INTRA-PLEURAL TENSION

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Received December 20th, 1897—Read May 10th, 1898

UNDER normal conditions the lungs within the chest are in a constant state of tension. They are on the stretch and tend to contract by virtue of their own elasticity. Thus the two layers of the pleura, which in health are in close contact, are subject to a constant strain which tends to pull them apart.

This is called the intra-pleural tension. It is for all practical purposes equal in amount to the elastic contractility of the lung, but opposite in direction, and thus the elastic tension of the lung is positive but the pleural tension negative.

Whether, under normal conditions, where the two layers of the pleura are in close apposition, there is a force existing between them (viz. that of cohesion, as I suggested some years ago) which balances the elastic contractility of the lung or not, is a question which admits of discussion. *A priori* it is not improbable, and I have shown by experiment that it is possible. However

this may be, as soon as the two layers of the pleura are separated, cohesion, if it existed as an active force when they were in close apposition, becomes eliminated and the lungs tend to collapse.

The factors which combine to produce the intra-pleural tension are, first and foremost, the elasticity of the lungs; secondly, the resistance of the chest walls.

These two factors, though varying considerably no doubt in different individuals, or even in the same individual under different conditions, are constant in any given individual under the ordinary conditions, and are much the same after death as during life.

Besides these there is a third factor, but it is one which is constantly varying, viz. the movements of respiration; for it is evident that on deep inspiration the lungs are more on the stretch and the pleural tension therefore greater, while on expiration the lungs are less on the stretch and the intra-pleural tension therefore smaller.

If the air were stationary in the air tubes, as it is after death, the pressure in them would be that of the atmosphere; but the movements of the air in and out during inspiration and expiration respectively, cause slight variations in pressure. Thus Landois and Stirling state that during inspiration the pressure in the lungs falls by a millimetre of mercury, *i.e.* half an inch of water, and during expiration rises by 1, 2 or 3 millimetres of mercury, *i.e.* $\frac{1}{2}$ to $1\frac{1}{2}$ inches of water. Thus the respiratory oscillation during ordinary respiration would be from $1\frac{1}{2}$ to 2 inches of water.

The condition of the tubes, *i.e.* whether there be obstruction in them or not, is very important to bear in mind in determining either the elasticity of the lung or the pressure within the pleura; for wherever there is any obstruction in them the elasticity of the lung will be *pro tanto* prevented from coming into play and the true results be not obtained.

If the forces of respiration be eliminated, as after death, then under the normal conditions the intra-pleural tension must be a negative quality, *i. e.* less than the atmospheric pressure, and less by as much as is represented by the contractility of the lungs.

During respiration also, so long as the breathing is quiet, *i. e.* not forced or violent, the intra-pleural tension must still be, both on inspiration and expiration, a negative quantity ; for, assuming the value of the elasticity of the lung to be that which Donders estimated, viz. 6 to 7 millimetres of mercury, and that the respiratory oscillations are, as stated above, from 1 to 2 or 3 millimetres of mercury, there remain still 3 or 4 millimetres of mercury below which the tension will not fall.

Where the respirations are forced, that is to say where inspiration is deep or expiration excessive, the conditions may possibly vary considerably. With deep inspiration it must, however, always be negative, and the larger in amount the deeper the inspiration is.

With forced expiration, as in straining, coughing or vomiting, the pressure may be raised even to the extent of 3 or 4 inches of mercury (*i. e.* 75 to 100 millimetres), but as long as the two layers of the pleura are in close contact with the chest wall this excessive pressure is supported by the chest walls and does not fall directly on the pleura. On the other hand, under pathological conditions, where the two layers of the pleura are separated, any changes of pressure of the air within the lung are at once transmitted to the contents of the pleura ; but the effect varies greatly, according as the contents of the pleura are practically incompressible as in the case of fluid, or easily compressible as in the case of air.

The methods by which the intra-pleural tension has been estimated in health are two :

1. By means of a trocar introduced into the pleura through the chest walls.
2. By means of a tube fastened into the trachea, the

pleural cavity being then opened by an incision through the chest walls, first on the one side and then on the other.

In either case the reading is made upon a mercury or water manometer connected with the trocar or the tracheal tube respectively.

In the second method, as the elastic tension in the lung is equal in amount to the tension in the pleura, but opposite in direction, the sign must be changed. Thus the positive readings showing the tension of the lung become negative readings in respect of the tension in the pleura.

For man both these methods of investigation are available after death, but the former only during life or under pathological conditions.

Intra-pleural tension is sometimes spoken of as intra-pleural pressure, and under pathological conditions the pressure is sometimes positive and sometimes negative. A certain amount of confusion is thus introduced both in thought and in expression.

This confusion will be removed if it be remembered that the records are expressed in manometer readings; thus, if the column of liquid in the manometer tube on connecting it with the pleura remains stationary, the reading will be zero, although in reality, of course, the actual pressure within the pleura is that of the atmosphere, and being equal to that within the air tubes might be called one; but as this is the zero point upon the manometer, it is more conveniently called "zero."

One, two, three, &c., millimetres of mercury or inches of water mean one, two, or three above or below the atmospheric pressure, as the case may be, and would be indicated by its appropriate sign of "plus" or "minus."

The readings are given in different investigations, sometimes in millimetres of mercury, sometimes in millimetres or inches of water. It is more convenient to use water, because the variations are greater, and therefore more obvious, and in the experiments which I have to

record I have measured it in inches ; but the conversion is easily made from one scale to the other ; thus one millimetre of mercury is approximately equal to half an inch or $12\frac{1}{2}$ millimetres of water, and one inch of water is equal to 25 millimetres of water.

The intra-pleural tension as estimated by Donders was equal to 7 or 8 millimetres of mercury, and this is the value usually given in text-books.

Perl's experiments, conducted according to the second method, give a somewhat lower value, about 6 millimetres of mercury ('*Deutsches Archiv f. klin. Med.*,' 1869, vi, 1—5).

As Perl's experiments are the most interesting and important from our point of view, it seems to me desirable to give a short account of the results obtained by him, but in doing this I shall add various comments of my own.

Perl's experiments were conducted with a view to determine the pressure relations within the thorax in different diseases.

They were conducted in the following way :

A tube was fixed air-tight into the trachea, connected with a manometer ; the right pleura was then laid freely open and the pressure read ; after that the left pleura was laid open, and the pressure read again.

The observations were made upon the dead body of man.

1. Where the lungs are healthy the opening of one side of the chest goes a long way to satisfy the elasticity of both lungs, as the following figures show :

Pressure on opening the right side	.	29	...	49	...	39	...	32
" " " left "	.	11	...	5	...	9	...	7
Total pressure (right + left)	.	40		54		48		39

This is very interesting, as it explains what we often observe in pneumothorax, viz. that the symptoms are the more severe the more healthy the patient has hitherto

been ; for if we take the total elasticity in the adult man, according to these experiments, to be on the average 50, the opening of the right side diminishes this elasticity by 40, leaving only 10 for the other ; and therefore the respiratory capacity of such an individual would be reduced by the opening of one side to the extent of four fifths—a difference which is not met with to the same amount where the lung is diseased.

2. Where the pleura is adherent the lungs are held back, and the recoil thus prevented, more or less, and sometimes entirely.

If the pleura be universally adherent, the contractility of that lung (or of both, if both be affected in the same way) will be entirely abolished ; for even if the elasticity be retained (and it seems to be retained sometimes), it will be prevented from acting. The degree to which the elasticity of the lung will be impaired depends upon the extent of the adhesions, and upon the secondary changes which they have produced in the lung tissue itself.

Where the pleura is partially adherent the opening of the remaining portion of the pleura on the one side does not produce the same marked effect upon the total elasticity of the lung as in the case of healthy persons.

If one pleural cavity be completely obliterated, the adhesions being universal, and the side be greatly contracted, the elasticity of the lung on the opposite side may be greatly reduced ; but, on the other hand, if the lung has undergone compensatory development, the elasticity may not only be not reduced, but may be actually increased. This is a very interesting and important observation, because it shows that the condition which is often described as “compensatory emphysema” is really a compensatory hypertrophy, as indeed there are the best clinical grounds for believing.

Thus, in a case of the kind, on the right side, where the pleura was completely adherent, the elasticity of that lung was reduced to 12 ; whilst on the opposite side (the left), where the pleura was free, although there was some

slight chronic tubercular change in the apex, the elasticity of the lung was 57.

The contractility of the lung is in all probability not purely elastic, that is to say it does not depend solely upon the elastic tissues in the lung, but probably also upon the muscular tissue with which the lung is so richly provided. Indeed, we may in this respect compare the lungs with the arteries and speak of "pulmonary tone" as we do of "arterial tone," and connect the tone in the lungs, as we do in the arteries, with the condition of the muscles, and possibly of the nerves.

If there be such a thing, therefore, as pulmonary tone, we might expect it to vary, as the tone of the vessels does, with the conditions of general health or of local disease, and there are some among Perl's observations which go far to prove this contention. Thus, without any local disease, the elasticity of the lungs was found greatly reduced in several cases of typhoid, in a case of severe hæmorrhage after ovariectomy, and in cases of delirium tremens, erysipelas, and phosphorus poisoning.

The condition would then be brought into close clinical association with that of acute tympanites, and like it would be a neuro-paralytic phenomenon, and of grave significance. For my own part, I believe there are good clinical grounds for believing that there is a neuro-paralytic condition of the lungs, strictly analogous with that condition of the abdomen called tympanites.

The two conditions might, of course, theoretically be associated, but when the abdomen is distended from any cause, whether it be from gas, effusion, or tumour, the displacement of the diaphragm upwards is so great that the tympanitic percussion obtained over the lungs may be due to mechanical rather than physiological or pathological causes.

Where the lungs are diseased, the other non-diseased parts may yield tympanitic resonance on percussion. This is a condition with which we have been long familiar

under the name of Skodaic resonance. There are several conditions under which this phenomenon appears. The commonest and easiest to explain is that in which it is associated with an effusion into the pleura. In this case, as the fluid forms the lung collapses and floats on the surface of the fluid, and over this collapsed lung, tympanitic percussion note is obtained. Its occurrence has been rightly referred to the relaxation of the lung tissue, and properly compared with the percussion note which is obtained over the lung removed from the body.

Again, conditions of complementary emphysema, where one part of the lung is collapsed and the other proportionately distended, may give rise to similar hyper-resonance.

But besides these there is, I believe, a third condition, and this requires a different explanation.

Nothing is much commoner with pneumonia than to find the parts of the lung above or in front of the affected portion yielding a highly tympanitic note, yet the pneumonic portions of the lung are certainly not collapsed or smaller than they should be, nor are they much larger; so neither of the explanations given is adequate, for where the hyper-resonance is obtained the lung is not collapsed on the one hand nor over-distended on the other. This condition, it appears to me, can only be explained on the assumption of a loss of lung-tonus, of neuro-paralytic origin, dependent on nutritive disturbance. This view is supported by some of Perl's observations; for example, there are several cases of pneumonia and some of embolism and gangrene, and in all of them the elasticity of the lung was very greatly reduced.

Acute miliary tuberculosis provides perhaps a still more striking instance of this condition, for the hyper-resonant or tympanitic note on percussion may be almost the only physical sign of disease yielded by the lung, and there can be little risk of error in assuming that the nutrition of the lung is profoundly affected in this disease.

In acute bronchitis, again, both in the child and in the adult, this nutritive disturbance in the lung probably explains in great part the tympanitic percussion obtained; but proof is more difficult in these cases, because of the other conditions which may take their share in producing it, viz. the collapse of some parts of the lungs and the complementary emphysema of others.

In estimating the intra-pleural tension and the elasticity of the lung the condition of the abdomen cannot be disregarded. During life there is the respiratory action of the abdominal muscles to reckon with, and after death we have to consider the effects of rigor mortis in them.

It is possible that the elasticity of the lung diminishes after death, but there are no direct observations which prove this, though it may be that this is the explanation of the difference as between Perl and Donders in their estimate of the average value of the elasticity of the lung.

Under pathological conditions the two layers of the pleura may be separated either by air or by fluid, and these present each of them its own peculiarities; thus fluid has weight, but it is practically incompressible; while air is compressible, but its weight may be disregarded. With fluid, therefore, the height of the column of fluid above the point of the trocar would effect the manometer reading, while with air the position of the puncture is indifferent.

With air the difficulties of the problem are in many respects less, and it will be well to begin with that condition first.

Intra-pleural Pressure in Pneumothorax.

The air may have gained access to the pleura either from without through the chest walls, or from within from the lung or some other air-containing viscus.

I. Where there is an opening through the chest walls.

(a) A punctured wound, such as is made by a stab with a penknife where the lung is injured as well as the external walls, we need not consider, for in this case, though the lung is wounded, air does not make its way in most cases into the pleura, but crosses the pleura and reaches the subcutaneous tissue. The explanation of this is very difficult, but need not be considered here.

(b) Where the opening through the chest walls is a large one, say at least as large as the section of the trachea, the air will pass freely in and out during respiration without let or hindrance as easily as it does through the trachea. In this case the pressure on the two sides of the lung, viz. that in the trachea and that in the pleura, will be equal always throughout all phases of respiration. Then the elasticity of the lung is free to come into play, and the lung collapses.

No doubt, as Donders said, the elasticity of the lung itself is able in time to produce complete collapse, but that this is not the result during life, daily observation demonstrates in cases in which the pleura is laid open by operation. In many of these cases where the opening is free, the lung, instead of being found completely collapsed, as might be expected, is on the contrary found distended to a certain point, and reaching, it may be, within an inch or so of the walls of the thorax; or if it has been collapsed, as for instance by an empyema, as soon as this has been evacuated the lung often rapidly expands so as to occupy the position just indicated.

Although it is true, when the respiratory movements are absent, as they are in the dead body, that the atmospheric pressure being equal on the two sides the elasticity of the lung will lead to its complete collapse, this is not true while the respiratory movements are acting, for the air does experience some obstruction in the air tubes, both on entrance and on exit; thus the pressure of the air in the air tubes during inspiration is about half a millimetre of mercury ($\frac{1}{4}$ inch of water) below that of

the atmosphere ; while on expiration it is as much as 2 to 3 millimetres of mercury (1 to $1\frac{1}{2}$ inches of water) above it, and these differences are still greater if the respirations are violent and forced.

Under these circumstances it is clear that the lungs would only collapse until the pressure within them was equal to 2 or 3 millimetres of mercury, and that they would then cease to contract any more. If the lungs can be completely expanded under a pressure of 6 millimetres of mercury they could certainly be not less than half expanded, at any rate, under a pressure of 3 millimetres. What the exact relative volume of the lung might be actually under a pressure of 3 millimetres of mercury there are no observations to show.

When the expirations are forced, as in coughing, straining, or vomiting, the expiratory pressure becomes much higher than 3 millimetres of mercury, and the lung may then, if the external opening be free, be forced out again into actual contact with the chest walls or even made to protrude through the opening in the side.

Actual protrusion of the lung during coughing in these circumstances, however, rarely occurs, but the movements of the lung may be seen to be very free.

(c) Where the external opening is not large enough to allow the air to pass freely out during expiration, the excess of air which cannot escape is compressed, and the pressure rises above that of the atmosphere ; the lung is in consequence squeezed, and is more and more compressed until it becomes completely collapsed. The condition is then much the same as that in which the air has gained access to the pleura from the lung.

II. Where the opening into the pleura is from within.

Theoretically there might be a condition in which the opening into the lung was large enough to permit the air to pass freely in and out on respiration without obstruction. This, however, as a matter of fact, hardly ever occurs. Air practically always enters the pleural cavity

more easily on inspiration than it finds exit from it during expiration.

The result is, as in the former case, that the lung becomes compressed during expiration, and the more rapidly so the greater the difference between the pressures of inspiration and expiration.

The division of pneumothorax into open, closed, and valvular thus becomes of little value from the present point of view, for all pneumothorax is at first more or less valvular, *i. e.* the air enters more easily during inspiration than it finds exit during expiration, and when the lung is completely collapsed, the pneumothorax practically becomes closed, whether the aperture be sealed permanently or not.

In pneumothorax the pressure conditions during inspiration and expiration require to be considered separately.

1. *The inspiratory pressure.*—When pneumothorax has occurred air will continue to enter on inspiration until the pressure within the pleura is equal to that of the atmosphere; as soon as this point is reached no more air can enter; consequently with a simple pneumothorax the inspiratory pressure cannot exceed that of the atmosphere, *i. e.* the reading on the manometer will be zero.

This is subject to one condition. In the early stages of pneumothorax the respirations are excessive and air will enter the pleura until the pleural pressure is equal to that of the atmosphere on deep inspiration. When after a time the urgent dyspnoea has passed off, so that inspiration is not so deep, the pressure may be above that of the atmosphere, to the extent of the difference between deep inspiration and ordinary inspiration; probably to a great extent this relieves itself in time by the absorption of a sufficient amount of the air to remove the difference.

If the inspiratory pressure is permanently above that of the atmosphere, that is to say is positive, there is required some other factor to cause the rise, and this, as a matter

of fact, we find to be the development of effusion, so that, speaking generally, a positive pressure in pneumothorax during inspiration indicates the presence of fluid.

2. *The expiratory pressure.*—Except in the theoretical and rare conditions in which there is no obstruction to the exit of air from the pleural cavity during expiration, the expiratory pressure in pneumothorax will always be positive in the early stages.

In these cases the heart and mediastinum are found displaced to their maximum and the lung completely collapsed.

The collapse of the lung, though capable of being brought about in time by its own elasticity, is always in chief part due to the compression exercised by the rise of expiratory pressure.

In the same way the displacement of the mediastinum, though capable of being brought about by the elastic traction of the opposite lung alone, is assisted by the raised expiratory pressure and caused to develop more rapidly or to be more extreme.

The respiratory oscillation.—The respiratory oscillation is the difference between the pressure on inspiration and the pressure on expiration, and this presents also some interesting points for consideration.

If the movements were violent the respiratory oscillations would, it might be thought, be considerable; but as a rule this is not so, for on the affected side the chest is in the position of maximum inspiratory distension, so that no further expansion on inspiration is possible; while on the opposite side, owing to the displacement of the heart and mediastinum, the elasticity of the sound lung is reduced, so that the respiratory excursion is diminished, the result being that even with considerable dyspnoea the respiratory oscillation is not only not raised above the normal but may be considerably below it. It is, indeed, upon the defective respiratory excursion of the two sides that much of the dyspnoea depends.

Yet there are cases in which extensive respiratory

oscillations are seen, and it is important to consider the interpretation which may be given of them.

The number of cases of pneumothorax in which I have estimated the pressure in the pleura is eleven. In several of these paracentesis was performed more than once and the pressure determined, so that I have returns of the pressure in twenty paracenteses. In most of them I am able to record the inspiratory pressure, the expiratory pressure, and the difference between them, *i. e.* the respiratory oscillation.

In two cases the inspiratory pressure was that of the atmosphere, *i. e.* the manometer reading stood at zero at the time of the operation. In both these cases fluid was present as well as air, and from this it is evident that as the fluid formed the air must have been absorbed, since the opening in the lung in both cases was closed.

In another case the inspiratory pressure, after having been in the first two paracenteses positive, in the last two fell to zero, and in this case the change in pressure was due to the opening in the lung having become patent and being of considerable size.

In all the other cases the inspiratory pressure was positive, and fluid was present as well as air,—sometimes pus, sometimes serum. Although, as I have already stated, the inspiratory pressure in the early stages of pneumothorax may be positive to some extent, it can never be high, and practically we may conclude that when the inspiratory pressure is much raised, fluid is present as well as air in the pleura. This was at any rate the fact in the instances recorded.

The range of pressures in the different cases is considerable—thus the inspiratory pressure varied from 0 to 9 inches, the actual pressures being $0\frac{1}{2}$, 1, $1\frac{1}{2}$, $2\frac{1}{2}$, 4, $4\frac{1}{2}$, 5, $6\frac{1}{2}$, $6\frac{3}{4}$, $6\frac{7}{8}$, 7, $8\frac{1}{4}$, 9; the expiratory pressure varied from 0 to $13\frac{1}{4}$, the respiratory oscillation from 0 to $6\frac{5}{8}$.

The amount of fluid removed is no indication of the amount of fluid present, for the effusion lies in the

diaphragm as it were in a saucer, and unless the point of the trocar be carefully manipulated so as to be beneath the level of the fluid, air will escape, the pressure fall, and no fluid be removed unless suction be used.

Suction, and especially the use of the aspirator, is in all cases of pneumothorax risky, for where the hole is patent it is useless except for the purposes of removing fluid, and then the trocar must be carefully manipulated; and if the opening be closed, suction is very likely to tear it open afresh.

The aspirator, therefore, is a dangerous instrument in pneumothorax, and should be rarely employed.

The ordinary syphonage apparatus, if properly manipulated, is all that is required and is devoid of risk.

It is not to be wondered at that the inspiratory pressure rises when fluid forms in pneumothorax, but it is surprising that the pressure is not much higher than it is. The highest pressure that I have to record is seven inches of water; and a pressure as high, or even higher, is met with in simple serous effusions.

It follows, therefore, that when fluid forms in pneumothorax a large amount of the air present must be absorbed as the fluid forms.

That the absorption of air from the pleural cavity is very rapid we know from experiments on animals and also from observations made in cases of accident and operation in man.

There are cases of pneumothorax even in man, in which, in spite of the presence of fluid, the inspiratory pressure is not above that of the atmosphere, as in the two cases mentioned.

There are two conditions under which the pressure might rise rapidly in an ordinary pneumothorax:

(a) Where the effusion is poured out rapidly; the fluid must then be serous in character, so that time enough has not been given for the air to be sufficiently absorbed.

(b) Where the lung or pleura is so far affected that

the absorption of air does not take place as readily as it should, and this no doubt is the fact in most cases of pneumothorax, and I think we shall not be going too far if in cases of pneumothorax of long duration we accept as an indication of somewhat extensive disease of both lung and pleura, the existence of a high intra-pleural pressure.

In two cases the pressure was determined at the end of the paracentesis as well as at the commencement of it. In the one case no suction was employed and the pressure fell to that of the atmosphere, the manometer registering zero. In another case some suction was used, and at the end of the operation the pressure registered — 9 inches of water. In another case the pressure, which on the first two paracenteses had been positive, in the third and fourth proved to be zero. The aspirator was then used, nothing but air was removed, and as this had no effect upon the pressure it was evident that the air passed freely into the pleura from the lung; in other words, that the opening was patent. This proved to be the case on the autopsy.

Further comment will be most usefully made in connection with the description of each case.

No. of case.	Sex.	Age.	Side.	I. Pr.	E. Pr.	R. O.	Amount of fluid removed.	Remarks.
1	M.	31	R.	0	0	0	oz. 80, turbid serum	11 weeks' duration; ultimately recovered.
2	M.	31	L.	0	1½	1½	11, pus	3 weeks' duration; marked phthisis.
				0	8	8	10	Great dyspnoea; 2nd paracentesis 7 weeks after the first; ultimate incision; necrosis of 6 inches of rib; death from exhaustion.
3	M.	30	L.	1½	2½	1	Pus	3 weeks' duration; at end of operation, Pr. = — 9; ultimately side discharged; incision; death.

No. of case.	Sex.	Age.	Side.	I. Pr.	E. Pr.	R. O.	Amount of fluid removed.	Remarks.
4	M.	24	L.	8½ — 7	— — 8½	— — 1½	oz. 34, pus 41, pus Much	3 weeks' duration. 14 days later. 3 days later; free opening formed in lung; incision subsequently; death. 3 months' duration.
5	M.	22	R.	6½ 6½	13½ 8	6½ 1½	41, sero-pus 22, sero-pus	3 weeks later; ultimate recovery.
6	M.	21	R.	1 ½	5 2½	4 1½	28, sero-pus 36	5 weeks' duration. 17 days later; subsequent incision; death from hæmoptysis. P.M. Ruptured pulmonary aneurysm.
7	M.	24	L.	4	5	1	22, serum	3 days' duration; recovery.
8	M.	35	L.	6½	7	½	6, pus	Many months' duration. At end of operation I. Pr. = 0, R.O. = 2. Was pointing at time; therefore pointing pyopneumothorax may have low or even zero pressure. Interesting case to compare with empyema.
9	F.	23	L.	2½ 4½	9 5	6½ ½	Several, pus —	3 days' duration. Much dyspnoea at time of operation. At end of operation R.O. = 1, dyspnoea passed off. 3 days later, owing to little fluid being obtained, suction employed up to - 36 inches of water, but only 6 oz. more fluid obtained. ∴ Lung bound down. Incision. Death from shock immediately on incision. No p.m.
10	M.	46	—	9 1	9 4½	0 3½	74 Serum	10 days' duration. 16 days later, on deep Inspir. Pr. = - 1; on cough Exp. Pr. = 7. Ultimately puncture suppurated, though fluid was serous. Patient lived 12 months, and died of phthisis.
11	M.	22	L.	5 1 0 0	8½ 6 0 ½	3½ 5 0 ½	36, serum A few — 40, pus	A few days. 9 days later. Opening had become patent. On death large patent opening found in lung.

Intra-pleural Pressure in Pleural Effusion.

1. *Serous effusion*.—In health the pleural cavity contains no fluid, and we often speak of it as dry; yet this is inaccurate, for there is in all probability a constant circulation of fluid into the pleura and out of it, the fluid being effused by the blood-vessels and carried away by the lymphatics. The mechanism by which this is performed has been described as the lymphatic pump. It consists of the lymphatic vessels with their stomata and valves, and is worked by the respiratory movements. The course of the circulation in the lung is from the pleural surface (the periphery, that is to say), towards the root of the lung, as has been determined by experiment, and there is a similar circulation from the pleural surface through the diaphragm and through the chest walls.

There are two ways, therefore, in which fluid may accumulate in the pleura; either it may be poured out into the pleura in larger quantities than the pump can remove, or the amount of fluid may not be above the normal but the pump be defective.

In the case of pleural inflammation both these processes probably come into play; the amount of transudation is considerable, and the stomata and smaller lymphatics are often plugged by a deposit of fibrin. Thus in inflammatory cases the fluid may accumulate with very great rapidity and soon reach a large amount.

In a case of dropsy of the pleura consequent, for instance, on heart disease, the explanation is probably also mechanical. Exudation under these conditions takes place from the blood-vessels into the lymphatics of the lung, which become water-logged or choked, and thus unable to carry off the fluid from the pleural cavity, so that it accumulates in it.

With dropsy, however, the development of fluid is much slower and the amount as a rule much less.

When fluid collects in the pleura it falls by its weight

to the lowest part, and although the tension in the whole pleural cavity will be diminished in proportion to the amount of fluid present, still the effect upon the different parts of the lung will be different; thus the lowest parts will suffer most and become collapsed as we know they do, while the upper parts of the lung remain distended; but the tension in the upper part of the pleural cavity is lower than it otherwise would be, as is shown by Calvert's observations ('St. Barthol. Hosp. Rep.,' 1892, p. 131).

This diminished tone in the lung or tension in the pleura explains the hyper-resonant note which is obtained in those parts of the lung which are floating upon the fluid.

In determining the intra-pleural pressure in cases of fluid effusion something will depend upon the seat of puncture, as Calvert also has shown, for if the mouth of the trocar be 1, 2, or 3 inches respectively below the level of the fluid, there will be the pressure of a column of fluid of this height to allow for.

If, for example, the intra-pleural tension be equivalent to 3 inches of water, and the amount of fluid exuded into the pleura be sufficient to reduce this 3 inches negative pressure to 2 inches negative pressure, it follows that if the mouth of the trocar be 2 inches below the level of the fluid, a positive pressure of 2 inches will have to be added to the negative pressure in the rest of the pleura, which will reduce the pressure-reading to zero; or if the height of the fluid be 3 inches instead of 2, would convert the pressure at the point of puncture to a positive pressure of 1 inch.

In most of my cases I have taken as far as possible the same relative position in the chest for puncture, viz. the middle of the axilla, as the patient is lying upon the back; the results are therefore more or less comparable with one another; but although I have endeavoured to make some allowance for these considerations, the readings in pleural effusions have not anything like the same value as those in pneumothorax.

I have determined the pressure in twenty-seven cases of serous effusion, some of which were tapped more than once, so that I have thirty-one pressure-records to found these observations upon.

In the table which follows I have arranged the cases in order according to the size of the effusion.

No. of case.	Sex.	Age.	Side.	Amount of fluid removed.	Pressure.	Resp. osc.	Remarks.
1	M.	46	R.	oz. 139	2½	0	At end of operation Pr. = -9½; resp. osc. = ½; spontaneous coagulation in fluid.
2	M.	41	—	130	18	$\frac{4}{10}$	
3	M.	32	L.	112 After 6 days 70 " " 72	-1 -1 +3½	3 1 1	
4	M.	39	L.	106	8	1½	After paracentesis fluid rapidly disappeared; Pr. after first paracentesis = -1.
				19	5	1½	
5	M.	35	L.	98	4	1	Fluid apparently formed in 5 days. Blood-stained fluid.
6	M.	39	—	85	6	½	
7	M.	51	R.	77	5	2	
8	Cf. Case	3		72	3½	1	
9	M.	42	L.	72	1½	1	
10	Cf. Case	3		70	-1	1	
11	M.	22	L.	68	8½	—	On second paracentesis Pr. = 0; resp. osc. = 8 (from +4 to -4); a few ounces of pus. Died shortly after of acute phthisis.
12	M.	33	R.	62	0	1½	
13	M.	54	L.	60	0	0	
14	M.	63	R.	55	4	2	Sp. gr. of fluid 1040; spontaneous coagulation.
15	F.	25	R.	52	8½	1	
16	M.	34	R.	50	2½	—	
17	F.	29	L.	47	2	¾	Fluid blood-stained. 100 ounces removed previously, but pressure not taken.
18	M.	83	L.	45	11	1½	
19	F.	46	L.	46	11	—	
20	M.	23	L.	40	0	¾	
				20	3	¾	

No. of case.	Sex.	Age.	Side.	Amount of fluid removed.	Pres- sure.	Resp. osc.	Remarks.
21	F.	—	—	oz. 40	1½	¾	Sudden death. Pleurisy secondary to sarcoma of vertebræ.
22	—	—	—	34	0	2	
23	F.	53	L.	30	½	½	
24	Cf.	Case 19		20	3	¾	
25	Cf.	Case 4		19	5	1½	
				Amount not specified.			
26	F.	34	—	Very large	1	¾	At end of operation Pr. = + 3. Cast expectoration subsequently from trachea. Death.
27	M.	26	L.	Large	8	—	
28	M.	—	L.	Moderate	0	2	
29	M.	62	L.	Large	11½	—	
30	M.	46	L.	Small	0	—	
31	F.	19	—	Moderate	-4	½	

A glance at this table will show that the pressures vary greatly and irregularly, and that there is no definite relation between the size of the effusion and the amount of pressure; for there are among the cases instances of large effusions with low or even negative pressures, and of small effusions with high pressures.

Thus a **large** effusion was found:

With *negative* pressure in—

Case.	Amount.	Pressure.
3 ...	112	— 1
A few days later	70	— 1

With *low* pressure—

26 ...	large	1
1 ...	139	2½

With *high* pressure—

2 ...	130	18
4 ...	106	8
5 ...	98	4
6 ...	85	6
27 ...	large	8
29 ...	large	11½

In the same way with **moderate** effusions the pressure may be negative, zero, moderate, or high.

With *negative* pressure—

Case.		Amount.		Pressure.
10	...	70	...	- 1

With *zero* pressure—

12	...	62	...	0
13	...	60	...	0
28	...	moderate	...	0

With *high* pressure—

7	...	77	...	5
11	...	68	...	8½
15	...	52	...	8½
14	...	55	...	4

Small effusions in the same way :

With *zero* pressure—

20	...	40	...	0
22	...	34	...	0

With *low* pressure—

23	...	30	...	½
21	...	40	...	1¼

With *high* pressure—

18	...	45	...	11
19	...	46	...	11
24	...	20	...	3
25	...	19	...	5

If we look at these cases again from the point of view of pressure, we find :

A **negative** pressure—

With *large* effusion in—

3	...	112	...	- 1
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With *moderate* effusions in—

10	...	70	...	- 1
31	...	moderate	...	- 4

A zero pressure—With *moderate* effusions in—

Case.		Amount.		Pressure.
12	...	62	...	0
13	...	60	...	0
28	...	moderate	...	0
30	...	small	...	0

With *small* effusions in—

20	...	40	...	0
22	...	34	...	0

Very high pressures—With *large* effusion in—

2	...	130	...	18
29	...	large	...	11½
4	...	106	...	8

With *moderate* effusions in—

11	...	68	...	8½
15	...	52	...	8½

With *small* effusions in—

18	...	45	...	11
19	...	46	...	11

It might be thought that this irregularity could be explained by the different stages in which the inflammatory effusions were at the time of investigation; for instance, that where the inflammation was active the pressure might be high; where the inflammation was subsiding the pressure might be low; and that when the fluid was being absorbed the pressure might even become negative.

Case 6 might, perhaps, bear this interpretation, for the fluid in this case was accumulating with very great rapidity, and within four or five days from the very commencement of the illness 85 ounces of fluid were removed from the pleura; the pressure in that case was + 6; but this theory is not supported by many other cases, and I am afraid the whole matter is by no means so simple as this would make it.

Two cases present a special interest because the para-

centesis was repeated, and the pressure determined on subsequent occasions. Case 3 is an instance of the kind. On the first paracentesis 112 ounces were removed, and the pressure was -1 . Six days later 70 ounces more were removed, and the pressure was -1 again. Six days later still 72 ounces were removed, and then the pressure was $+3\frac{1}{2}$.

Case 19 is another instance of the kind. On the first paracentesis 100 ounces were removed, and the pressure was not determined. On the second 40 ounces were removed, and the pressure was zero. On the third 20 ounces were removed, and the pressure was $+3$.

It is tempting to suppose that by the third paracentesis in each of these cases the fluid had become encapsulated, the pleura being adherent elsewhere, and that this might account for the curiously sudden rise in pressure which occurred in both cases.

In three cases the pressure was determined at the end of the operation as well as at the commencement of it. In Case 1, where 139 ounces were removed, the pressure at first was $2\frac{1}{2}$, while at the end of the operation it was $-9\frac{1}{2}$. In Case 4 the pressure at the commencement of the operation was 8, and at the end of the operation -1 ; and in Case 29, where the pressure was $11\frac{1}{2}$ at the commencement, it still remained $+3$ at the end.

From this analysis of these observations I think it is clear that the intra-pleural pressures in serous effusions are by no means so simple as they might be thought to be, and that the whole subject requires a great deal of further investigation.

The Respiratory Oscillation.

The respiratory oscillation is, as already stated, in health about 1 to $1\frac{1}{2}$ inches of water, and in fifteen of the cases recorded the respiratory oscillation was of about this amount.

It was reduced to zero, that is there was no respiratory oscillation at all in three cases: Case 1 with 139 ounces of

fluid; Case 12 with 62 ounces of fluid; and Case 13 with 60 ounces of fluid.

It was also below 1 inch in nine cases. On the other hand, it was increased only in one, viz. Case 3, in which with 112 ounces of fluid and a negative pressure, - 1, there was a respiratory oscillation of 3 inches.

The respiratory oscillation, therefore, is generally reduced in serous effusions, and may be completely absent.

Inasmuch as the action of the lymphatic pump depends upon the respiratory movements of which the respiratory oscillation is the index, it is evident that where the respiratory oscillation is absent the mechanism of the removal of fluid must be defective.

Where, with a large effusion at the time of introducing the trocar, the respiratory oscillations are seen to be absent, they frequently return after the fluid has been removed, and may even be above the normal. Even the partial removal of the fluid, *i. e.* the removal of a part of it and not the whole, may cause the respiratory oscillations to return. This explains what is frequently observed and is difficult otherwise to understand, viz. that the removal of a small quantity of fluid not infrequently leads to the rapid spontaneous disappearance of the rest; the interpretation is that the lymphatic pump has been started to work again.

Other points which these tables show are that pressures have no relation to age, for the cases range from the young to the old; nor to sex; nor to the side affected; nor is the pressure any help in prognosis, for nearly all the cases in this list recovered with the exception of three, one of which died of rapidly advancing phthisis, the second of cancer, and the third with sarcoma of the vertebræ.

These observations have a bearing also upon the mechanism of the displacement of the heart and mediastinum in pleuritic effusion, for they show that displacement does not necessarily depend in any way upon pressure, for the maximum displacement may be found with a

zero pressure or even with a negative pressure on the affected side.

We may conclude that the intra-pleural pressure in serous effusions is not as simple as it might *a priori* be thought to be; that it depends upon many factors, which are difficult to allow for, and that the whole subject requires further investigation.

Intra-pleural Pressure in Empyema.

I have ten observations of the pressure in empyema. They show that the question of intra-pleural pressure in empyema is much simpler than in the case of serous effusion, and completely in accord with what we should expect from our knowledge of suppuration in other parts.

We know that in other places the formation of pus is attended with considerable tension,—in other words, that it goes on under considerable pressure. It is only in the very chronic, so-called cold abscesses that the tension is low; but even then the pressure is probably considerably above that of the atmosphere.

No. of case.	Sex.	Age.	Amount removed.	Pres- sure.	Resp. osc.	Remarks.
1	M.	34	oz. 91 56	16 10	0 0	Subsequently expectorated pus. Developed pneumothorax and died with phthisis. After whooping-cough.
2	M.	5	6	4	0	
3	F.	19	2	3	$\frac{1}{2}$	
4	M.	28	14 $\frac{1}{2}$	5 $\frac{1}{2}$	—	On subsequent paracentesis 15 ounces removed twice; incision and recovery. On subsequent paracentesis 6 ounces and 1 ounce removed; cure by paracentesis only.
5	M.	29	20	3	1	
6	M.	6 $\frac{1}{2}$	40	8	$\frac{1}{2}$	
7	M.	26	15	8	0	On subsequent paracentesis 15 ounces removed twice; incision and recovery. On subsequent paracentesis 6 ounces and 1 ounce removed; cure by paracentesis only.
8	F.	13	15	5	$\frac{3}{4}$	
9	M.	35	6	7	$\frac{1}{4}$	

It will be seen that among my observations in all cases the pressure is considerably increased. The lowest of the series is 3, and the highest 16.

The highest pressure is found, as it happens, with the largest effusion, for in this case 91 ounces were removed with a pressure of 16, and on a subsequent paracentesis 56 ounces were removed with a pressure of 10.

There is, however, no necessary relation between the size of the effusion and the height of the pressure; for if small empyemata are localised, as they frequently are, the pressure may be very high, though the amount of pus contained in them be very small.

There is one interesting case which bears upon this among the observations in serous effusion, where a serous effusion of some size was tapped, but the pressure not raised.

On the third paracentesis the pressure was found to be +3, but the effusion then was no longer serous but purulent, the conclusion being that the serous effusion had been followed by a small local empyema. This was incised and the patient recovered.

The respiratory oscillation in empyema also is interesting; in all cases it was reduced, and in many of them absolutely disappeared. In four cases it was zero, in one $\frac{1}{4}$, in two $\frac{1}{2}$, and only in two instances did it approach 1.

The two points, therefore, in reference to the pressures in empyema are, first, that it is always positive, and sometimes considerably raised; and secondly, that the respiratory oscillation is practically absent.

(For report of the discussion on this paper, see 'Proceedings of the Royal Medical and Chirurgical Society,' Third Series, vol. x, p. 130.)