Three lectures on respiratory exercises and pulmonary physics / by Harry Campbell.

THREE LECTURES

ON

RESPIRATORY EXERCISES AND PULMONARY PHYSICS.

BY

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PREFACE.

I AM led to publish these lectures because in them are set forth for the first time certain principles which I believe to be important.

H. C.

23, WIMPOLE STREET, W.;
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RESPIRATORY EXERCISES AND PULMONARY PHYSICS.

LECTURE I.

GENTLEMEN,—In order to understand the subject of respiratory exercises as a means of treating disease it is needful to know something about what one may term "Pulmonary Physics," a subject, I venture to think, of great importance to the physician. The respiratory apparatus presents many purely physical problems, the understanding of which not only enhances our interest—at least I have found it so—in diseases of the chest, but, as I think, our means of combating them also. For this reason I propose to devote most of my remarks in these lectures to a consideration of the physical problems involved. I shall make the lectures as informal as possible, and I hope no one will hesitate to put questions that may suggest themselves on points which I do not make clear. In this lecture I shall first say a few words as to the shape of the chest and the changes it undergoes in

development; I shall then make a brief reference to the pleura and the movements of the lungs; after which I shall pass on to a consideration of the muscles of respiration; and finally I shall deal with the methods by which the chest can be altered in size.

First, as to the shape of the chest. Here I have a series of diagrams illustrating the changes which the chest undergoes in shape from fœtal life onwards. I show you a diagram of the chest at the third month of fœtal life. Taking 100 to be the transverse diameter, the sagittal or antero-posterior diameter at this period is represented by 116, and we say that the thoracic index is 116. Thence onwards the chest flattens sagittally: at the fourth month it is 110; at the seventh month 104; at birth it is 100, the sagittal and transverse diameters being then exactly equal; by the age of five years the chest has flattened so considerably from before backwards that the index is now 85, and the flattening process continues till adult life is reached, when the index is 70; after this the thorax begins to deepen sagittally, and in old age, when the individual is apt to become emphysematous, its shape tends to return to that of the new-born child,—that is to say, to become round. Now you are familiar with the fact that in quadrupeds the thorax is compressed laterally; it is deeper sagittally than transversely, the scapulæ being placed laterally rather than posteriorly, obviously for the purpose of facilitating

the backward and forward movement of the fore-limbs in progression. And there is no doubt, I take it, that in the change in shape which the human thorax undergoes during development we have, as in so many other instances, an epitome of man's evolution, or, as we say, of his phylogenesis, —that is to say, man has evolved from a creature which progressed on four legs, from a being whose chest was, like the chests of the dog and the ape, deeper sagittally than transversely. The sagittal flattening of the human chest during development is no doubt connected with man's upright position: the child as it comes into the world is allied to the quadruped, and the chest is round, but as the upright position is assumed the thorax flattens sagittally so as to throw the centre of gravity vertically above the narrow bases of support afforded by the feet.

There is a variety of chest termed the "flat chest," which is not, however, as a matter of fact, a flat chest at all, but actually deeper than the normal, its index being about 80. This was pointed out by Professor Woods Hutchinson in a learned and philosophical course of lectures delivered here to an audience all too small. He showed that the flatness in these cases is only apparent, and is due to the forward position of the shoulder girdle, which makes the upper part of the chest seem to be flat when viewed anteriorly; for if you measure the so-called "flat chest" you will find that it is deeper than the normal

chest. The same is true also of a large number of phthisical chests; if you compare their diameters you will frequently find that the sagittal diameter is above the normal, and that the thoracic index is about 80 instead of the normal 70. I shall show in due course that this is due to expansion of the thorax from preponderating action of the inspiratory muscles.

I now ask your attention for a moment to the pleura. I would in the first place remind you that this membrane extends upwards about three quarters of an inch beyond the clavicle; that the two pleuræ meet in the middle line in front, from the second to the fourth pair of ribs; also that they do not extend downwards to the extreme limit of the costal arch, one of the reasons for which being that the diaphragm takes its origin from the inner aspect of the ribs forming that arch: in the nipple line the pleura extends to within about one and a half inches of the costal arch, and in the axillary line to within about two and a half inches; posteriorly, near the spine it goes down to the last rib, or even below it; the left pleura is somewhat lower than the right.

The functions of the pleuræ are twofold, the first being to allow the lungs to expand and contract equally in all directions during the respiratory movements: suppose, *e. g.*, the lungs were agglutinated to the diaphragm and to the lower chest walls, it is obvious that a diaphragmatic breath would lead to an expansion of the lower part of the lungs

alone, the upper portions remaining wholly unexpanded; and such an arrangement would be most injurious, for it would lead to an undue stretching of the alveoli at the base of the lungs. By means of the pleuræ, however, this difficulty is obviated, for they enable the lungs to expand more or less equally in all directions when a diaphragmatic breath is taken, as anyone can easily test for himself by means of a binaural stethoscope: if you listen with this to the breath-sounds at the pulmonary apices during diaphragmatic breathing you will hear a murmur quite as distinctly there as over the lower parts of the chest, and though I would not like to say that the lungs expand with this kind of breathing as freely in the upper portions as they do in the lower portions, it would be difficult to prove otherwise.

The lungs tend during inspiration to travel forwards and downwards: during a deep inspiration they cover the heart, and they travel downwards to the lowermost limit of the pleuræ, the movement being greatest in their lower portions. When, therefore, adhesions form here, breathing is more interfered with than when they occur higher up. I have said that the lungs tend to move down to the extreme limits of the pleuræ in a complete inspiration, but although, as we have seen, those limits do not reach as far as the costal arch, one nevertheless gets a resonant note at the extreme limit of the costal arch at the termination of a complete inspiration. I percuss over my liver at the

extreme limit of the costal arch in the nipple line, and get, as you hear, a dull note ; I now, while continuing to percuss, take a deep inspiration, and you observe how the note varies in tone, how the dulness gets less and less, and how the resonance steadily deepens until it ultimately becomes very pronounced, although all this time I am percussing over the liver beyond the lung—namely, over the edge of the costal arch ; now, as I let my breath go, the note you will observe gets dull again. For some time this puzzled me ; I could not tell why there should be resonance here over the liver where no lung intervenes. I have little doubt that the explanation lies in the fact that the costal arch is lifted up from the liver during deep inspiration, and thus yields a resonant note.

The second great function of the pleura is to serve as a lymphatic sac ; each constitutes a large lymph-space. By the movements of the diaphragm the lymph is pumped from the peritoneal cavity through the diaphragm into the pleuræ, and from the pleural cavities into the lymphatics of the pleura, thence into the bronchial glands, and so into the lymphatic ducts. The movements of the lungs aid the flow of lymph from the pleuræ onwards, and thus facilitate the absorption of lymph and fluid from the pleural sacs ; and in this connection it must not be forgotten that the pulmonary lymphatics are provided with valves. Hence the lack of respiratory movement observed in abundant pleural effusions may hinder the

absorption of fluid, and sometimes after removing a small quantity of the fluid the rate of absorption is increased, owing, it would seem, to the increased freedom of movement permitted.

In regard to the muscles of respiration, I shall only refer to those which are of practical importance. First of all we have the muscles which dilate the nares. The nares tend to dilate with every inspiration, and in all cases of dyspnœa this dilatation is apt to be exaggerated. Especially is this the case with children; if in them you find marked movement of the nostrils you may be sure that there is urgent dyspnœa, and you may with tolerable certainty diagnose an urgent affection of the chest, such as cardiac failure, or—especially if the breathing is very rapid—some acute pulmonary affection, such as pneumonia, large pleural effusion, or acute phthisis. A knowledge of this fact is, clinically, very helpful; if a child is breathing quietly, with no movement of the nostrils, you may be perfectly certain, simply by looking at it, that there is no acute trouble of the lungs or heart; if, on the other hand, the alæ nasi are working vigorously, you may be pretty certain that some urgent cardiac or pulmonary trouble is present, very rapid breathing pointing to the latter.

The next inspiratory muscles which I shall speak of are the abductors of the vocal cords. During inspiration the glottis opens in order to facilitate the ingress of air, and when a deep inspiration is taken it opens very considerably; hence paralysis

of the abductors is accompanied by very serious inspiratory dyspnœa: there is great difficulty in getting the breath in, and but little difficulty in getting it out. This condition is happily very rare.

We now come to the important group of inspiratory muscles situated in the neck. They are—on either side—the sterno-mastoid, the trapezius, and the muscles in the floor of the triangle formed by them and the clavicle—namely, the scalene muscles (which, you may remember, pass from the cervical vertebræ to the upper two ribs), the levator anguli scapulæ—the omo-hyoid stretching across the triangle, and finally, the platysma. All these muscles are, in dyspnœa, thrown into vigorous contraction. As regards the sterno-mastoids, it is important to note that they do not come into inspiratory action until the end of an inspiration, as anyone can prove for himself: if you take a deep inspiration you will find that it is only towards the very end of it that the sterno-mastoids become firm; and this is a point of some practical importance, implying as it does that wherever in dyspnœa you see the sterno-mastoids standing out, the dyspnœa is extreme. With regard to the platysma, for some reason not easy to explain this muscle is thrown into considerable action in dyspnœa, and in chronic dyspnœics it may get considerably hypertrophied. I do not quite see how it can help inspiration, but there is the fact; you may observe the sheet of muscle,

especially in elderly, lean subjects, tightly stretched under the skin, from the lower jaw to the upper part of the chest. Often, too, one can see the omo-hyoid rhythmically starting out in dyspnœa.

Clinically, the most interesting of all the respiratory muscles, if we except the diaphragm, are, in my opinion, the scalene muscles. In all cases of dyspnœa you find these muscles acting powerfully; if upon placing the hand above the clavicle you feel them working vigorously, you may be sure that there is dyspnœa, and you may measure its extent by the vigour of their contraction; and this is true no matter how quietly the patient may seem on superficial observation to be breathing. All these cervical muscles tend, in chronic dyspnœa, to shorten, and so to fix the chest in a position of expansion. I shall go into this matter in a future lecture, when I shall point out a fact which does not appear to have been generally recognised—namely, that in practically all cases of dyspnœa there is a preponderant action of the inspiratory muscles, one of the results of which in the case of the cervical muscles is that they shorten, and thus tend to fix the chest in a position of inspiration, so that the individual cannot empty his lungs properly, as is well seen in old cases of emphysema, in which the chest is fixed in a position of super-extraordinary inspiration. Physicians have not seriously set themselves to explain this thoracic fixation; I believe myself that the explanation lies in the fact that the inspiratory muscles shorten from

prolonged over-action, just as happens in the case of certain muscles in talipes.

Next we will take the thoracic muscles of inspiration. Those I would direct your attention to are (on either side) the pectoralis major and minor, the serratus magnus, and a few fibres of the latissimus dorsi. You will remember the latter muscle arises from the crest of the ilium and from some of the spines of the lumbar vertebræ; it also takes origin from the last three ribs at the side, and the fibres springing thence elevate these ribs when the humerus is fixed. The pectorals and the serratus also require that the humerus and scapulæ shall be fixed in order that they may act as inspiratory muscles. This inspiratory action is increased by the elevation of the shoulders, a movement which renders the inspiratory fibres of these muscles more vertical—less horizontal—and thus increases their efficiency as costal elevators. I show you on the screen the portrait of a little girl with very serious heart disease; she has an enormous heart; its transverse diameter must be eleven or twelve inches, and, as may be well imagined, she suffers from urgent dyspnœa. Observe how the shoulders are elevated so that the outer ends of the clavicles are considerably higher than the inner, and how the arms are kept close to the body with the hands pressing down on the bed so as to fix the shoulder girdle. Notice how prominently the lowermost fibres of the pectoralis major stand out in this position, forming

a hypertrophied band, the result of prolonged over-action.

Let us now turn to the abdominal muscles. All the anterior abdominal muscles are expiratory—the external obliques, the internal obliques, the transversales, and the recti. Other expiratory muscles are, on either side, the quadratus lumborum which arises from the ilium and sends some of its fibres to the last rib, and certain fibres of the erector spinæ, the outer part of which sends processes to the last five or six ribs, and is thus obviously expiratory; then there is another interesting expiratory muscle, the serratus posticus inferior, which springs from the spines of the lumbar vertebræ and passes upwards and outwards to the last two or three ribs.

In regard to the anterior abdominal muscles, there is one pair which strike me as being especially interesting, and the action of which appears to have been overlooked. I refer to the transversales. These muscles, I may remind you, arise below from the crest of the ilium and the outer third of Poupart's ligament, and above from the last six ribs, while the middle fibres take their origin from the lumbar fascia which is attached to the spine. It is these middle fibres which are of special interest: the upper fibres by their contraction obviously tend to draw together the sides of the costal arch, and the lower fibres to flatten somewhat the anterior abdominal wall, but the contraction of these middle fibres brings the

anterior into close contact with the posterior abdominal wall; in other words, the transversales muscles retract the abdomen. By actively retracting the anterior abdominal wall considerable pressure is exerted upon the abdominal viscera, and that is done mainly by the transversales, which are thus very interesting muscles.

Under normal conditions the abdominal muscles are moderately contracted—that is to say, in a state of tone,—and they exercise a moderate pressure on the underlying viscera. This is one of the functions of the abdominal muscles—to support and keep in proper position the stomach, liver, and other abdominal viscera. When these muscles are weak and flabby, as happens, *e. g.*, in sedentary people, in women who have worn stays and have thus prevented the abdominal walls from developing properly, in subjects in whom the abdomen has been greatly distended by fluid or fat which has become absorbed,—in all such cases as these the anterior abdominal walls are flaccid, and fail to exert a due amount of pressure upon the underlying viscera, which in consequence tend to move downwards: there is, in fact, a ptosis of the viscera which tends with every added year to get more and more pronounced. Now this downward movement is apt to lead to a dragging upon and stretching of parts—as, for example, the mesentery, —and thus to give rise to a series of unpleasant symptoms such as backache and abdominal discomfort.

Another disadvantage of having flabby, ill-developed abdominal walls is that the accumulation of blood in the splanchnic veins is thereby favoured. Most of you are familiar with the work of Leonard Hill, who has studied the influence of gravitation upon the circulation ; he has pointed out that in the upright position the blood tends to gravitate into the most dependent parts of the body, and especially into the great splanchnic veins, which alone, it will be remembered, are capable of holding the entire blood-mass. In order to counteract this a vaso-motor constriction of the splanchnic arteries takes place; I should expect a similar constriction also to occur in the arteries of the lower extremities, though I do not know that Leonard Hill has studied this point. In some this compensating mechanism is especially efficient, enabling them to stand in the upright position without feeling faint for a longer time than others ; in others it is feebly developed, and such are very apt to get tired and faint from standing, owing to the tendency of the blood to accumulate in the splanchnic veins.

Vaso-motor contraction does not, however, constitute the sole factor in compensating the effects of gravity on the circulation: the abdominal walls constitute a second line of defence. When these are firmly contracted the blood is much less apt to rush into the splanchnic area than when they are relaxed, for you will remember that all the veins in the body are highly compressible, being in this respect very different from the arteries,

which are comparatively incompressible; by pressing upon the belly it is quite easy to squeeze the blood out of the splanchnic veins, while the blood in the corresponding arteries is but little affected. You can therefore readily understand how, when a person has well-developed abdominal walls, and those walls are always kept firmly contracted upon the splanchnic veins, there is much less tendency for the blood to accumulate in the splanchnic area, and for the individual to become faint, than when those walls are feeble and flaccid. We have here, no doubt, one of the reasons why stays are so popular; doubtless they are worn largely because they are supposed to improve the figure, but they are also undoubtedly worn for "support." If a woman has been in the habit of wearing stays which exert pressure on the abdominal wall as far as the umbilicus, and has thus allowed an artificial support to take the place of Nature's own stays as constituted by the abdominal muscles, especially by the transversales, she becomes dependent upon this artificial support to exert that firm and constant pressure upon the belly which in the upright position tends to retain the abdominal viscera in position and to prevent the blood from gravitating into the splanchnic veins.

The third evil resulting from weak abdominal walls is constipation; nothing, however, need be said on this head here.

I will not take up your time in describing exercises for developing the abdominal muscles,

because suitable ones will readily occur to you. I must content myself with emphasising the importance of securing their adequate development.

We now pass on to consider the means by which the chest can be altered in capacity. There are two chief kinds of breathing—(1) the costal and (2) the diaphragmatic. By means of costal breathing the chest is increased in its sagittal and transverse diameters. Let us consider what happens in a complete costal breath: the clavicles are raised to the utmost, and if I now lift them [here the lecturer demonstrated his remarks] as high as I can, you will observe that they practically touch my chin; at the same time the ribs move upwards and the sternum is carried forward, and the chest is increased in its sagittal and transverse diameters; then towards the end of complete inspiration the spine moves backwards. (This forward bend of the sternum and backward bend of the spine are well observed in the emphysematous chest.) In the upward movement the ribs are bent and altered in their curves, but I shall not attempt to describe exactly what that alteration is, for the simple reason that I do not know, but I hope soon to get a tracing of a rib in the neutral position as well as in the positions of extreme inspiration and expiration; at present I must be content to point out that the ribs undergo considerable change in shape during deep breathing, and that this is needful for the due expansion and contraction of the chest,—that is to say, if the ribs were all per-

fectly rigid it would be impossible for the chest to expand and contract in the normal way: hence the advantage of having elastic, pliable ribs rather than rigid, unbendable ones.

The complete clavicular breath, in which the clavicles are elevated to the utmost, causes the maximum expansion of the lungs—gives the largest "vital capacity." In my book on respiratory exercises I have termed this mode of breathing the "pan-costal," because, though I think the term "clavicular" more accurately describes it, this latter term has been used by singers in a somewhat different sense.

This is one variety of rib-breathing. I have now to direct your attention to a second variety, in which the clavicles are kept fixed, or only allowed to move slightly at their sternal ends, while the ribs are elevated; and inasmuch as it is chiefly the lower ribs which move in this kind of breathing I have called it the "lower costal."

These are the two great varieties of costal breathing, and every one who wishes to understand the subject must learn to execute them himself; certainly, by no other means can he teach them to his patients. Clavicular breathing is easy enough, but it is somewhat more difficult to breathe by the lower costal method.

It is possible to breathe with one side of the chest alone, and it is sometimes advisable to adopt this mode of breathing as a respiratory exercise. In learning it the palm of one hand should be

pressed firmly against the corresponding side of the chest, and the body inclined as far as possible towards that side, which should be kept as fixed as possible, while an endeavour is made to expand the opposite side to the utmost.

Table showing the quantity of air which can be breathed by the different methods.

Clavicular . . .		400 cub. in.
Lower costal . . .		210 ,,
Lower costal supplemented by abdominal . . .		210–270 cub. in. According to the degree of diaphragmatic descent.
Abdominal .	Thorax kept fixed in its mean position .	110 cub. in.
	Thorax kept fixed in position of costal expiration . .	170 ,,
	Thorax kept fixed in lower costal expansion .	90 ,,

This table represents my own breathing capacities when I tested them three or four years ago. The clavicular breath was 400 cubic inches, while the lower costal breath was only 210,—that is to say, very little more than one half. When I take a full clavicular breath you will observe that my epigas-

trium falls in; this is because the lungs are not, under normal conditions, large enough to fill the chest at its potential maximum capacity, *i. e.* when the clavicles are pulled up to their highest and the diaphragm is lowered to its utmost; consequently when a complete clavicular breath is taken the diaphragm tends to be sucked upwards by the lungs, and the costal arch being carried forward, you can understand why there should be a falling in of the epigastrium. Notice how extreme is my epigastric recession at the present moment; so long as I sustain the complete clavicular breath I am unable by the most forcible contraction of the diaphragm to bulge the epigastrium to any but the very smallest degree. Were I emphysematous the case would be different: in advanced emphysema the thoracic cage is often fixed in a position of full clavicular inspiration, and, in spite of this, full diaphragmatic breathing is still possible; indeed, the breathing in such a case may be said to be purely diaphragmatic. The explanation is simple—the lungs in hypertrophous emphysema are greatly enlarged, sufficiently so to fill the chest when the ribs are raised and the diaphragm lowered to their respective maxima.

It will thus be seen that there are two forms at least of epigastric recession—an active recession brought about by the contraction of the transversales; a passive recession due to the suction upwards of the diaphragm following on extreme clavicular breathing.

I now show you on the screen the photograph of a man who holds the world's record for weight-lifting. You see that the muscles of his chest are enormously developed; nevertheless his blow ("vital capacity") is only 270. This shows that great muscular development of the chest does not necessarily mean great vital capacity, and it leads me to say that the stories which one reads and hears regarding the extent to which the chest can be altered in capacity are not altogether reliable. One hears of "strong men" being able to modify their chest-girth by thirteen or fifteen inches. Now, 400 is an exceptional blow, and yet in making it I find I only alter my chest-girth by three inches; and such being the case, those who claim to alter their girth by fifteen inches should be able to blow something like 2000! The fact is that the great alteration in thoracic girth which these men are able to effect is mainly brought about by a cunning disposition of certain extrinsic muscles of the chest, notably of the latissimi dorsi; the chondro-osseous thorax itself can never, except in the case of freaks, be altered in its girth by more than a few inches.

I may observe, in conclusion, that "vital capacity" depends in large measure upon the degree to which the lungs can be emptied of air. With advancing years the chest tends to get bigger, but while the actual air capacity of the lungs thus tends to increase the vital capacity steadily diminishes, the individual becoming less and less

capable of emptying his lung, owing to the increasing immobility of the thoracic cage.

The largest vital capacity I have as yet registered was in the case of a man of thirty (6 ft. 4 in. in height) whose chest measured only thirty-five inches in girth. His blow was 420 cubic inches.

LECTURE II.

GENTLEMEN,—In the latter part of the last lecture I dealt with the subject of costal breathing, and I pointed out, you may remember, that there are at least two kinds of costal breathing, that in which the clavicles are raised to the utmost—the clavicular type—and that in which the ribs are raised while the clavicles are kept fixed or nearly so. In both types all the ribs are raised, but in the latter the lower ones chiefly, for which reason we may call it the "lower costal" type. The degree to which the upper ribs move in this second type of costal breathing depends upon the depth of the breathing: if a moderate breath be taken the upper ribs remain comparatively stationary, but in a complete lower costal breath they move decidedly, the sternal ends of the clavicles being at the same time carried forward and somewhat upward.

We have now to ask whether any other kind of costal breathing is possible—whether it is possible, *e.g.*, for a person to breathe chiefly with the upper ribs, the lower being kept comparatively fixed. Is there, in fact, an "upper costal" type? It is asserted that such a mode of breathing is employed

by some singers, but it does not seem to me to be practicable with the normal unsupported chest. Theoretically it is possible, but practically it is impossible, with the *normal* unsupported chest, except, perhaps, as a laboriously acquired feat, to fix the lower ribs and elevate the upper ribs only. When stays are worn, however, this kind of breathing becomes possible; for if, while the lower part of the chest is fixed by tightly fitting stays, a costal breath be taken, expansion is necessarily confined to the upper part; and this leads me to say a few words on the subject of the corset, and its influence on the chest and on the movements of respiration. Unless worn so loose as to give practically no support whatever, the corset must of necessity interfere with breathing, and if worn before maturity is reached it must obviously interfere with the proper development of the chest also. Suppose the corset is made to fit the chest closely in the position of ordinary mean respiration but without exerting compression—and there are very few corsets which are put on as loosely as this, indeed, I should imagine it practically never occurs, for even those who do not "tighten in" in the ordinary acceptation of the term, yet compress the chest in some degree,—suppose, I say, the corset is worn so loose as just to fit the chest during mean respiration without compressing it, it will nevertheless interfere with adequate expansion of the lower chest, and when the wearer is called upon to take an extraordinary breath she is compelled to expand the

upper part of the chest unduly. The only way in which the corset could be worn without interfering with the normal movements and development of the chest would be by fitting it to the chest in the position of extraordinary inspiration, and if that were done it would, of course, be so loose under ordinary conditions as to be a mere encumbrance. We find in consequence that the thorax of every woman who has worn stays is abnormal, being over-developed and unduly mobile in the upper regions—hence the "heaving bosom" of the woman of the novelette. This state of things is pathological; there is normally little or no difference in the breathing of the man and of the woman, such differences as are observed being essentially due to the use of the corset.

I have said that we may regard "upper costal" breathing as impracticable in the *normal* unsupported chest. That there are, however, some corset wearers who are capable, even when the stays are removed, of breathing chiefly with the upper part of the chest, there can be no doubt. I have such a case now under observation in the person of a girl æt. 17, in whom the upper part of the chest moves very freely in breathing, while the lower part is comparatively immobile. This condition must, however, be regarded as pathological.

We come now to the subject of diaphragmatic breathing. The diaphragm springs from the spine, the lower six ribs, and the xiphoid car-

tilage. From these origins the fibres proceed to be attached to the central tendon ; those from the xiphoid cartilage pass almost horizontally backwards, while the remainder travel in a more upward direction, especially those originating laterally and posteriorly. When the diaphragm contracts, three things happen, or tend to happen : (1) the curves of the fibres which, when in a state of rest, are convex (some markedly so) upwards, straighten out ; (2) the central tendon is pulled downwards ; and (3) the lower ribs are pulled upwards.

In ordinary, quiet, diaphragmatic breathing the first of these three actions is the most pronounced, the central tendon being held in position by the pericardium which is continuous with the deep cervical fascia, while the upward movement of the lower ribs is partially antagonised by the quadratus lumborum and by other muscles which need not be mentioned ; I say partially antagonised, because in an ordinary diaphragmatic breath not only does the abdomen protrude but the ribs are slightly raised ; it is, however, possible with a little practice to fix the lower ribs during the contraction of the diaphragm, and this constitutes a purely *abdominal* breath. You see then that a diaphragmatic and a purely abdominal breath are not quite the same. On the other hand, by fixing the central tendon the diaphragm is enabled to bring about by its contraction considerable elevation of the lower ribs. This fixation may be

effected by actively retracting the epigastrium by means of the transversales muscles, and thus thrusting up the abdominal viscera against the under surface of the central tendon, which being thus rendered fixed, the whole force of the contraction of the diaphragm is spent in elevating the ribs. When the abdomen is retracted and the tendon supported in the way described, it will be found that the lower chest can be expanded to its fullest, and this expansion is effected largely by the diaphragm. If the transversales are then relaxed and the tendon left unsupported from below, the lower chest can be no longer expanded to the same extent as before.

During quiet diaphragmatic breathing the central tendon moves, as I have said, but little, but during a full diaphragmatic breath with flaccid abdominal walls it moves downwards appreciably.

The diaphragm is a very powerful muscle. An ordinary individual, who has full control over this muscle, will find little difficulty in lifting a person of his own weight seated upon his epigastrium, as he lies supine, simply by taking an abdominal breath; and I have been struck by the great strength of this muscle even in old and debilitated patients right up to the moment of death. As we shall see, the descent of the diaphragm influences the circulation by pressing the abdominal veins and thus forcing the blood into the right heart, and in the slackening circulation which is wont to herald death one may fre-

quently observe the diaphragm making vigorous efforts to help the failing heart.

The best way to learn diaphragmatic breathing is to lie on the back, to put both hands on the abdomen, and, while concentrating all the attention on this region, to take a breath, at the same time

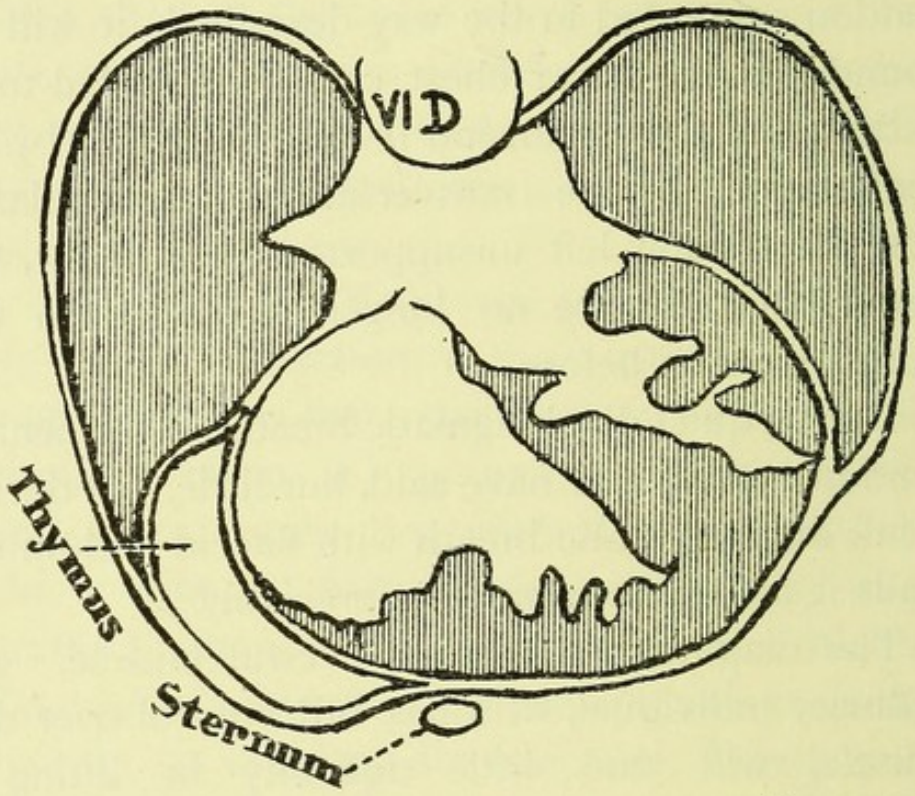

FIG. 1.—Transverse section through thorax of fœtus at the level of the sixth dorsal vertebra. (From frozen specimen.) It will be observed that the solid uninflated lungs lie chiefly at the back of the thorax and only come into limited contact with the heart at its sides.

endeavouring to push up the hands by protruding the abdomen. The same movement should afterwards be practised in the upright position, care being taken to protrude the abdomen during inspiration. When control over the diaphragm has been

obtained in this way the diaphragmatic expirations, which ordinarily take place simply by passive recoil, may be aided by active retractions of the abdomen through the instrumentality of the transversales. In this manner the movements of the diaphragm are made to attain their greatest amplitude.

I come now to a somewhat difficult subject. Like many others, when once understood it is easy enough, but you will, I think, find some difficulty in understanding it from your physiological text-books ; such, at least, has been my case. I refer to the influence of the respiratory movements and the lungs upon the circulation of blood and lymph. This subject is of great importance to the physician, but the lessons which it teaches seem to me to have been largely lost on him. In attempting to elucidate it, and to indicate its applications to medicine, I shall, I feel sure, have your sympathetic indulgence.

The lungs in the fœtus are solid and liver-like. They lie in the posterior aspect of the chest, and only come into limited contact with the heart laterally (Fig. 1). At that most momentous epoch of its career when the child takes its first breath, the chest is expanded in all its diameters—vertical, transverse, and sagittal. The collapsed air-vesicles are pulled asunder, the air being thereby sucked in, and soon all the vesicles are inflated, the lungs having moved the while forwards and downwards

so as to occupy the chief part of the thorax and almost entirely to envelop the heart (Fig. 2). I show you on the screen drawings of transverse sections of the chest taken from the frozen cadaver before and after birth, and you will notice the different position of the lung in the two cases. I would like you especially to observe the intimate relation of the right lung to the right auricle after birth; this relation is admirably brought out in the drawing (Fig. 2), and is full of significance in connection with what I shall have to say later. The left auricle is enveloped by the left lung to a much less extent.

I have said that at birth the air-vesicles are pulled asunder and opened out. To what extent, I would now ask, does this take place?—a pregnant question, the answer to which is illuminating. It might happen, for aught we could predict to the contrary, that they are opened just sufficiently to admit the air without being put actually on the stretch. But such is not the case: the inspiratory muscles go on contracting, not only until the alveoli are opened up, but *until the whole of the elastic tissue of the lungs is put well on the stretch*, just as you might stretch a piece of elastic. I would like you to bear this fact clearly in mind. And *throughout life these inspiratory muscles make it their business to maintain the lungs in this state of stretch.* The elastic lungs thus stretched are always striving to retract towards their points of attachment, *i. e.* their roots, always striving to

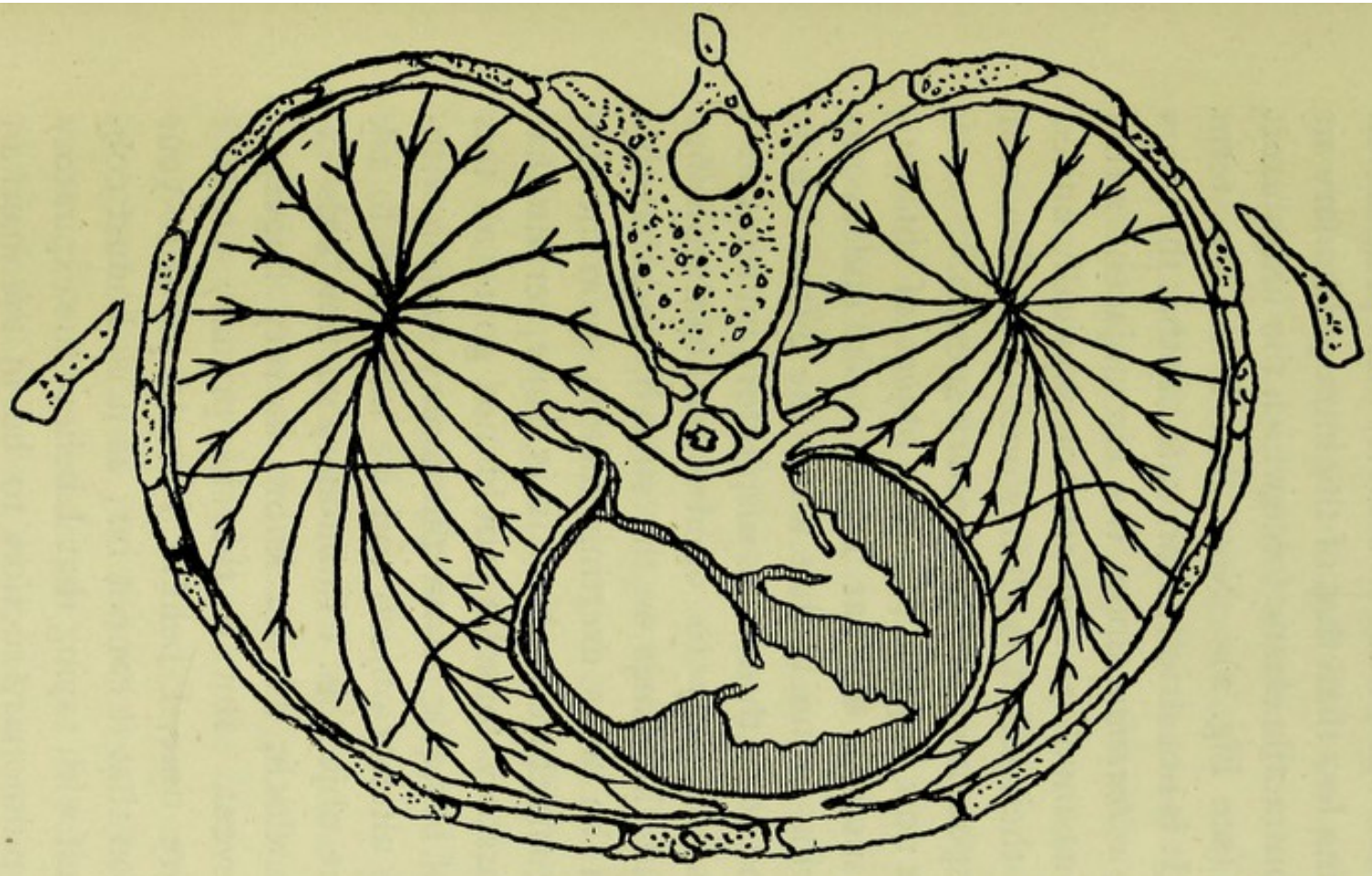

FIG. 2.—Transverse section through the thorax (frozen specimen) after birth, at same level as in Fig. 1; It will be observed that the lungs have moved forwards and now almost entirely envelop the heart. The lungs are intersected with lines marked with arrows in order to indicate how these organs exercise suction on the circumjacent structures. This action can perhaps be the better realised if we imagine each of the lines to consist of a piece of stretched elastic. Note how much more extensively the right lung comes into contact with the right auricle than does the left lung with the left auricle, showing that the former chamber is more under the influence of pulmonary suction than the latter.

escape from contact with the chest wall and other circumjacent structures,—in other words, *the lungs exert suction upon these structures*, their pressure on them being less than that of the intra-pulmonary air by the contractile elastic force which the lungs exert upon it (see Fig. 2). Note that I use the term *suction*. It is not strictly correct to say that the lungs exercise a *traction* upon the circumjacent structures. Inasmuch as they are not physically united to them, their effect is clearly one of suction, and may be spoken of as "pulmonary suction."

Let us now inquire into the object of this pulmonary suction. What I have been endeavouring to bring home is that when the infant begins to breathe it so throws its inspiratory muscles into action as not only to open out the alveoli but also to put the lungs on the stretch, and that this results in the lungs exerting suction upon the surrounding structures. Now it is quite certain that the inspiratory muscles would not go to all this trouble of bringing about pulmonary suction without some ulterior object, and we have next to ask what that object is. Pulmonary suction aids expiration, which, as you know, is very largely a passive event. But is this its primary object? Were there time, I believe I could prove to your satisfaction that it cannot be; as it is, I must content myself with saying that I believe the expiratory effect of pulmonary suction to be in the main an accidental one. What other structures, then, besides the thoracic walls does pulmonary suction in-

fluence? There are the mediastina; above all, the great blood-vessels and the heart to be influenced by it. Has pulmonary suction any special influence upon the flow of blood in the great blood-vessels, *e. g.* the aorta and the large veins within the chest? It has practically no influence upon the aortic flow, nor on that of the great arteries leading out of it; the pressure of blood in them is so great that slight suction leaves them practically unaffected. Does it favour the flow in the great veins of the chest? Certainly it does, its effect being to suck the blood into the venæ cavæ. But I do not think that this can be a primary object, and I will make it clear why I do not. Of the two currents, that in the superior and that in the inferior cava, which is the more in need of help? In the horizontal position of the body the blood flows as readily along the one as the other, but in the upright position the flow in the superior cava is greatly helped by gravity, while that in the inferior cava is as greatly retarded by it. If, then, the direct object of pul. suction be to influence the caval currents, surely it is the inferior and not the superior that should receive the greater influence. Now the inf. cava only runs a bare fraction of its course within the chest, while the sup. cava and its two great tributaries run the whole of their course there. For this reason we may, I think, safely conclude that the main object of pulmonary suction cannot be to act upon the large intra-thoracic veins. What other important thoracic structure have we

left? We have the heart; and I have no doubt that the main object of this pulmonary suction is to facilitate the working of this organ. The lungs almost entirely envelop the heart, and *the inspiratory muscles are busy from birth to death in striving to keep up this suction upon it.* We may express this fact in another way: *Nature has placed the heart, the great central pump, in a partial vacuum.* What is the object of this? I have here a Higginson's syringe; I squeeze it, and water is driven out; I remove the pressure, and the contracted ball, by virtue of its elastic recoil, gradually expands. Now there is no difficulty about the systole, because it is a muscular act; diastole, however, which is a mere passive recoil, is effected with much greater difficulty, and every now and then you will observe the ball to stick for a moment in systole, and show a reluctance to dilate promptly. But if this syringe were worked in a partial vacuum diastole would be effected with far greater promptitude than is the case now, the passive recoil of the ball being aided by the suction from without. And so it is with the heart: *the heart is placed in a partial vacuum for the express purpose of facilitating diastole, above all auricular diastole.* That the ventricles may possess some aspirating power of their own I can well believe, and I would gladly have gone further into this question had there been time; but that the thin-walled auricles must, unless helped from without, often experience considerable difficulty in diastole is self-evident. Especially is

this true of the right auricle in the upright position of the body, when the blood in the inferior cava is necessarily flowing at low pressure. You see now why I was so careful to direct your attention to the fact that the right auricle is practically entirely enveloped by the lungs, and is therefore under normal conditions sucked outwards during diastole, thus sucking the blood out of the cavæ—above all, out of the inferior cava, which in the upright position of the body is in urgent need of some help to its sluggish upward flow. The left auricle stands in far less need of help from pulmonary suction, and it is of interest to note in this connection that it is much less enveloped by lung than its fellow.

Pulmonary suction is not a constant quantity, but varies according to circumstances. It is largely influenced by the intra-pulmonary air-pressure. Thus during a forcible inspiration with closed glottis there is a considerable fall in this pressure, and the suction action of the lungs is considerably augmented ; when, on the other hand, a forcible expiration is made with closed glottis, the intra-pulmonary air-pressure becomes so great as not only to annul all suction but actually to exert a positive pressure on the heart. Concerning the influence on suction of this factor I shall say no more. Another factor determining the degree of pul. suction is the extent to which the lungs are stretched : the more they are stretched, the greater the suction ; hence, setting aside the influence of the air-pressure, pulmonary suction is at its maxi-

mum at the end of a complete inspiration, while at the end of a complete expiration it is entirely annulled, the lungs being then no longer put on the stretch. You see therefore that the position of complete inspiration is the one most favourable to cardiac diastole; then it is that the blood is sucked most forcibly from the cavæ into the right heart, and from the lungs into the left heart. On the other hand, the position of complete expiration is the one least favourable to the diastole of the heart, which in this position experiences no little difficulty in getting an adequate supply of blood from the cavæ and the lungs.

So much for pul. suction and its influence on the heart and circulation. Let us now consider the effect on the pulmonary blood-vessels of the degree of expansion of the lungs. I show you two diagrams bearing upon this matter (Fig. 3). The one represents an infundibulum with its alveoli in a state of extreme inspiration; the other represents the same in extreme expiration. You will observe that in the former condition the lungs contain the maximum amount both of air and blood; in the latter, the minimum amount of each. In other words, when the lungs are expanded to their utmost in inspiration, not only do they contain the maximum quantity of air, but the maximum amount of blood also,—that is to say, in this condition the blood-vessels are opened out to their utmost, the pulmonary circuit offering the minimum resistance to the right heart, and permitting the largest

quantity of blood to pass through to the left heart.

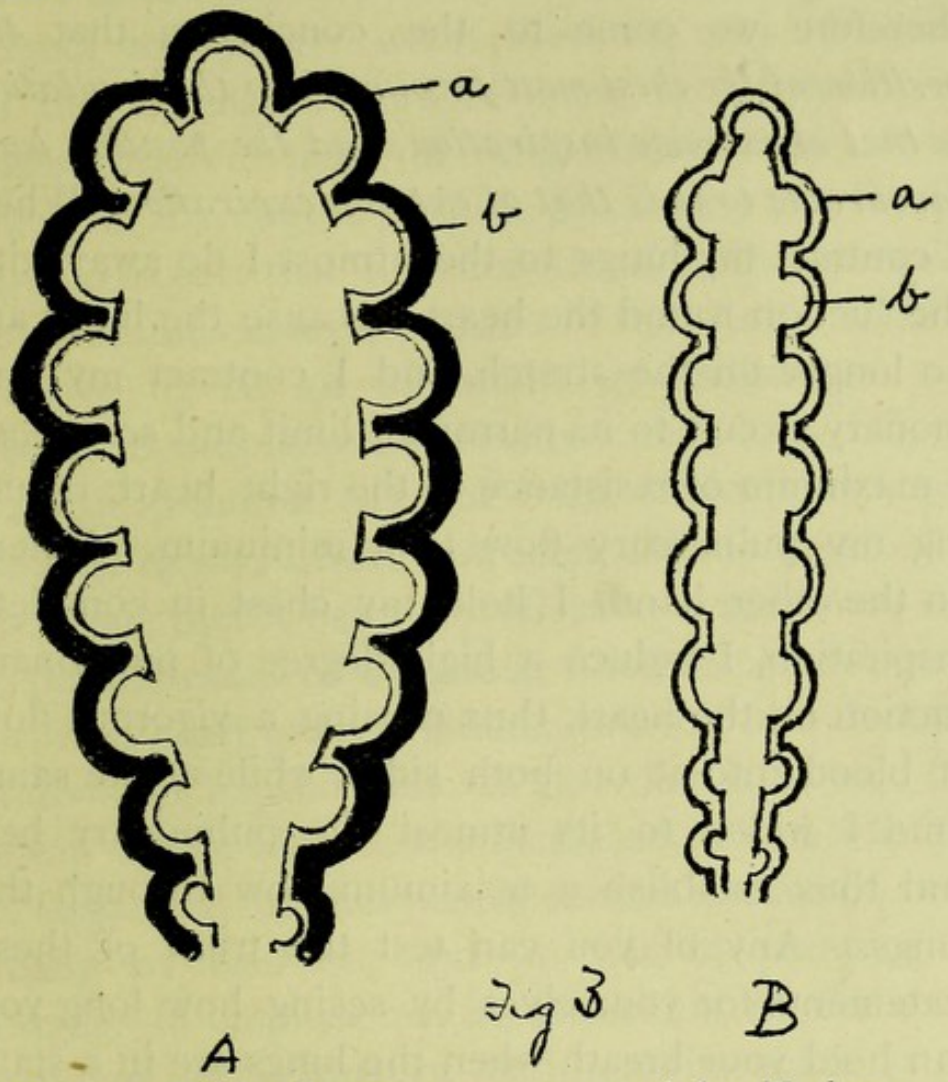

FIG. 3.—These diagrams represent an infundibulum, A, at the end of a complete inspiration, and, B, at the end of a complete expiration. *b* = alveolar wall; *a* = blood-vessels of the same. It will be seen that the amount of blood in the lungs varies directly with the amount of air, and that the pulmonary blood-vessels are most dilated and the resistance to the right heart least at the end of a full inspiration; while, contrariwise, the vessels are most contracted and the resistance to the right heart greatest at the end of a complete expiration.

But we have just seen that when the chest is expanded to its fullest, pulmonary suction is at its

maximum and the heart most favourably circumstanced for sucking blood into its chambers; and therefore we come to the conclusion that *the position of the chest most favourable to the circulation is that of extreme inspiration, and the position least favourable to it is that of extreme expiration.* When I contract my lungs to the utmost I do away with the suction round the heart, because the lungs are no longer on the stretch, and I contract my pulmonary circuit to its narrowest limit and so oppose a maximum of resistance to the right heart, reducing my pulmonary flow to a minimum. When, on the other hand, I hold my chest in complete inspiration, I induce a high degree of pulmonary suction on the heart, thus causing a vigorous flow of blood into it on both sides, while at the same time I widen to its utmost the pulmonary bed and thus establish a maximum flow through the lungs. Any of you can test the truth of these statements for yourselves by seeing how long you can hold your breath when the lungs are in a state of complete expiration and complete inspiration respectively. I find I can hold my breath two and a quarter times as long with my chest in extreme inspiration as I can when it is held in the position of complete expiration. Or again, you may make the following experiment:—Take a moderately deep expiration, so as to diminish the mean size of the chest, and then take breaths of ordinary amplitude round that mean. You will find that you soon become dyspnœic.

The position of inspiration being that which is most favourable to the circulation, what should we expect to happen when there is any difficulty in the circulation—when there is dyspnœa, for instance, from heart or lung disease, and urgent need for the largest possible pulmonary flow? We should expect that to occur which we find actually does occur, viz. an expansion of the chest. This I believe to be an important generalisation. It seems so simple and self-evident that it is difficult to believe that it has not been made before, yet, strange to say, I have not been able to obtain any evidence that it has. In all save certain very exceptional cases of dyspnœa, whether physiological or from heart or lung failure, you will find that the individual expands his chest,—that is to say, the mean size of his chest is increased. You will find it expanded in the breathlessness produced normally by exertion, and in that of pathological origin—in emphysema, in bronchitis, in the acute capillary bronchitis of children, in acute pneumonia, whether croupous or catarrhal, in pleurisy with effusion, in pneumothorax, in spasmodic asthma, and in cardiac failure. For some years past I have carefully examined the chest in these various conditions, and I have never failed to find it expanded.

How is the expansion brought about? Obviously by the over-action of the inspiratory muscles. These muscles contract vigorously, just as in a deep inspiration, but they do not completely relax

during expiration; hence there is an increase in the mean size of the chest; were they completely to relax during expiration the chest would return to the position of ordinary expiration. In these cases, with certain exceptions to be mentioned in due course, the expiratory muscles come little if at all into action.

Hitherto we have considered how the *degree of expansion of the lungs* influences the circulation. I have said nothing as to how far the *movements of the chest* influence it. This is another question altogether. The movements of the lungs have, in my opinion, under ordinary circumstances, no influence whatever in accelerating the blood-flow, inasmuch as the increase in pulmonary suction and the width of the pulmonary vascular bed which take place during the inspiratory expansion of the lungs are balanced by a corresponding diminution in each during the expiratory contraction; or, to put it in another way, the mean pulmonary suction and the mean width of the pulmonary bed are exactly the same during inspiration as during expiration. As I read it, then, the mere opening up and contraction of the alveoli have no influence in accelerating the circulation of blood. Were there valves in the pulmonary blood-vessels the case would be otherwise. The lymphatics of the lungs *are* provided with valves, and hence the pulmonary movements quicken the circulation of lymph through the lungs.

While, however, the *ordinary* movements of the

lungs do not quicken the blood circulation, it is possible so to modify them that they shall have an accelerating influence. If, for instance, I take a sudden rapid breath, the sudden inflation of the lung produces an equally sudden negative intra-pulmonary pressure, and this by increasing pulmonary suction will aid the circulation; and if, following upon such an inspiration, I hold my chest for some little time in the inspiratory position, *i. e.* the position most favourable to the circulation, and then allow a moderately rapid expiration to take place, I believe the inspiratory acceleratory effect will more than outweigh the expiratory retarding effect. Now this is the form of breathing largely resorted to in physiological breathlessness. If you sprint a hundred yards you will find not only that the mean size of your chest is increased, but that the inspiratory period is more than double as long as the expiratory period.

In the time remaining I will apply these remarks, first of all, to physiological breathlessness, and, secondly, to emphysema.

First of all, let us consider physiological breathlessness. In all forms of physiological breathlessness the inspiratory muscles are thrown into vigorous contraction, while expiratory muscular action is conspicuous by its absence; and this leads me to say that the best way to develop the lungs is by exercises which induce breathlessness. If you want, *e. g.*, to develop a little girl's lungs, get her to dance and skip; this will make her breathless, the

inspiratory muscles will be thrown into vigorous play, and this action will develop the lungs better than any gymnastic exercises with which I am acquainted. No better means can be devised for developing the lungs of children than skipping, dancing, and the running and jumping which find a place in so many outdoor games. Such games are, moreover, far better calculated to develop the physique in a normal manner than the methods by dumb-bells and gymnastics which are now all the rage and the effect of which is to make the individual muscle-bound. As I pointed out in my last lecture, "strong" men have not necessarily good lungs; they increase their chest-girth mainly by developing, not the lungs, but the muscles, and, moreover, this development of the thoracic muscles takes place at the expense of thoracic mobility.

Next as to emphysema. This disease is very common: all of us, as we get older, tend to suffer from it more or less; it gives rise to much trouble, and, as might be expected, much has been written about it—and yet, in spite of all this, I do not find that some of the simplest facts concerning it have been properly interpreted. I have not, for instance, met with any satisfactory explanation of the enlargement which the chest undergoes in emphysema, nor of its fixation in the inspiratory position; but if the main conclusions I have put before you are correct, I shall be able to offer you a very simple interpretation of both these features. The

chief facts of pathological anatomy to remember in regard to emphysema are—(1) the loss of pulmonary elasticity, and (2) the atrophy of the smaller blood-vessels. (1) As we get older all our tissues tend to lose their elasticity; we see this truth illustrated somewhat painfully, we may think, in the case of the skin: during youth it is closely applied to the underlying tissues, but with advancing years it loses its elasticity and, becoming stretched, is thrown into wrinkles. And just as our skin loses its elasticity, so do our lungs lose their's also. Now if pulmonary suction plays a useful part in the economy—if, as I have endeavoured to show, the inspiratory muscles are, so to say, at great pains throughout life to keep the elastic fibres of the lung on the stretch so as to maintain a certain degree of this suction action,—what should we expect to happen when the lungs begin to lose their elasticity? What but that the inspiratory muscles should come into extra play so as to tighten up the relaxed pulmonary tissue? The pulmonary fibres have, with advancing years, to be strung up continually, even as a stringed instrument has to be from time to time, for otherwise pulmonary suction must steadily grow less and less. Were my lungs at this moment suddenly to grow twenty years older, I am quite sure that my chest would forthwith expand to a notable degree through extra action of the inspiratory muscles, ever on the watch (through their nerve-centres) to maintain pulmonary suction at its normal level.

Hence the steady increase in thoracic girth which tends to take place after middle life, or even before it, and the shortening of the neck, as every tailor and dressmaker can testify. Many a man proud of his forty-inch chest would be much better off with a thirty-six circumference. The moderately flat chest with an index of 70, capable of a wide range of movement, is the ideal one. Deepness of chest is a sign of degeneration, and goes along with defective motility.

Now in emphysema this loss of pulmonary elasticity which occurs in all sooner or later, if they live long enough, takes place in an exaggerated degree, and hence in it the chest tends to be so much the more expanded.

(2) The second anatomical characteristic of emphysema is the atrophy of the small pulmonary blood-vessels. What does that imply? It implies an increased resistance to the flow of blood through the lungs, and a diminished respiratory area. Now we have already agreed that when the lungs expand in inspiration the pulmonary blood-vessels expand at the same time: there is an increase in the amount of blood in the lungs, and a diminution in the resistance of the pulmonary circuit, as well as an increase in the respiratory area. Consequently, as the blood-vessels in the alveoli atrophy, thereby diminishing the respiratory area and imposing increased work on the right heart, the inspiratory muscles come to the rescue and tend to neutralise these evils by expanding the chest. There is,

therefore, a twofold reason why the chest should be expanded in emphysema—first, because of the loss of pulmonary elasticity; and secondly, because of the atrophy of the pulmonary blood-vessels. This explanation, I trust you will agree with me, is a common-sense one, and I may here say that I have always found common sense to be my best friend in my profession. I have no doubt it is the right explanation. Those given in medical writings —even the most recent works on diseases of the lungs—do not meet the facts of the case.

It is argued that, inasmuch as the elastic recoil of the lungs helps expiration, as the elasticity of the lungs diminishes, the inspiratory muscles, not being properly antagonised, gain the upper hand, and that this leads to an expansion of the thorax—a view first enunciated by Stokes, and accepted by nearly all writers on the subject since his time. This implies that the expansion is a necessary evil and not, as I contend, a cunning device adopted by Nature to counteract certain evils. If it were to the advantage of the emphysematous patient to have his chest diminished instead of increased in size—if such a diminution would facilitate the circulation and the aëration of the blood,—may we not be quite sure that the organism would find means to bring this about? All that would be necessary would be for the inspiratory muscles to contract less vigorously and the expiratory muscles more vigorously than they usually do. *A priori* argument is therefore all against

Stokes's view; *à posteriori* argument conclusively proves it to be wrong : I have carefully examined the action of the respiratory muscles in emphysematous patients for some years past, and I have found that while, in the early phases of the disease at least, there is not the slightest tendency on the part of the expiratory muscles to extra action, the inspiratory muscles contract with unwonted vigour. The individual does not expire to the normal position, for the simple reason that *he does not want to*, his object being to increase the mean size of the chest—with what ends in view I have already indicated. During the more advanced phases of the disease, when the thorax gets fixed and the individual depends mainly upon his diaphragm for breathing, certain expiratory muscles come into extra play, but these are not so much those which depress the thorax as those which push up the diaphragm, for owing to the extreme diminution of pulmonary suction in advanced emphysema the diaphragm is not properly *sucked* up during expiration but has to be *thrust* up by an active retraction of the belly by means of the transversales ; and this extra action of the transversales tends to lead to a hypertrophy and shortening of their fibres, and to a narrowing of the costal arch. I have watched this mode of breathing many times.

A few words as to the therapeutic application of these conclusions. First of all, it is manifest that there is no use whatever in applying (as some have

recommended) an elastic bandage to the emphysematous chest with a view to make up for defective pulmonary elasticity. The only effect of such a procedure will be to give the unfortunate inspiratory muscles more work to do than they have already, for the emphysematous patient will at any cost keep his chest expanded; but I may here observe that, were Stokes's view correct, it ought to be possible, but I am sure it is not, to prevent the emphysematous chest from enlarging by means of such a vest. If you could envelop each lung in an elastic covering, that would be a somewhat different matter, because then you would increase pulmonary suction, although there would still be need for increased expansion of the lungs in order to open out the pulmonary vessels.

I shall refer to other practical applications in my next lecture, and propose to occupy the time remaining at our disposal by showing you some photographs on the screen of patients suffering from various diseases causing dyspnœa.

LECTURE III.

GENTLEMEN,—In the last lecture I pointed out that the lungs of the fœtus lie at the back of the chest, that at birth they are expanded so as almost entirely to envelop the heart, and that throughout life they are kept stretched by the inspiratory muscles, with the result that they exert suction on, and aid the diastole of, the heart. I have here a simple apparatus to illustrate this constant suction action of the lungs upon the heart, and the part played in this result by the inspiratory muscles. [Instrument shown.] Even in the mean position of the chest the lungs are moderately stretched and in consequence exercise suction upon the heart, thus aiding its diastole; during inspiration they are stretched still further, pulmonary suction increasing, and the cardiac diastole being aided to a corresponding extent. I also told you that when the lungs are fully expanded their blood-vessels are most capacious and offer the least resistance to the right heart. And I argued from these facts that the position of the chest most favourable to the circulation is that of full inspiration, because then the influence of pulmonary suction

on diastole is most pronounced, the pulmonary bed widest, and the flow of blood through the lungs most abundant and most rapid. I then went on to point out that when it is desirable to facilitate the circulation, especially that through the lungs, the chest is put in the position of inspiration; that this is the case whether one has to do with simple physiological breathlessness, such as that produced by running and jumping, or with pathological breathlessness, such as that which accompanies heart disease, spasmodic asthma, emphysema, pneumonia, phthisis, pneumothorax, pleurisy, and the like, in all of which cases the chest is found in the position of inspiration. I further pointed out that this position of inspiration is brought about by the predominant action of the inspiratory muscles: the only way the chest can be held in an expanded position is by a tonic contraction of these muscles; without this it would, by virtue of its elastic recoil, resume (or nearly so) the position of ordinary expiration directly the inspiratory muscles ceased to act; hence in all these cases the inspiratory muscles come into vigorous action, only partially relaxing during the period of expiration. Lastly, I drew your attention to the fact that the rhythmical expansion and contraction of the lungs, unless modified in a peculiar way, have no influence on the circulation of the blood, although they tend to accelerate the flow of lymph through the lungs. I do not know whether physiologists teach this; indeed, I think they do

not. So far as I follow them they maintain that the ordinary pulmonary movements do influence the circulation of the blood; according to my reading of the facts these movements have no such influence.

One important fact, however, I omitted to point out in the last lecture—namely, that the rhythmic movements of the diaphragm in respiration materially aid the circulation of the blood. Every time the diaphragm descends it compresses the intra-abdominal veins and tends to squeeze the blood out of those veins into the right auricle at the very time when, owing to the descent of the diaphragm, the suction round that chamber is augmented. Hence the diaphragm plays the part both of a force-pump and a suction-pump. It should be noticed that the blood in the intra-abdominal veins cannot pass into the abdominal walls, or into the lower extremities, because the veins in these parts are provided with valves. You will readily understand that when the movements of the diaphragm are vigorous they must exercise a very considerable influence on the circulation. In extreme failure of the circulation, such as often occurs in the last hours of life, one may frequently observe the diaphragm acting very forcibly, and one can hardly doubt that the main object of this is to hurry on the sluggish blood-stream. I have learned to regard this convulsive action of the diaphragm as a bad sign.

I said just now that "the expanded position of

the chest is that which is most favourable to the circulation." This will serve as a text for my remarks this evening. I propose to apply this proportion to a number of chest diseases.

Emphysema.—First as regards emphysema. We agreed last time that in this disease there is a loss of pulmonary elasticity and an atrophy of the small blood-vessels of the lung, and that as a result of these changes the inspiratory muscles come into augmented action—first, to secure the necessary degree of tension in the elastic fibres of the lungs, which would otherwise, having lost their elasticity, be relaxed and sinuous ; and secondly, in order to open out the pulmonary vessels. Hence we find that the chest in emphysema gets year by year larger and larger, as the lungs get less and less elastic and their blood-vessels more and more atrophied, until in course of time we find it permanently fixed in a position of inspiration. Were I asked to describe the advanced emphysematous chest, I should describe it as one fixed in a position of supra-extraordinary inspiration—*i. e.*, it is larger than the normal chest at the end of a complete inspiration. Now we have to ask how it comes about that the emphysematous chest gets *fixed* in this position. This is surely a pertinent question; yet curiously enough it does not appear to have interested physicians much. Of recent books on disease of the lungs which I have consulted, many do not raise it, and but few attempt to explain it. One eminent physician suggests that it is due to

some impediment to the egress of air from the alveoli. This view I cannot accept, and for this very simple reason: the emphysematous patient breathes, as we have seen, chiefly with his diaphragm; he takes a deep diaphragmatic breath and then drives up the diaphragm by retracting the belly, in this way forcing the air out of his chest; and seeing that there is no difficulty whatever in getting the air out in this way, it is clear that there can be no great impediment to the egress of air from the alveoli. The reason why he cannot get the normal amount of air out of his lungs is simply because the chondro-osseous walls of the thorax are fixed. But why are they fixed? There are, I believe, two reasons for this fixation: we have seen that the inspiratory muscles in emphysema are thrown into preponderating contraction, and whenever a group of powerfully acting muscles is not properly antagonised, they shorten, as is well illustrated in the case of talipes and in many other conditions; consequently, there occurs in emphysema a shortening of the inspiratory muscles, and in this way the thorax gets fixed in the expanded position. But a second factor now comes into play; the chest tends to "set," so to speak, in the expanded position—the ribs tend permanently to assume the curvatures of inspiration, the joints to alter in shape, the ligaments to shorten, and so forth.

We may next inquire whether any advantages accrue from this fixation of the chest in emphy-

sema. I have little doubt that it serves a good purpose. We have seen that the maintenance of the inspiratory position is of advantage to the emphysematous patient; now the fixation of the chest in this position necessarily tends to save the inspiratory muscles; without it these muscles would require to maintain a powerful contraction during the period of expiration in order to keep the chest expanded; with it they are, to that extent, relieved of work.

But does the fixation entail also disadvantages? I think that it does. First, it tends to interfere with easy expectoration; patients often experience considerable difficulty in bringing up sputum; this may be because it is very viscid, but it may also result from feebleness of the expiratory blast; in order that the sputum may be brought up easily it is not only required that it shall not be too viscid, but also that there shall be a vigorous expiratory blast; and if the chest is fixed, this latter desideratum is not secured. Another disadvantage attaching to fixation of the chest is the inability to expire deeply; there are occasions when it is of great advantage to be able to take a good deep breath; but this an individual with a fixed chest cannot do, and thus he is handicapped considerably, especially if he develops some acute pulmonary disease, *e. g.* bronchitis or pneumonia, owing to the great limitation of his respiratory range.

For these reasons we should not allow the

inspiratory muscles to have it all their own way in emphysema ; we should get the patient to practise deep expirations from an early period of the disease ; we may order him to take expirations from the mean position of respiration so as to empty his lungs as much as he can ; this he should do for ten minutes twice or thrice daily. Secondly, we should advise him to avoid doing anything which is apt to cause breathlessness, which, as we have seen, excites the inspiratory muscles.

Heart Disease.—Let us next consider our principle in relation to heart disease. In most cases of organic cardiac disease the heart is enlarged, sometimes enormously. Especially is this the case with children ; I speak haphazard, but I feel sure I cannot be far wide of the mark when I say that it is not rare for a child's heart to occupy as much space as a single lung normally does. Now it is obvious that whenever the heart enlarges there must, unless the chest expands proportionately, be a relaxation of lung tissues. In order, therefore, to keep the lungs at their normal degree of expansion, it is necessary, when the heart enlarges, that the inspiratory muscles shall come into extra play, so as to expand the chest. But there is a reason why the chest should expand even beyond this : directly there is any failure of the central pump the circulation, especially that through the lungs, wants all the help it can get ; clearly, then, it is not enough that the lungs should be expanded to the normal amount ; they require to be expanded

beyond this, so that the full circulatory advantages attaching to their ample expansion may be secured. Hence it is not surprising to find that in all cases of serious heart disease the inspiratory muscles are thrown into violent contraction. In advanced cases what we actually find is this: the patient sits up in bed; he has, as we say, orthopnœa; he fixes his arms and raises his shoulders, so as to get all the purchase he can for the inspiratory muscles attached to the shoulder girdle, with the object of expanding his chest to the utmost. I have no doubt that one of the purposes served by orthopnœa in heart disease—and I should not be surprised if it were the chief purpose—is to give the inspiratory muscles the fullest possible scope, for when the patient is lying down these muscles are placed at a very great disadvantage.

I say the inspiratory muscles in cardiac dyspnœa are thrown into powerful contraction. What about the muscles of expiration? I have carefully examined a large number of cases of heart disease as regards the condition of the muscles of respiration, and I find in most cases the expiratory muscles do not act much, if at all. In children they scarcely act at all, and you can see why; in them the thoracic cage is very pliant, and if the expiratory muscles were to contract vigorously the heart would be subjected to a positive pressure; therefore the child dare not expire vigorously; all his efforts—and it is painful to witness them in bad cases—are directed towards maintaining the tho-

racic cavity at its maximum expansion, so as to keep the lungs well expanded and to protect the heart from the positive pressure to which it must otherwise be subjected. Imagine what would happen if, in an extreme case, the expiratory muscles were to contract powerfully and thus compress the faltering heart and crumple up the overburdened lungs!

When, on the other hand, the chest is rigid, we may sometimes see in cases of cardiac dyspnœa the abdominal muscles contracting with some vigour, but this, I think, is mainly for the purpose of driving up the diaphragm and thus aiding the flow in the inferior cava.

Seeing that well-developed, well-expanded lungs facilitate the pulmonary circulation, it is obvious that we should, in heart disease, pay due attention to the health and development of these organs. I have, indeed, sometimes thought that in it it is more important to consider the lungs than the heart itself—in mitral disease at all events. I cannot now enter into the lengthy question of exercise for heart disease, but this much I may say: that exercises which induce a certain amount of breathlessness may benefit by favouring the pulmonary circulation, though I do not say that this is the only way in which they may do good.

Pericardial Effusion.—Next let us consider pericardial effusion in relation to our text. As the fluid accumulates in the pericardium the lungs are encroached upon, and in order to get them properly

expanded the inspiratory muscles must come into extra action. So long as the effusion is moderate in quantity the lungs may in this way be expanded to the normal, and suction normally maintained, so that no rise in the pericardial pressure occurs; as pericardial distension increases, however, it becomes increasingly difficult to maintain the normal pulmonary suction—although even with a large effusion it may still be possible to maintain some negative pressure in the pericardium,—and sooner or later a time is arrived at when it is no longer possible to get pulmonary suction, the fluid in the pericardium attaining a positive pressure which increases with every increment of the effusion. The heart is now actually compressed—a very serious condition of things. We have seen how great an effort is made throughout life to secure a negative pressure round it; but when the pericardium is distended with fluid under positive pressure, the heart, far from being sucked upon from without, is actually compressed by its enveloping fluid, a serious obstacle being thus opposed to the entrance of blood into the auricles.

The pressure effects of pericardial effusion are usually grouped under four heads:

1. From pressure upon the œsophagus, dysphagia may result. I have never seen an instance of this.

2. Pressure upon the lungs may cause pulmonary embarrassment.

3. Pressure upon the sternum and ribs may, it is said, thrust them outwards.

4. Last, and most important of all, pressure upon the heart impedes its proper action.

With regard to the bulging of the precordium in pericardial effusion, I very much doubt whether this can be due to pressure from within. If the pressure of fluid in the pericardium is sufficiently great to thrust outwards the precordium, it would surely so effectually squeeze the auricles as to prevent the blood from flowing into them, and it seems incredible that the heart could go on beating under such conditions. Therefore, whatever precordial bulging may occur in pericardial effusion I should be inclined to attribute rather to the action of the inspiratory muscles in pulling the precordium outwards away from the heart, so as to minimise as much as possible the pressure upon that organ, than to a thrusting outwards of the chest-wall from within by the distended pericardium.

What are the circulatory symptoms resulting from pericardial effusion? Cohnheim injected oil into the pericardium of animals, and the results he obtained were just what might have been anticipated: the blood-pressure in the veins continued to increase because the blood was prevented from flowing properly into the right heart, while the pressure in the great arteries fell because the blood could not reach them in sufficient quantity. And this is what happens clinically. We find great dyspnœa and distress, distension of the veins of the head and neck, and a feeble pulse, the patient dying from arterial anæmia and passive venous

engorgement. You may ask how it is possible for the circulation to be carried on at all when the heart is subjected to a positive pressure from without. The only possible way by which the blood can get into the auricles under such conditions is by being driven along the cavæ under a decidedly positive pressure; and the only way to get up a sufficiently high venous pressure for this purpose, as far as I can see, is by means of muscle contractions: the diaphragm and anterior abdominal muscles come into play and exert considerable pressure upon the intra-abdominal venous blood, squeezing it along the inferior vena cava into the right auricle; possibly the cervical muscles have a similar influence on the superior veins.

Phthisis.—Now let us consider the subject of phthisis from the point of view of the principle which we have laid down. By phthisis I mean of course pulmonary tuberculosis; in it the lung structure is corroded and the respiratory area diminished. To make up for this the inspiratory muscles come into extra play so as to expand the unaffected portions of the lungs, and so effectually may they do this that there may be no diminution in thoracic girth, even though there be wide-spread destruction of lung tissue. That is a fact which I do not think is sufficiently recognised. I venture to say that in a very large number of cases of phthisis, especially in the earlier stages, you will find no evidence whatever of contraction of the chest. You may, in fact, get one lung completely

destroyed, without any contraction of the affected side; in such a case there will probably be hypertrophy of the opposite lung, and the mediastinum will be drawn over and the diaphragm drawn up. But a patient with considerable destruction of one lung may show no thoracic contraction, even though no such displacements occur, owing to extreme stretching of the pulmonary tissue and bronchi by the vigorous action of the inspiratory muscles. In this way great dilatation of phthisical cavities as well as of the bronchial tubes and air-vesicles may be brought about, producing, it may be, a condition of bronchiectasis and emphysema.

Contraction of the chest does, however, occur in phthisis, but when this happens it is not, be it noted, by a *falling in* of the thoracic walls. Such a term suggests that the thorax is normally supported from within, and that in phthisis this support is withdrawn; whereas, as we know, its walls are subjected normally to the suction action of the lungs. The so-called *falling in* of the chest is, of course, a *dragging in* due to the contraction of cicatricial tissue in the pleura and lung. And this leads me to say that the tension of the pulmonary tissue, and therefore pulmonary suction, is (so I believe) augmented in phthisis, partly owing to contraction of the cicatricial tissue in the lungs, and partly to the excessive stretching of the atrophied lungs by the exaggerated action of the inspiratory muscles. Hence the over-action of the inspiratory muscles in phthisis has not (as in the case of most

other chest affections in which such excessive action occurs) as one of its objects a maintenance of normal pulmonary suction for the sake of facilitating cardiac diastole: its sole object is to open out the pulmonary blood-vessels and increase to the utmost the respiratory area.

The fact that the inspiratory muscles are ever on the alert to maintain an adequate expansion of pulmonary tissue in this disease is one of great practical importance, because it shows us that there is no need to resort to special exercises in it to develop the lungs. The patient himself instinctively secures all the expansion that is desirable, the slightest dyspnœa throwing the in spiratory muscles into exaggerated action.

At a Congress on Tuberculosis, held in London in 1901, Professor Clifford Allbutt asked whether it might be advisable, in a certain stage of phthisis, to put the patient under respiratory exercises. I ventured to address a letter to the 'Lancet' in response to that question, and with your permission I will read a part of it, because it exactly expresses my views on the subject. "Among the many weighty questions raised by Professor Allbutt in his eloquent address on the above subject, not the least important is that relating to the use of gymnastics in phthisis. After observing that the subject is 'little understood,' and that in the more active stages of pulmonary disease 'gymnastics must be inappropriate,' Professor Allbutt continues: 'In healing stages, when softening has ceased and the

lung is drying and laying down protective fibre, may not gymnastics, under supervision as skilled as for cardiac disease, do much to expand and thus to call into healthy function the parts which the tubercle has spared? I seek the answer from those who are dealing daily with these problems.'

"This question is, I think, capable of a decisive answer. In no form of lung disease, not even excepting extensive collapse left by a vanished pleural effusion, are special exercises for the purpose of bringing about pulmonary expansion either needful or desirable. The fact is that in all cases of lung disease sufficiently pronounced to cause dyspnœa, the organism of its own accord puts into operation, by virtue of the dyspnœa, a process by which the lungs are expanded just so much as is desirable. Now in phthisis, dyspnœa, even though it should be absent during complete rest, is readily provoked by even moderate exercise, and, therefore, no special exercises are required to promote pulmonary expansion in this disease."

Before leaving the subject I would mention a case bearing upon the same point. Not long ago a medical man wrote in one of the medical journals describing a remarkable result which he had achieved in a case of phthisis by a new method of treatment; he claimed to have increased the girth of the chest in the course of six weeks by three inches. Well, gentlemen, I am quite sure that statement was made in good faith; but I am equally sure it was the result of inaccurate ob-

servation ; no phthisical chest could be made to expand three inches in six weeks ; moreover, even if such expansion could be effected, it could only be by a violent rending of pulmonary tissue at the expense of considerable damage to the lungs. As the result of his dyspnœa the poor phthisical patient's inspiratory muscles are, I repeat, continually thrown into vigorous contraction and expand the lungs just as much as is good for them, and an expansion of three inches beyond this could only take place to the serious detriment of those organs.

Spasmodic Asthma.—Now let us pass on to consider spasmodic asthma and to see if we can apply our principle to it also. During an asthmatic paroxysm the chest is in the position of full inspiration. The shoulders are high ; the chest is deep ; the diaphragm lies low ; further, the breathings are altered in their time relations : instead of inspiration and expiration being as 6 : 7, they may be as 1 : 4. Another feature of the attack is the small amount of air passing into and out of the lungs with each breath.

How are we to account for these phenomena? First, let us briefly look at the pathology of spasmodic asthma. There is no general agreement amongst physicians in respect of it, but this much we may say, that it is either due to a narrowing of the tubes or to an inspiratory spasm. Now in regard to the latter view it is certainly the fact that the chest during the attack is in a position of in-

spiratory spasm ; but such is the case in practically all cases of dyspnœa, as I have again and again insisted, and I think there can be little doubt that this inspiratory spasm in spasmodic asthma is secondary—secondary, in fact, to dyspnœa produced by the narrowing of the tubes. Whether this narrowing is itself due to spasm of the tubes or to swelling of their lining membrane, I shall not stop to discuss—probably each of these factors plays its part,—but this we may, I think, safely predicate : that, given a wide-spread narrowing of the tubes producing dyspnœa, an expansion of the chest necessarily occurs in obedience to the principles I have enunciated, because when only a small quantity of air is permitted to pass into and out of the chest, the distress is far less urgent with an expanded than with a contracted chest, extreme expansion giving the maximum respiratory surface and the maximum pulmonary blood-flow. I made a very simple experiment upon myself yesterday and obtained precisely the result I anticipated. I closed my mouth and partially closed my nostrils so that I was only able to breathe a meagre current of air, and was therefore so far in the position of a patient during an attack of spasmodic asthma. Now what happened? I found my chest gradually expanding and eventually assuming the position of complete inspiration—that position, namely, most favourable to the circulation through the lungs and the aëration of the blood. If a man can only breathe, say, one tenth of the normal quantity of

air, he will naturally put his chest in that position which is most favourable to pulmonary circulation and blood aëration, and it is thus I explain the expansion of the chest in the asthmatic paroxysm.

Possibly some other factor must be invoked to explain the prolongation of the expiratory phase, a feature suggesting that there is a greater impediment to the egress than to the ingress of air.

Acute Pneumonia.—Next let us apply our principles to acute pneumonia. In this disease, in croupous pneumonia, for instance, a large number of pulmonary air-vesicles are tightly packed and distended with inflammatory products. As a result a large portion of the lung tissue is placed *hors de combat*, rendered useless as a respiratory medium. But more than this, a considerable part of the pulmonary circuit is blocked by the pressure of inflammatory products upon the small blood-vessels. To make up for this curtailment of the respiratory surface and this narrowing of the pulmonary vascular bed, the inspiratory muscles are incited to vigorous action, so as to cause a general expansion of the sound parts of the lung. In this way there occurs an expansion of both sides of the chest. There is, however, a further reason why the chest should expand in pneumonia—namely, for the protection of the inflamed lung; were the chest wall not pulled out by the inspiratory muscles it would press upon the swollen and sensitive lung, and this would hardly be advantageous to the patient. Hence the in-

spiratory muscles make it their business to pull the thoracic wall away from the inflamed lung, so that it shall exercise no pressure upon it at all. They are not always successful in this, for you may sometimes—though I believe this is rare—find post mortem the surface of the consolidated lung marked with ribs.

Pleural Effusion.—We now come to the subject of pleural effusion. How far does our principle apply here? In this condition fluid is poured out into the pleura and encroaches upon the space which should be occupied by the lung, which thus tends to relax. To counteract this the inspiratory muscles come into extra play, and to such good effect as frequently to maintain pulmonary suction at the normal, even when the effusion is copious, so that you may sometimes get a negative pressure in a pleura containing upwards of 100 ounces. Sometimes, however, quite a small pleural effusion proves, when tested, to be under a positive pressure; and such is always the case with empyema, owing, no doubt, to the agglutination of the lung to the chest wall, so that the fluid being hemmed in, its tension necessarily increases as the effusion goes on increasing.

I think you will agree with me that the fact that a pleural effusion of upwards of 100 ounces may show a negative pressure is a most interesting one; such negative pressure can only be produced by a very powerful contraction of the inspiratory muscles; and in all cases of copious effusions you

will find evidence of this : you will find, as might be expected, the chest expanded on both sides—not only on the affected side but on the other also, though rather more, if anything, on the former. It will thus be seen that the expansion on the affected side is not due, as a rule, to the chest walls being pushed out, but to the action of the inspiratory muscles on that side and to their endeavour to secure the normal degree of expansion of the compressed lung.

Catarrhal Pneumonia.—Before passing to pneumothorax let me say a few words on the expansion of the chest in catarrhal pneumonia. If you look at a child in the acute stage of catarrhal pneumonia, or capillary bronchitis—they are practically the same disease,—you will notice that the chest, notably the upper part, is expanded. We have here, again, the same story ; the expansion is brought about by the powerful contraction of the inspiratory muscles, and is compensatory. The upper parts of the lungs, being least involved in the disease, undergo the greatest expansion ; the lower portions, indeed, may show areas of collapse (from plugging of the smaller tubes). Hence we frequently find the upper part of the thorax much more prominent than the lower ; so greatly, in fact, may the unaffected part of the lung be expanded that the vesicles may be stretched beyond the power of immediate recoil, as shown by the fact that after death the excised lungs maintain this hyperinflated condition. The condition is

manifested clinically by the great distension of the chest and by its hyper-resonance on percussion. It is sometimes spoken of as acute emphysema, but it is not a true emphysema, *i. e.* a running together of the alveoli, but merely a hyperdistension of them. Its pathology has been variously explained. To me the explanation of it seems simple, and to be sought in the direction just indicated : it is brought about, I suggest, by the forcible action of the inspiratory muscles ; the child instinctively utilises to their full extent all the available alveoli by expanding the lungs to their utmost, in order to get as much air into and as much blood flowing through them as possible ; and inasmuch as large tracts of pulmonary tissue are prevented from expanding, the remaining portions are subjected to an extreme degree of stretching by the vigorous action of the inspiratory muscles, so much so that, as I have already said, they temporarily lose their power of recoil.

Pneumothorax.—Finally I ask your attention to pneumothorax as admirably illustrating our principle. By far the most common cause of this condition is the rupture of a phthisical cavity : a small valvular aperture forms in the surface of the lung and overlying pleura, and allows the air to be pumped out of the lung into the pleura during expiration, when the intra-pulmonary air-pressure is positive, but does not permit it to be sucked back again during inspiration. Thus, as Samuel West contends, the layers of the pleura are forcibly

separated, and the pleural cavity gradually pumped up with air during the expirations. In this way the lung on the affected side gets encroached upon, and may in the end be completely collapsed, for the pressure of air in the pleura, during the expiratory periods at least, will be decidedly positive. The sound lung tends to be encroached upon also, for the mediastinum, being no longer subjected to pulmonary suction on the affected side, is sucked over to the sound side, as well as pushed over by the positive pressure on the side affected. There is thus a great diminution in the pulmonary area, for not only is there in extreme cases complete collapse of one lung, but the other lung is encroached upon. Hence it is imperative that the inspiratory muscles should come into vigorous action in order to get every available air-vesicle well expanded, and every available capillary opened out to its maximum, and what we actually find in these cases is that the chest is in a position of extreme inspiration or even hyperinspiration. Expansion on the affected side would tend to occur, even without excessive inspiratory effort, from the pumping up of the pleura with air, but as a matter of fact the inspiratory muscles contract powerfully on this side as well as on the other.

I would draw your attention to the fact that in all the cases of expanded thorax which we have considered the breathings are shallow. This may seem strange, but it should be remembered that it

is only in this way that the mean size of the chest can be increased: if with the increased inspiratory effort there were a corresponding increase in expiratory effort, the mean size of the chest would remain unaltered, and the advantages which follow upon an increase in the mean would not be secured.

I have now cited several instances of dyspnœa in which the chest is expanded, and did time permit I might add to their number, but I have given enough to illustrate my thesis. I trust I have succeeded in establishing to your satisfaction what seems to me at least to be a not unimportant clinical generalisation, linking together as it does several disjointed clinical phenomena.

I do not wish to imply that in all cases of dyspnœa there is a predominant action of the inspiratory muscles, resulting in thoracic expansion. Cases occur in which the expiratory muscles are thrown into convulsive action, but these cases are peculiar. In whooping-cough, for instance, there occurs a series of expirations, followed, however, by a period in which the chest is greatly expanded by vigorous inspiratory efforts; again, the presence of a foreign body in the larger air-passages, such as the larynx or trachea, may lead to violent expiratory efforts to dislodge it; while, again, the sudden and acute dyspnœa produced in animals by blocking the trachea, or by opening both pleural sacs, leads to expiratory efforts of a convulsive nature. In reference to these latter cases, I may point out that

such sudden and urgent dyspnœa would in the ordinary course of nature signify generally the presence of some foreign body in the larger air-passages, and it seems not improbable that the expiratory convulsions which occur in the cases just mentioned may be due to the organism falsely interpreting the cause of the dyspnœa.

In conclusion, gentlemen, let me thank you for the interest you seem to have taken in these lectures. It has gratified me not a little to have been followed with such close attention.

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