**Why does phthisis attack the apex of the lung? : a discussion at the Medical Society, London Hospital, October 29th, 1903, opened by Dr. Arthur Keith.**

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# *WHY DOES PHTHISIS ATTACK THE APEX OF THE LUNG?*

*Statement of the problem.* Why is it that, in its initial attack, the tubercle bacillus usually selects the apex of the lung? Most of you will answer, and the answer I admit is true, that it is because the apex of the lung expands least. But if I ask you further—"Why does the apex of the lung expand less than the other parts of the lung, and why should its comparative immobility make it more susceptible to tuberculosis?"—I then put questions to you that have never been satisfactorily answered; yet full and satisfactory answers are urgently needed to these questions. Every year about 80,000 people are swept out of this country by that form of tuberculosis which first attacks the apices of the lungs. If it be the immobility of the apices that render them susceptible to tuberculosis, then every endeavour to prevent this disease must lie in the direction of securing complete apical expansion of the lung. How then is the apex of the lung expanded?

*Other factors of the problem.* Before answering that question, I must point out that any theory which aims at giving an explanation for apical tuberculosis must also account for the following facts:—(1) Why the apex of the lung becomes increasingly susceptible to tuberculosis from the fifth to the twenty-fifth year: (2) Why the apex in the female, from the tenth to the twentieth year, is more susceptible than that of the male during the same period: (3) Why children that suffer from stenosis of the pulmonary aorta are so liable to phthisis: (4) Why people who suffer from heart disease are less liable to phthisis. The theory must also explain why it is, as Dr. Kingston Fowler has shown, that tuberculosis usually attacks the various parts of the lung in definite sequence—for after it has appeared in the apices of the upper lobes, it then seeks out the apices of the lower lobes, subsequently burrowing in the dorsal parts of the lobes, usually leaving the ventral or sternal parts free.

*Correlated problems.* There are certain correlated problems, too, which must be taken into consideration. These are:—Why does pneumonia attack more frequently the basal lobes of the lungs—of the right more frequently than of the left? Why is it that emphysema attacks, for the greater part, just those parts of the lung which are least liable to tuberculosis?

*Statement of the explanation.* What I shall try to demonstrate to you is that the incidence or localization of pulmonary disease can be explained by a truer conception of the mechanism of respiration. I admit that the current teaching of respiration throws little or no light on these problems. That is because the current teaching is only partly true; it leaves out of sight a number of most important factors. The points I wish to emphasize are these:—

(1) The expansion of the apex of the lung is obtained by the contraction of the diaphragm.

(2) That from birth to the twenty-fifth or thirtieth year the diaphragm becomes relatively smaller in size and less important as a muscle of respiration, the intercostal and abdominal muscles taking over a corresponding increase of function; hence the gradual rise in mortality from the fifth to the twenty-fifth year.

(3) The thoracic or female type of respiration, in which the diaphragm plays a less part than in the male, is ill adapted for expansion of the apex of the lung; hence the rise in female mortality at early puberty (tenth to twelfth year): it is then the female type of respiration is assumed.

(4) Cardiac dyspnœa calls the diaphragm into active use, hence the lessened susceptibility in heart disease.

(5) In pulmonary stenosis the decreased supply of blood results in part of the pulmonary factory being shut down: that is the part least used in health, namely, the apex.

(6) The apex of the lower lobe is second in the sequence of attack, because after the apex of the upper lobe has been rendered partially fixed or immobile by the onset of disease, the top of the lower lobe becomes the functional apex, and therefore offers the same susceptibility as the real apex. It is well known clinically that disease of the apex of the lung leads at once to a limitation of the movements of the diaphragm. Disease in an organ brings about instantly an inhibition of the muscles which act on the diseased or injured part.

(7) The circulation of blood and lymph in any part of the lung is dependent on the respiratory movements of that part. The part with a sluggish blood and lymph circulation is, I

presume, a part offering a favourable nidus for the tubercle bacillus. See, for instance, that portion of the epididymis which lies at the junction of the blood supply of the testicle and the blood supply of the vas deferens.

*Behring's theory assumed to be right.* Further, I assume that Behring is right when he says that the tubercle bacilli are not carried to the lung by the air passage, but commonly by the blood. A partially expanding apex receives less blood and less air than the other parts of the lung; it is not a matter of the apex being more, but less, exposed to infection than the rest of the lung. As Dr. Salaman pointed out to me, it is immaterial to this theory of apical tuberculosis, whether the bacilli be air borne or blood borne. Owing to its lessened circulation of blood, and especially of lymph, the apex is rendered the better bed for their growth.

*The question of susceptibility.* I am not competent to deal with this question. The microscopical structure of the apex is the same as that of the other parts of the lung, but that does not exclude the possibility of the apex being more susceptible to tuberculosis than the rest of the lung, not from any weakness of its circulation, but on account of other unexplained reasons. Naegeli and Behring have shown, and my experience in the dissecting room quite bears out their observations, that all of us by the fiftieth year have been the subjects of apical tuberculosis. How far our mechanism of respiration, and how far a natural immunity has preserved us, are problems on which I can throw no light.

*Expiration is due not to an elastic recoil, but to a muscular act.* Having now laid before you the problem, and a theory to explain its phenomenon, I find it necessary, before describing the mechanism of apical expansion, to deal with certain current errors in the teaching of the mechanism of respiration. A reference to text-books of physiology will show that one and all of them regard expiration as a mechanical non-muscular act. That view can be traced to the classical work of Dr. Hutchinson published in 1835. One has only to examine the respiratory movements to see that such a conception is erroneous. At the commencement of an inspiratory movement the muscles of respiration can be seen to start into action as well as those of expiration. In bending the elbow the active relaxation of the triceps has much to do with that movement as the active contraction of the biceps. From the beginning to the end of a respiratory movement all the muscles of respiration can be seen to be in action: in inspiration the inspiratory muscles overcome the expiratory; in expiration the expiratory overcome the inspiratory. From the beginning to the end of the movement each rib is balanced, steadied and controlled by the action of the muscles which begin or end on it. That statement is part of a well recognised law that a muscle never acts *without* the active co-operation of its opponent. It has been observed over and over again that the internal intercostals are in action during the phase of inspiration, and have therefore been regarded as inspiratory muscles. They are really expiratory, being *more strongly* in action during expiration than in inspiration. Hutchinson's conception of the elastic recoil of the ribs and cartilages as a cause of expiration, was due to an error of observation. When the muscles are removed from a thorax, the ribs assume an expiratory position when the thorax is held apex upwards; they fall into a position of inspiration when the thorax is held apex downwards. Powell showed long ago that the ribs and cartilages presented no elastic resistance within the limits of normal respiration. The elasticity of the lungs plays only a passive part in expiration; were the whole expiratory movement caused by the elasticity of the lungs, everyone of us would suffer from emphysema in three months' time. So delicate is the elasticity of the lung that artificial respiration quickly destroys it. The rate at which the lung collapses during expiration is solely determined by the muscles of respiration.

*The nature of the pleural bond.* The surface of the lung is bound to the chest wall in a very cunning and ill-understood manner, viz.—by an atmospheric ligament. The rubber sucker I show you has a base of one square inch; if applied to a piece of glass I can lift it by the sucker as long as the glass does not weigh more than 15 lbs. The sucker may be moved along the surface of the glass without losing its hold. Now every square inch of the surface of the lung is so bound to the chest wall, and by adopting an atmospheric bond the surface of the lung can glide freely within the walls of the thorax, visceral pleura rubbing on parietal pleura, without the bond being injured or broken. The total strength of the pleural bond of one lung—the weight required to separate the visceral from the parietal pleura is over half-a-ton; for both lungs, a whole ton. The greatest force our muscles of inspiration can exert is only one sixth of that amount. By no muscular effort can the pleural bond be broken.

*Surfaces of direct and of indirect pulmonary extension.* The lungs are bound to the walls of a movable cage, every expansion of which leads to a direct enlargement of the underlying part

of the lung. Each enlargement or contraction of the chest wall thus brings about an increase or diminution of the air and blood contents of the lung.

*Certain surfaces of the lung expand directly, others only indirectly.* Another partial truth which has been propagated in physiological text-books for 60 years is that, with the movement of inspiration, the thorax is expanded in every diameter—from base to apex by descent of the diaphragm—from side to side by the upward movement of the ribs—from back to front—also by the upward movement of the ribs. Now the deduction that the student draws from these statements is that the lung expands in every direction, and I know most medical men are of that opinion. The truth is that there are three directions in which the lung does not expand: it does not and cannot expand upwards, nor backwards, nor inwards. It cannot expand upwards—in fact, with inspiration, as Dr. Colbeck and other physicians have observed, there is really a slight *descent* of the apex—because the *real* apex, which is that part of the lung in front of the neck of the first rib, lies in contact with a non-muscular, yielding part of the chest-wall. The lungs cannot expand inwards because they are in contact with the heart and other structures of the mediastinum; and they cannot expand backwards because the posterior surfaces of the lungs are in contact with the spinal column and the spinal segment of the ribs—the segment to which the spinal muscles are attached. The only movement which takes place in the spinal segment of a rib is one of pure rotation, a movement which cannot and does not help to enlarge the thorax. Nay, in full costal inspiration the spinal segments of the lower ribs (with the exception of the twelfth) move slightly forward and thus somewhat diminish the thorax. In inspiration, then, the lung cannot expand upwards, inwards or backwards; expansion at the apical—mediastinal or posterior surfaces of the lungs can only be obtained indirectly. Therefore, I name these three surfaces those of indirect expansion—expansion obtained by the enlargement of the opposite wall.

*Surfaces of direct expansion.* Of the three surfaces of direct expansion, diaphragmatic, ventral and lateral, the first named is by far the most important, and the last, least. Mere depression of the diaphragmatic surface to the extent of one centimetre—a common extent in quiet inspiration gives an increase to the pulmonary capacity of 250 cc.—the amount of air taken in when an ordinary breath is taken. In this movement the apex of the lung is expanded indirectly. Next in importance to the diaphragm for the direct expansion of the lung is the anterior or ventral wall of the thorax (*see* Fig. I.) In women with a well marked costal or chest-heaving type of respiration, expansion of the lung is effected by the expansion of the anterior wall more than by the diaphragm. The forward movement of the chest wall occasions an indirect expansion of that part of the lung beneath the dorsal wall of the thorax (*see* Figs. II., III., IV.) Least important of all is the lateral wall. In many people, especially men with a pure abdominal type of respiration, there is no outward movement of the lateral wall of the thorax.

*Dorsal, lateral and ventral walls of the thorax.* The demarcation of the surface of the thorax into three walls—dorsal, lateral and ventral—need explanation and justification. It is a strictly physiological division, founded on the observation that a typical and complete costal arc—the rib with its corresponding cartilage—is made up in every air breathing vertebrate of three physiological segments, without a recognition of which it is impossible for a physician to understand the respiratory capabilities of the many types of chest he has to percuss and explore. The spinal segment of a costal arc is that part to which the spinal muscles are attached; the *angle* of the rib marks the junction of the spinal with the lateral segment. The spinal segment is the axis round which a costal arc rotates; while these segments form the spinal wall of the thorax they are only indirectly concerned with respiration. These segments are really lateral levers of the spine; every strand of muscle attached to them—spinal parts of the intercostals, *levatores costarum*, erector spinæ—are in no sense muscles for directly enlarging the chest; they are in every sense spinal muscles for spinal movements. Thus the angles of the ribs mark the junction of the dorsal and lateral walls of the thorax; if one would indicate that junction in the living body, a line must be drawn on each side of the back, beginning above on a level with the vertebra prominens, two and a half inches from it, and ending below at the twelfth rib, three and a half inches from the mid line of the spine.

The recognition, on the surface of the chest, of the line of junction between the lateral and ventral segments of the costal arcs, is also a matter of importance. Fig. I. will give material assistance in its recognition. In that figure a thorax is viewed from above on its apical surface, the successive costal arcs being seen in profile. Practically only the anterior or ventral wall is seen when the thorax is so viewed. The lateral segment, it may be said

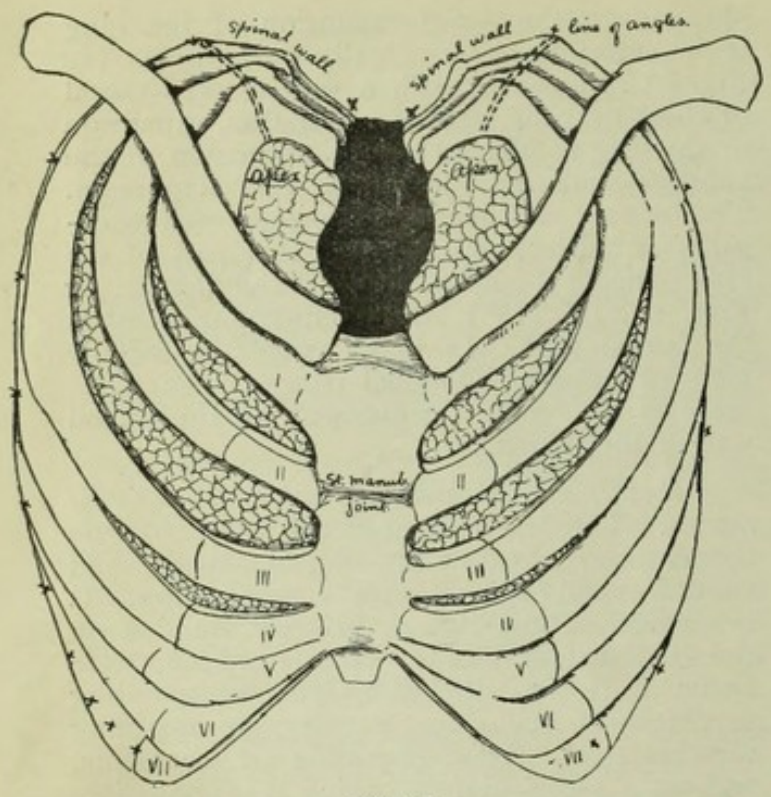


FIG. I.

A thorax in the position of extreme inspiration, viewed from above. The part of the thorax thus seen makes up what is described in the text as the anterior wall of the thorax. The crosses mark the position of the great fissure of the lung. Part of the spinal wall is also seen.

at once, is absent in the first, second and third costal arcs; it begins in the fourth, and increases, both in length and obliquity, from the fourth to the eighth; after the eighth the lateral segment diminishes in length and has disappeared in the twelfth. These lateral segments are nearly on the same plane and are not seen when the thorax is viewed from above (Fig. I.) They form the peculiar flattened area which everyone must have noticed on the side of the dried and mounted thorax. In people with wide chests (*i.e.*, flattened from back to front) the lateral segments are comparatively short; in round and narrow chested people (*i.e.*, flattened from side to side) these segments are long. The characteristic of the *ventral segment* of the costal arcs is (*see* Fig. I.) that each succeeding segment from above downwards is part of a greater circle than the one above it. When the thorax is viewed in profile from above, each of the ventral segments is seen standing out further forwards than the one above, whereas we have seen that in the lateral segments the last segment is on the same plane as the first. The cartilages are included in the ventral segments; each succeeding cartilage from the first to the eighth is longer than the one above it, and more obliquely placed. Beyond the eighth rib the ventral segments cease to exist.

*Relative functions of the lateral and ventral segments of the costal arcs.* Since each of the ventral segments of the ribs stands out some distance in front of the one above it, it is plain that, when these segments are elevated, all that part of the lung which lies beneath them is directly expanded forwards. Now if you look at the part of the lung which lies beneath the ventral segments, you will see that it is exactly the costal surface of the upper and middle lobes of the right lung, and the upper lobe of the left lung (Fig. I.); that is to say the line of demarcation between the ventral and lateral segments of the costal arcs corresponds to the great fissure of the lung. It will be seen that this observation is an approach towards the solution of a problem which has never been explained—"Why is the lung divided into lobes?"

Since each costal cartilage increases in length and obliquity from above downwards, it is plain that the elevation of the cartilages will also cause an increase in the *transverse* diameter of the chest. Thus the ventral segments of the costal arcs cause, by their elevation, a direct enlargement forwards of the area of the lung beneath them, and indirectly an enlargement of the transverse diameter of the chest, which is attended by an expansion of the lung on their lateral surface.

The lateral segments of the costal arcs cover the lateral surface of the lower lobe of the lung, and have a direct action in expanding that surface, and an indirect effect in the expansion of the ventral surface in the following manner:—when the lateral segments are elevated by the external intercostals, they not only move upwards, but also outwards. The outward movements cause a direct expansion of the lateral surface of the lung; the upward movement, since each lateral segment increases from the fourth to the eighth, leads to an increase in the antero-posterior diameter of the thorax, and therefore to an expansion forwards of the ventral surface of the lung. The lateral segments, however, decrease from the ninth to the twelfth ribs, therefore the elevation of the lower segments, while they do cause an increase in the lateral diameter of the chest and a direct expansion of the underlying lateral surface of the lung, lead to a *decrease* of the antero-posterior diameters.

*The muscles of respiration.* As a rule the first to be enumerated amongst the muscles of respiration are the levatores costarum, which are not in any sense respiratory, but spinal muscles. The muscles of the abdominal wall on the other hand are usually included in the list of extraordinary muscles of respiration, and yet no one

ever saw a man or animal breathe—except in disease, and then under great stress—without a movement, more or less free, of the belly walls. Whether the type of respiration be diaphragmatic or costal, the muscles of the belly wall are used; they are first and foremost muscles of respiration; in comparison, their other functions are of minor import. If the divisions of the thorax I have given, as dorsal, lateral and ventral surfaces, are accepted as real functional divisions, it will be found that an explanation has also been provided for the arrangement of the abdominal musculature. It also falls into dorsal, lateral and ventral zones or groups. The rectus and the triangularis sterni are the respiratory opponents of the inter chondrals—the intercostals of the ventral segments. The external oblique is the respiratory opponent of the external intercostals, the chief muscles of the lateral wall of the thorax. The internal oblique is a functional compliment to the internal intercostals—they are parts of the same physiological sheet and are expiratory. All the muscles of the dorsal segment—quadratus lumborum, spinal segment of the external intercostals, levatores costarum, ilio-lumbalis, accessorius, cervicalis ascendans, are essentially spinal muscles; the only part they play in respiration is as fixing or synergic muscles; they steady and restrain the spinal segments of the ribs, round which the lateral or real respiratory segments of the ribs turn.

*Movements of the roots of the lungs.* The roots are implanted on the mediastinal surface of the lung, and it is important to notice that they lie nearer the dorsal than the ventral surface, and nearer the diaphragmatic than the apical surface. Now the apical and dorsal surfaces are fixed; there is no expansion either upwards or backwards. Consequently, were the root fixed, as every physician and physiologist teaches, then it would follow that that part of the lung which lies between the apex and root—two fixed points—and that part which lies between the dorsal surface of the lung and the root—also two fixed points—could not expand. To permit the expansion of the lung, then the root must be freely movable. Through the pericardium acting as a tendon, the muscular crura of the diaphragm are attached to the roots of the lungs and to the base of the heart. The heart is the pump of the lungs, and in every movement the lungs carry their pump with them. With X rays these movements of the base of the heart can be seen; with an ordinary inspiration the base of the heart and the roots of the lungs move forwards and downwards about half-an-inch; with pure diaphragmatic breathing, the movement is almost entirely in a downward direction; in costal breathing the movement is a forward one. The trachea and main bronchi are constructed like the body of a concertina, for expansion and contraction, and permitting freely the respiratory migrations of the roots; with each deep breath the rings of the trachea can be felt, with a finger placed in the suprasternal depression, separating and collecting.

There is another movement of the roots of the lungs, besides the downward and forward one, which ought to be, but at present is not, recognised—that is a movement in an outward direction. The mediastinal surface of the lung on which the root is implanted, lies against a wall that permits of no expansion. Were the root fixed, no expansion on this surface were possible. Hence with each breath, as one can prove in performing artificial respiration with the lungs still *in situ* within the opened thorax, the two bronchi, instead of descending to the lung in oblique directions, as in the phase of expiration, assume a more horizontal position, the angle between the right and left bronchus rising from one of 70° to one of 120°. A discussion has been carried on by anatomists, regarding the proper size of the interbronchial angle; some give the smaller, some the larger. Both are really right, only the smaller one represents the respiratory, the larger one the inspiratory, phase.

*What is the apex of the lung?* There can be no doubt as to what is the anatomical apex of the lung; it is the conical pulmonary point lying in front of, and on a level with, the neck of the first rib. From a clinical point of view, such an apex is of no importance. Unfortunately, the clinical apex of the lung is like a West African Colony—it is sharply defined on one border, but the rest is an indefinite hinterland. The clinical apex certainly includes that part of the lung which lies within the semi-circle of the first rib, but as to how much more of the upper part of the lung should be included with this to form the apex, probably no two physicians would agree. Certain it is that when phthisis invades the apex, it appears at a point which lies on a level with the first rib or first intercostal space, vertically below the mid-clavicular point. Can any definite line be drawn which will separate from the rest of the lung that part which is first and most liable to tuberculosis, and which therefore will demarcate what may be called the clinical apex? I think such a line can be drawn. It commences in front, at the first sterno-costal junction; it ends behind at the upper end of the great fissure of the lung which is marked on the surface of the body by the second dorsal spine.

*The normal respiratory expansion of the clinical apex.* Is there then less respiratory movement of the clinical apex than of the rest of the lung? By what means is it normally expanded? The means are two in number; the first and most efficient, although indirect, is the diaphragm; the second, direct but less efficient, are the movements of the first, and perhaps I should add, of the second rib. By these two means only—by one alone, or both combined—can the clinical apex of the lung be expanded.

To estimate the respiratory value of the first rib, it is of advantage to examine its action on the apex of the lung in what is called the pure costal or thoracic type of respiration, such as is commonly seen in women. In them the diaphragm descends to only a slight extent; it spends its force in lifting the thoracic cage off the abdominal viscera. In such a type the sternal end of the first rib will rise upwards and forwards to the extent of half-an-inch. Its influence on the apex of the lung is shown in Figs. II. and III. There are two other structures move with the first rib, namely, the manubrium sterni and Sibson's fascia, which covers over that part of the apex of the lung which appears within the concavity of the first rib. When the first rib and manubrium sterni are viewed from the side, they are seen to lie in the same line, the manubrium prolonging the first rib to the sterno-manubrial joint. Not only do they lie in one line, but, owing to its short, thick, jointless cartilage, the first rib and manubrium sterni form a common lever. When a full costal inspiration is taken, the manubrium sterni, first rib and Sibson's fascia rise together; a considerable bending takes place at the sterno-manubrial joint (the angulus Ludovici becoming more prominent). A slight rotatory movement takes place in the first costal cartilage at the same time. The three structures just named—first rib, Sibson's fascia and manubrium sterni—form the operculum or lid of the thorax. The lid is hinged to the vertebral column by the neck of the first rib. Now, as the lid neither expands or contracts as it rises and falls, that area of the lung which is in contact with the lid neither expands nor contracts. On the other hand, as the manubrium sterni rises upwards and forwards, it recedes further and further from the spinal segments of the second and third ribs, and therefore that part of the clinical apex which lies between the sternal end of the first rib and the spinal ends of the second and third is expanded—although not efficiently. On the other hand, the real apex of the lung—that part in front of the neck of the first rib, which lies just in front of the axis of movement—is stationary. Thus it will be seen that the costal type of respiration is one ill adapted for a satisfactory expansion of the pulmonary apex; statistics show that women become more liable to phthisis just before puberty; that is also the date at which their peculiar type of respiration is assumed, and this assumption has nothing to do with their style of dress.

The diaphragm, although an indirect agent, is a very efficient agent in expanding the apex of the lung; at least, as long as the chest is approximately normal in shape. The clinical apex of the lung is nearly a perfect cone. In the descent of the lung, caused by the diaphragm, each segment of the apex is brought within the sphere of a wider circle, and therefore expands in every direction. Without a free and full movement of the diaphragm, there can be no efficient expansion of the apex of the lung.

*Retrogression of the functional importance of the diaphragm.* I cannot give a satisfactory explanation of why it is so, but there can be no question of the fact, that from the second year onwards—that is, from the time a child learns to walk—the importance of the diaphragm as an agent of respiration becomes relatively less, while the other two respiratory surfaces of the thorax—the ventral or anterior, and the lateral—become relatively of greater importance. This is seen in the growth changes that affect the three corresponding surfaces of the lung. In the child at birth, as in the dog, cat or ordinary mammal, the area of lung in contact with the diaphragm constitutes nearly one fourth of the total superficial area of the lung; by the age of 25 it has become relatively reduced, so that it then constitutes from one sixth to one eighth of the total surface area. The relative reduction of the diaphragmatic surface, and relative increase of the ventral surface, is greater in woman than in man. Now when one remembers that the diaphragm is the only efficient means of expanding the apex of the lung, and when one sees that the years in which a relative decrease in the use of this muscle takes place are also the years in which the mortality from phthisis keeps mounting up, one cannot resist the conclusion that here there is a correlationship of cause and effect.

When phthisis occurs, there is a limitation in respiratory movement in that part of the chest wall that overlies the lesion, but there is also a very marked limitation in the movements of the diaphragm on the affected side.

*The pterygoid chest.* From of old it has been recognised that people with this type of chest are the choice subjects of phthisis. Now

the pterygoid chest is one which has moulted all its infantile characters prematurely. Its leading feature is that the diaphragmatic surface has lagged in development far behind all the others; the diaphragmatic surface is pathologically small. The ribs which, in the child, were nearly horizontal, are maintained in that position by all the muscles of respiration, but especially by the intercostals and diaphragm, and it was the maintenance of the ribs in the horizontal position that gave the child its rounded, tubby chest and extensive diaphragm. With the failure of the diaphragm as a muscle of support, the anterior ends of the ribs drop down and down until they assume a position half-way between the horizontal and the vertical, and with this fall the chest becomes pterygoid.

*The movement of the lung as a whole.* It is usually said that the fissures of the lung are of no functional value for the reason that they may be absent in part or in whole, and because they may be closed up by disease with no recognisable detriment to the patient. The inference is not a fair one, for there are few parts of the body which have not been removed by the surgeon, and yet, when removed, have occasioned little or no disturbance to the economy of the body as a whole; still, in many cases, we know that the parts removed are of functional value. It

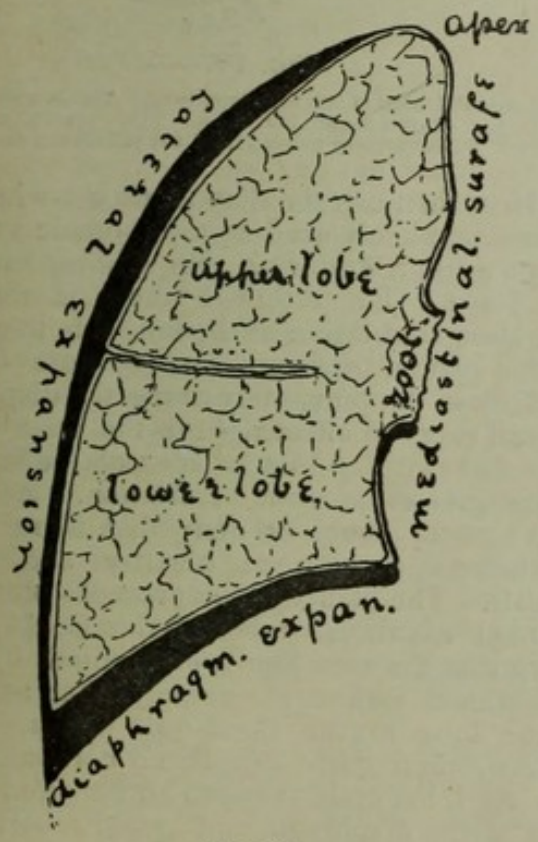


FIG. II.

Vertical section in a transverse plane of the right lung. The blackened zones represent the amount and direction of expansion due to inspiration.

would be strange indeed if the great fissures of the lung, which are wonderfully constant in size and position, were of no value. Certain it is that their functional value will never be recognised as long as the current teaching of the mechanism of respiration is accepted as completely true.

The meaning of the fissures will be seen when the movements of the lung as a whole are examined. These are represented diagrammatically in Figs. II., III., IV. The movements represented are those which take place in the ordinary type of respiration where the abdomen is used as much as the thorax. Take the movement of the upper and middle lobes first; they lie above the great fissure of the lung. Now we have seen that the line of junction between the ventral and lateral walls of the thorax correspond approximately to the great fissure (*see* Fig. I.) The surface of the upper lobe is, for the greater part, in contact with the ventral wall of the thorax, and must therefore follow the movements of that part. Only a small part of the upper lobe, behind the sternal insertion of the sixth rib, lies in contact with the diaphragm. Now the anterior inferior angle of the upper lobe moves very little in an upward or downward direction in ordinary respiration, for the upward movement of the sternum and costal cartilages compensate and neutralize the comparatively slight descent of the anterior part of the diaphragm. The diaphragm has little direct influence on the upper lobe; it is expanded chiefly by the ventral wall of the thorax, and since that wall moves forwards and upwards, the underlying part of the upper lobe also expands in that direction. The direction of the expansion is shown in Fig. III., by the direction of the arrows. The upper lobe, properly speaking, is a costal lobe; it is worthy of remark that it is relatively large in women. While the the upper lobe only reaches the diaphragm at its anterior-inferior angle, the lower rests by its base on the diaphragm; it tapers to an apex above. The arrows in Fig. III. show the main inspiratory movement in this part of the lung. One knows, from observations with the X rays, that the movement of the diaphragm is in a downward and forward direction, the posterior part descending more than the anterior. There is no expansion of the lower lobe in a backward direction, for that rests against the spinal wall of the thorax; the lateral aspect of the lower lobe is in contact with the lateral wall of the thorax, but the movements in that wall is limited in extent. Hence the main movement of the lower lobe is in a downward and forward direction (Fig. III.), but the extent of the downward movement becomes greater as one passes

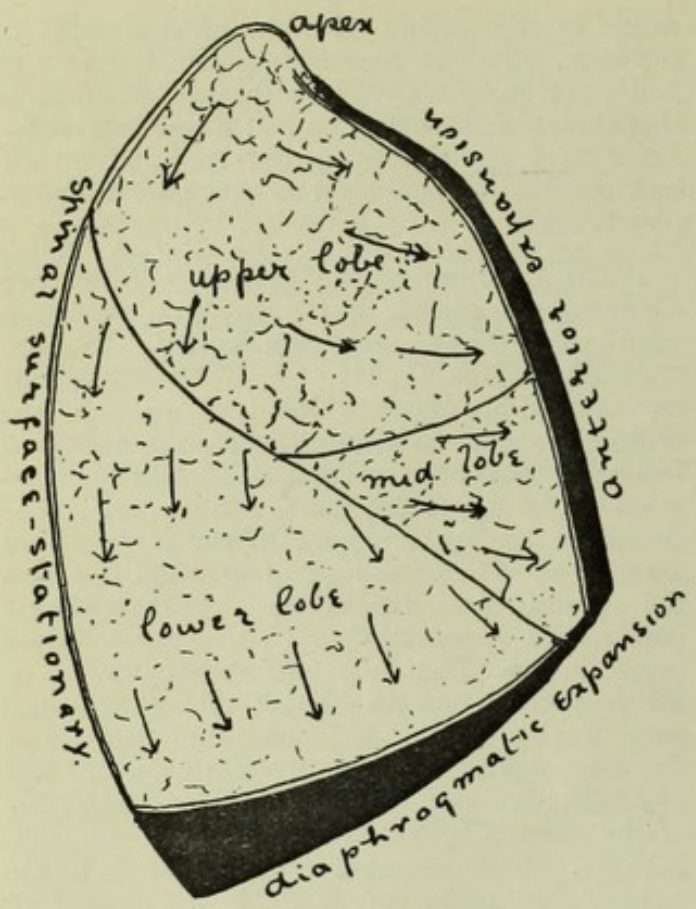


FIG. III.

The right lung viewed from the side. The blackened areas represent the expansion of inspiration. The arrows show the direction in which the expanding movements are effected.

from the anterior to the posterior angle of the lower lobe. Thus it will be seen that the great fissure of the lung is the dividing line between two respiratory forces, diaphragmatic and costal. The diaphragmatic force, however, crosses the great fissure of the lung and expands the posterior part of the upper lobe right up to the anatomical apex. It will be observed (*see* Fig. III.) that while the lower or anterior end of the great fissure is almost stationary, the posterior part descends on inspiration, and thus the fissure tends to assume then a more horizontal position.

The part of the lung which is specially subject to the traction of the diaphragm can be recognised on the dead lung in the following way. When a lung is removed from the body and moderately *inflated*, the impressions of the ribs will be seen on the anterior costal surface of the upper and middle lobes of the right lung (the upper lobe of the left), but on the spinal aspect of the upper lobe not a single impression will be seen. The whole of the lower lobe is destitute of costal impressions, with the exception of a limited area in the neighbourhood of the lower or anterior end of the great fissure. The interpretation I put on this observation is that those parts of the lungs which show costal impressions are parts which are directly expanded by the overlying ribs and chest wall, while the smooth areas are those which glide beneath the costal walls, under the influence of the diaphragm. If such an inference be just, then the pleuritic sounds—excepting those caused by localised cicatricial contraction of lung tissue—should seldom be heard above the great fissure, but should be confined to the lower lobe, and should be greater in length and loudness the nearer one approaches the posterior part of the diaphragm.

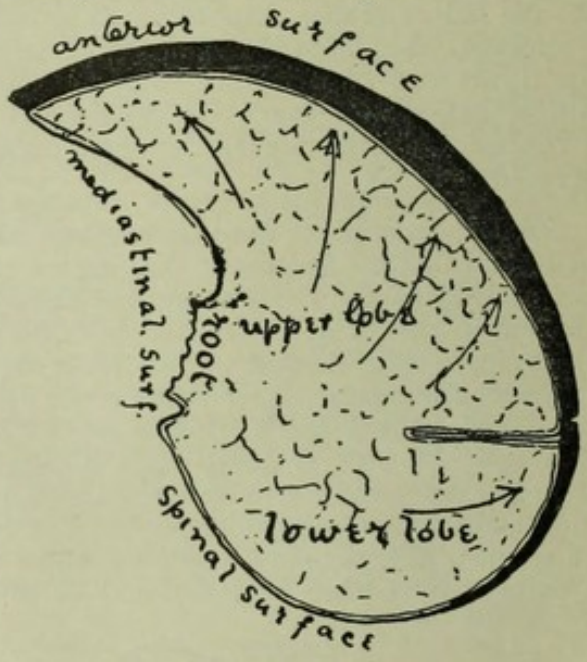


FIG. IV.

Horizontal section of the right lung. The blackened area indicates the expansion of inspiration. The arrows show the direction of the inspiratory movement of the pulmonary tissue.

How then can the diaphragm act when the lung is completely fixed to the thoracic walls by firm pleuritic adhesions? No gliding can then take place. But although there is then no inspiratory translation of the lung due to gliding of the pleural surfaces, yet, because of the elasticity of the pulmonary tissue, the diaphragm can still exert its effect right to the apex of the lung, but with a loss of muscular effort due to the greater amount of friction. It is the habit now to doubt or deny that there is any correlationship between pleurisy and apical phthisis. The pleurisy that precedes phthisis is regarded as always tubercular. Could it be shown that the view firmly entertained by older clinicians is erroneous—namely, that adhesion of the lung to the chest wall predisposes to phthisis, then there can be no correlationship such as I maintain between an impaired movement of the diaphragm and apical tuberculosis, for without doubt pleuritic adhesion render the respiratory efforts of the diaphragm less effective.

*The effect of using the minimum of our respiratory capacity.* One may look on the lung as a factory into which the venous blood and the pure air are carried as raw materials, out of which the arterial blood passes as the finished article, and the vitiated air as the waste product. It is only in brawny people that these factories are worked to their maximum capacity. In many of us, and it is notoriously so in those that become the subject of phthisis, the lungs are only used to the minimum of their capacity; the parts that work most easily are used most, and those which require the greatest exertion are used least. Now, from what has been shown, there can be no doubt that in the human lung it is the upper part which is the most difficult to work; when a shutting down process sets in, the apex is the first to feel it.

*Why should a relative immobility of a part of the lung give a predisposition to phthisis.* If it be granted that the apex of the lung, when the respiratory function is used to only a minimum of its capacity, is the least movable part, why then should it be the most susceptible to phthisis? My reason for broaching this problem is not that I can give a full and satisfactory answer to it, but that, in the course of my investigations, I came across a number of unconsidered facts which seem to bear on it. These observations deal with three different points:—

(1) With certain features of the elasticity of the lung.

(2) With the manner in which a negative pressure is produced and regulated in the lung, and the relationship of the radicles of the pulmonary vein to the surrounding pulmonary tissue.

(3) With the lymphatic circulation of the lung.

The theory under which I made these observations was that the blood and lymph circulation of any part of the lung depend on the respiratory movements of that part; with a limitation of movement there follows a sluggish and diminished blood and lymph circulation—conditions which I assume to be favourable to the success of a tubercular attack. I believe it is now accepted by pathologists that phthisis begins primarily in the pulmonary (peri-arterial) lymphatics.

*The elasticity of the lungs.* It is usually assumed that the elasticity of the lungs is the same throughout, and that during inspiration every part of the lung undergoes an equal degree of expansion. Such is not the case, for if points be marked on the surface of a partially inflated lung one inch apart, and the lung be then distended to a greater degree, it will be found that the points marked are not any longer equal distances apart. On the diaphragmatic surface, for instance, those points near the circumference will have moved only slightly apart, while those nearer the centre will be $1\frac{1}{4}''$, $1\frac{1}{2}''$, $1\frac{3}{4}''$ or even $2''$ apart. So, too, on the other surfaces of the lung. The areas which expand the most under these circumstances are those at some distance above and some distance below the great fissures on the anterior and lateral aspects of the lung. Those which expand most easily are the last to collapse on deflation of the lung.

If a main bronchus is uncovered in a child's lung, and branch after branch of it be exposed until a termination is found in a lobule on the surface of the lung, it will be found, when this system is inflated, that the elasticity and amount of expansion increases as one approaches the surface of the lung. That is to say that under an equal force the terminal or surface part distends most, the part at the root of the lung least. Hence, on an inspiratory movement, we may infer that the parts towards the surface of the lung distends first and most, the parts towards the root last and least. This fact bears at once on an important point.

It is well-known that the initial lesion of apical phthisis appears about $1\frac{1}{2}''$ below what I have called the anatomical apex, and $1\frac{1}{2}''$ in front of the posterior or spinal surface of the lung. If the distension of the apex of the lung is dependent on the diaphragm, then the part most distant from that muscle—the tipmost part of the apex—ought to be the most susceptible part—so it would be if the elasticity of the lung were the same throughout. But since the surface layers of the apex are more easily distended than the deeper layers, they still undergo a respiratory expansion when the deeper parts of the lung are partially shut down. It is on this ground that I explain the appearance of phthisis some distance below the real apex.

The bronchi and bronchioles are richly supplied with elastic tissue, the fibres of which are arranged, not in a circular, but in a longitudinal, direction. With each breath the air-channel is elongated; with each expiration it retracts under the influence of its elastic coat. Hence it is that each bronchus and each branch of a bronchus lies in the axis or direction in which it expands. From the direction of the bronchi in any part of the lung, one can formulate exactly the direction in which expansion takes place in that part. Thus the two bronchi to the apex pass upwards almost

vertically; so does that to the apex of the lower lobe. They lie exactly in the line of force exerted by the diaphragm. As Birsch Hirschfeld showed long ago, it is at the junction of these vertical apical bronchi with their finer branches that phthisis sets in. The mistake he made was in supposing that the plane in which these tubes lay to the main bronchus offered a resistance to the entrance of air to the apex of the lung.

Here is another fact which seems to me of importance. If one takes a bronchus, with its accompanying pulmonary artery and vein, and tests the elasticity of each, it will be found that the artery is by far the most elastic, the vein by far the least, while the elasticity of the bronchus is intermediate to that of the artery and vein. Yet all three lie in the pulmonary tissue and must accompany that tissue in all its respiratory movements. It is usually said that there is no order in the arrangement of the veins and arteries of the lung. Quite the reverse is true; they are arranged on a most definite system. The veins form the fixed axis on which the respiratory movements of the lung take place; the arteries, on the other hand, lie in the part which expands most. It will be found that the artery lies quite close to one side of the accompanying bronchus; the vein lies at some distance on the opposite side of the bronchus. The side on which the vein lies is the side towards which the lung bends on inspiration. Hence the veins are arranged towards the mediastinal, spinal and diaphragmatic surface of the lung, for although the diaphragmatic surface descends greatly on inspiration, the enlargement of that surface is relatively slight.

*The expanding movements of the lung.* It is commonly taken as an axiom in the mechanism of respiration that the whole substance of the lung expands, as does a balloon, equally, and in all directions. Now the lung is not a balloon, but a myriad of small balloons *strung* together upon a system of broncho-vascular spokes which radiate from the root to the surface of the lung. We have seen that these compound pulmonary spokes are peculiar in two ways—(1) As they approach the surface of the lung they become more elastic and easier of extension; (2) They are composed of structures showing different degrees of elasticity. Now were the expansion of the lung on inspiration what it is usually assumed to be, merely an enlargement of its root-surface diameters, then the pulmonary tissue surrounding the comparatively inelastic structures of the root would undergo practically no expansion. This, however, is provided for by a second and unrecognised movement of the lung, namely, one in which the broncho-vascular spokes of the lung approach and diverge with each breath, in the same manner as a fan may be partly closed and opened by manipulating its rays. The curve of the dorsal spine, the downward and forward movement of the diaphragm, are special adaptatations for this movement (*see* Figs. II., III., IV.)

*The importance of the infundibula of the lung.* In describing the structure of the lung, most teachers speak of the air-cells or alveoli as the essential parts of the lung, and the large spaces or infundibula, of which the air-cells are but hemispherical diverticula, as of little import. Yet, as far as regards the mechanism of respiration—as one soon finds out from a study of the comparative anatomy of lungs—the infundibula are the essential parts. It will be remembered that, as a bronchus is traced to its termination in the lung, it breaks up into bronchioles, muscular, elastic walled tubes. These stand in the same relationship to the bronchi as arterioles do to arteries, only they open not into a capillary net work, but into large air spaces or infundibula, the wall of which are embossed with air-cells. The infundibula are covered with a net work of fine elastic fibres, between the meshes of which the air-cells protrude.

Now it may sound like a paradox, yet it is capable of proof, that when the lung undergoes an inspiratory expansion it is the infundibula that are enlarged mostly; the alveoli are flattened and widened, but, comparatively speaking, only slightly enlarged in capacity. If you take two sacs or balls with equally thick walls, but one large and the other small, you will find, if you inflate both at the same time, that the large one increases at a rate altogether out of proportion to the smaller one. That, of course, is according to a law which is well recognised. Or if a square sheet of rubber be taken (say $4'' \times 4''$), and a piece $3'' \times 3''$ be cut out of it to represent the infundibulum, and numerous small holes cut in the surrounding margin to represent the alveoli, it will be found, when the sheet is stretched equally, to measure $6'' \times 6''$, that 90 % of the increase is obtained by the enlargement of the infundibular space. There is no reason to believe that the law which holds good for elastic sacs outside the body, does not also hold good for the elastic-walled spaces of the lungs.

Were the elasticity of the lung the same throughout, were the infundibula of the same size, and did the chest wall, when it enlarges, pull equally on each part of the lung, then one could say with certainty that, on taking a breath, all the infundibula would enlarge

equally. Now we have seen that the superficial infundibula increase on inspiration more than the deep. Further, the infundibula are not all of the same size. If one were to cut the body into two from side to side, so that the lungs would be divided into a posterior half and an anterior, it would be found that the infundibula in the anterior half were larger than those in the dorsal division. The infundibula then in the anterior part of the lung are larger, and therefore expand more than those in the posterior part. Further, the inspiratory traction of the chest wall is not applied equally; we have seen that the thoracic wall acts most directly on the anterior and diaphragmatic surfaces of the lung: it is on the superficial layers of these aspects of the lung that emphysema is most frequently seen. Once an infundibulum begins to enlarge, the less does the force become that is necessary to expand it further. But on the other hand, and in this lies the pathology of emphysema, it has less power to empty itself. While the infundibulum has gone on enlarging, the bronchiole that leads to it may, and it commonly does, remain of the same size. When the smaller infundibula have become emptied of their *tidal* air, the expanded infundibula, acting thus at a disadvantage, with only a normal exit, have only got rid of a part of their tidal burdens, and hence encroach more and more on the space of the neighbouring healthy acting infundibula.

*The relation of the elasticity of the lung to the pulmonary circulation.* I have made this little divergence into the pathology of emphysema to bring out the important part which elastic tissue plays in the active circulation of the lung, and therefore its importance, if what I maintain is right, in the prevention of tuberculosis. It is the characteristic of an emphysematous patch that it is destitute of elasticity and is bloodless and dry. In what manner does the destruction of the elasticity affect the circulation of the an emphysematous part? Before answering or trying to answer that question, it will perhaps be best to point out that an emphysematous patch, being one in which the circulation of blood and lymph is partially or completely arrested, is one, on my theory, just the nidus that would suit tubercle bacilli. Now emphysema usually attacks one part of the lung, and phthisis quite another. The explanation I offer is this: that, as in heart disease, and that follows quickly enough on emphysema, the inspiratory efforts have to be increased; this increase of effort leads to an increased respiratory activity and movement of the healthy part of the lung. The respiratory mechanical compression and expansion of the lung still maintains the lymph circulation. Besides, there is a distinct difference between a part of the lung, normal in structure, but lying comparatively idle, and an emphysematous patch with its capillaries more or less impervious to the passage of blood.

*The effect of movements of the lungs on the pulmonary circulation.* With an inspiratory movement, the pulmonary circulation is accelerated; during an expiratory, it is retarded. The inspiratory acceleration is due to two causes, neither of which are clearly defined nor explained in works dealing with this subject. In the first place, an inspiratory movement not only expands the infundibula of the lung, but also the blood vessels. That can be proved by experiment. A lung may be placed within a pair of bellows, modified to represent a thorax, with the trachea, pulmonary artery and vein exposed and fixed in the wall of the artificial thorax. In the artery, vein and trachea, tubes are tied containing fluid. On performing an artificial inspiration, as much fluid will be drawn into the vessels as into the bronchus. In life, the pulmonary arteries are filled at a pressure which makes them little affected by inspiratory movements; but it is otherwise with the veins. The first beat of the heart, during an inspiratory phase, shows that the outflow from the pulmonary veins is temporarily checked, the inspiratory rise of blood pressure starts away on the second beat. That effect is due, I suppose, to the dilatation of the veins on inspiration. That dilatation cannot draw the blood back from the heart, but it may draw it on from the pulmonary capillaries. At the commencement of expiration the veins are compressed and partly emptied. The intimate manner in which the veins, especially the radicles of these veins, are united with the pulmonary tissue, is a special adaptation for the enlargement and compression of the venous system with each respiratory movement. The bronchus and artery, on the other hand, are surrounded by wide lymph spaces and vessels; it is chiefly these lymphatics which are expanded and compressed by the respiratory movements, and not the bronchus or artery. The connection of the venous radicles with the infundibular and alveolar air spaces is especially intimate; it is difficult in microscopic sections to tell a venous space or channel from an air space, so identical are their walls. The amount of elastic tissue is of a corresponding amount, and one must infer that these venous spaces expand with inspiration, and are partially compressed in expiration. A part of the lung which is lying in partial respiratory idleness is deprived of these accessory circulatory forces.

*Influence of negative pressure.* This is the second of the two conditions, set up by an inspiratory movement, which accelerates the pulmonary circulation, but, in point of importance, the creation of a negative pressure is of much greater power than the distension of the veins. How then does a negative pressure in the infundibular spaces accelerate—or perhaps a better way of putting the question—how does a positive pressure retard the pulmonary circulation? In this way: when you apply slight pressure to the nail of the finger, the underlying capillaries are partially or completely emptied of blood and their circulation stopped. Now the pulmonary capillaries lie in the partitions which separate neighbouring infundibula. If there is a positive pressure, especially if it rises to any extent, within two adjoining infundibula, the partition wall between them, and the capillaries in it, are compressed. In such conditions the infundibula become a species of Westinghouse brakes applied to the circulatory flow that goes on in their adjoining walls. By raising or lowering the infundibular pressure, the exact rate of the pulmonary circulation can be set and regulated; the higher the pressure, the slower the flow; the lower the pressure, the quicker the flow. It is evident, then, that the infundibula air pressure must be very definitely regulated—but in what manner?

*The function of the bronchial musculature.* The musculature surrounding the bronchioles and the branches of the bronchi, with that found in the posterior wall of the trachea and bronchi, make up an extensive and powerful contractile system, but, as to the function of this system, scarcely an allusion is made in works dealing with respiration. Just as the circulation and pressure of the blood is mainly regulated by the muscular coat of the arterioles, so is the air pressure within the lung, and especially within the infundibula, regulated by the musculature of the bronchioles. Thus the bronchiole musculature regulates and controls the pulmonary circulation. If it contracts or narrows the bronchiole during the inspiratory phase, as I believe it does, it heightens the flow of blood in the walls of that infundibulum because it increases the negative pressure within the space. If it contracts during expiration—normally, I believe it then relaxes—it heightens the infundibular pressure and thus retards the flow. The infundibular pressure has never been measured; certainly, the air pressure in the trachea or bronchi gives no index of the infundibular pressure, any more than the blood pressure in the aorta gives you a clue to the pressure in the capillaries. The ingress of air to the lung, and the whole pulmonary pressure, is regulated by the musculature of the glottis and of the trachea; the distribution and pressure of air to each lung, to each lobe of the lung, to each lobule, is regulated by the musculature of the bronchi and bronchioles.

*The lymph circulation of the lung.* Of the three forces which help the lymph circulation of the body generally, only two are found in action in the lung. When lymphatics appear first in animals they are arranged round the arteries, so that the expansion and contraction of the arteries by the heart-beat becomes the active force that forces the lymph onwards. Valves determine the direction in which the lymph will flow. In the human body the main lymphatics still cling to the course of the artery; in the lung they form a close network round the pulmonary artery and its branches; owing to its elasticity, the pulmonary artery expands and contracts with each pulse-beat, more than any artery in the body. The pulsation of the heart and pulmonary artery ensures a complete lymph circulation in the region of the pulmonary root. The negative phase of the respiratory pressure draws the lymph of the rest of the body towards the thorax; the lymphatics of the lung, on the other hand, are subject to the same respiratory compression and expansion as the thoracic duct, and are thus uninfluenced by the negative pressure. Muscular compression, which acts so powerfully on the lymphatics of the body generally, cannot act directly on the contents of the thorax.

The inspiratory expansion of any part of the lung affects three distinct pulmonary elements—(1) the infundibular air spaces; (2) the lymphatic spaces and vessels; (3) the pulmonary veins and perhaps capillaries. That is to say, the lymph circulation in any part of the lung depends on the respiratory movements of that part. Now the movement of any part of a lung depends on—(1) being so situated that the muscles of respiration can act freely on it (the posterior part of the apex is not so situated); (2) its resistance to expanding forces must be low—the surface layers of the lung offer the least resistance, and therefore expand the most (the point at which phthisis usually appears is 1½″ or more from the surface of the apex); (3) the infundibular spaces must be relatively large, to give free movement, for large spaces distend under a smaller force (the infundibular spaces are larger on the surface than in the depth of the lung, larger in the anterior part than in the posterior part of the lung); (4) the bronchiole muscles determine the amount of expansion, by regulating the entrance and exit of the air, to and from the infundibular spaces.

*The human or mammalian mechanism of respiration contrasted with that of other air breathing vertebrates.* The free and effective mechanism of respiration in birds and reptiles makes one suspect that there is some radical error in our present conception of mammalian respiration. In birds and reptiles the posterior part of the lung forms a thin-walled non-respiratory sac, or series of sacs; when a breath is taken the air-sacs expand at a very much greater rate than the lungs; hence the foul air which lies in the trachea and bronchi at the end of each breath is not drawn again into the lung, as in mammals, but passes to the air-sac or respiratory bellows. The true lung expands towards the end of the inspiratory movement when the purer air has reached it; the air spaces in the lung are then filled with pure air. In expiration, both sac and lung are partially emptied, the air passages being at the end of the act filled by the foul air. At the beginning of the next inspiration, that foul air left in the main passages is drawn into the non-respiratory reservoir, thus allowing the pure air to reach the lung.

Now it may be taken as an axiom that there is never any retrogression in the normal process of evolution; that the mammalian lung should be less effective in its mechanism of respiration than that of the bird or reptile is most improbable Think for half-a-minute what is taught concerning human respiration. An ordinary breath, it will be remembered, is about 250 cc.; the nose, trachea, bronchial passages, right to the ends of the bronchioles, have a collective capacity of about 160-170 cc. Now at the commencement of an inspiration these passages are filled with 170 cc. of the foulest air; yet we teach that this is drawn back into the infundibula of the lung, plus about 80 cc. (or perhaps more by diffusion) of pure air. Then with expiration the purer air in the air passages is expelled with an addition of about 80 cc. of impure air from the infundibula. It will be seen that matters are managed better in the bird.

But on the other hand, when one remembers the surface layers of the lung are less resistant to expansion than the deeper, and that the infundibula are larger in the anterior parts of the lung, it will be seen that these parts of the lung will expand first and most on inspiration, and will draw most of the foul air to these parts. Roughly speaking, I regard the surface layers of the lung, especially of that part of the lung which lies in front of a line drawn from the mid-clavicular line to the tip of the ninth costal cartilage, to represent the air reservoirs of lower vertebrates, and to be much less respiratory in function than the deeper and posterior parts of the lung.

That is to say, the usual form of emphysema is a disease of the respiratory-bellows part of the lung. The anterior part of the lung offers certain peculiarities which indicate that it is different in function from the posterior part. (1) The inspiratory sounds are louder, and yet the chest wall is not thicker; (2) an expiratory sound is heard—at the commencement of expiration; (3) it yields a lighter percussion note, because it contains more air than the posterior part—especially on inspiration; (4) it is less vascular, because its negative pressure is less.

*Conclusion.* To sum up the theory on which I am proceeding, is the following:—that there is a relationship between the mechanism of respiration and the incidence of disease on the various parts of the lung: that the anterior part of the lung, which includes the whole of the middle lobe of the right side and the anterior parts of the upper and lower, is peculiar in function and structure, and is therefore liable to emphysema; that the apical part of the lung is the least active part, and is therefore most liable to phthisis; that the basal part is the most active—receives the greatest amount of respiratory air, and is therefore the most liable to be infected by air borne germs such as the pneumococcus.