach itself or in the adjacent structures, within the area supplied by the vagus. The association of acute pleural inflammation in several of the cases at first led me to think that pleural pain was the determining cause of the inhibition of the diaphragm. That this is a contributing factor in some cases is probable, but that it is not indispensable is shown by the occurrence of massive collapse in the absence of any indications of pleural inflammation. I am disposed to attach more importance to the presence of acute disease below the diaphragm, if only for the reason that the evidence at present available shows that massive collapse is most often related to diaphragmatic failure, and that there are no reasons for supposing that inflammation of the pleura—with the possible exception of its diaphragmatic surface—ever leads to an arrest of diaphragmatic action so intense as to enable massive collapse of the bases to take place.

Common events, we know, are apt to be persistently overlooked until attention is specially directed to them, and I think this saying may be applied with some truth to the condition I have described to you this afternoon. If it has not been actually ignored, the importance of massive collapse has been underrated, whilst its clinical significance has been for the most part overlooked. I have endeavoured to place before you the reasons which lead me to conclude that under certain conditions an active collapse of the lung takes place, which may lead to entire deflation of one or more of its lobes. It is generally caused by paralysis of the muscles which are the direct distenders of the lungs—the diaphragm and the intercostals —but I have also shown that there are strong grounds for believing that reflex inhibition of diaphragmatic movement may lead to the same result, and that this observation may throw some light on the nature of post-operative lung complications.