ON

SOME EFFECTS OF LUNG ELASTICITY

IN HEALTH AND DISEASE.

BY

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THERE is, perhaps, no department of clinical medicine which has from time to time been worked at with greater success, yet in which greater errors of a physiological kind still prevail, than the department of chest diseases. The classical paper of John Hutchinson communicated to this Society in 1846 remains unsurpassed in the interest and importance of the facts related ; his spirometer may still be found lurking in some consulting rooms, and its teachings will ever remain a part of our knowledge, although the instrument itself is replaced by other more convenient and discriminating methods of physical diagnosis.

Mr. Hutchinson's doctrine respecting the mechanism of respiration may still be said to be the current doctrine at the present time. He taught that ordinary inspiration was effected mainly by the muscular action of the diaphragm, that deep inspiration was effected, on the other hand, mainly by the expansion of the chest by mus-

cular action, and that in each case the forces to be contended against were the elasticity of the lung and the elasticity of the chest-walls. Twenty years later, however, in a most able series of lectures delivered at the College of Physicians, Dr. Hyde Salter made one important modification of Hutchinson's teaching. In these lectures upon the causes of dyspnoea,¹ Dr. Salter contended that the mechanical process of respiration might be considered as starting from a position of equilibrium at the end of ordinary inspiration, in which the thoracic parietes and diaphragm had followed the recoil of the lung to that point at which lung tension was equalled by thoracic resilience. At the commencement of inspiration, therefore, the thoracic elasticity was, Dr. Salter maintained, a force acting in favour of, and not against, expansion. As inspiration proceeded, however, thoracic resilience became an expiratory force acting *with* lung tension against further expansion, and, finally, these two elastic forces served, on muscular action ceasing, to effect expiration. I have copied the tabular form in which Salter placed his views with some slight alteration of arrangement.

Coefficients of Respiratory Statics.

At level of—	Forces tending to expiration.	Equilibrium	Forces tending to inspiration.
A. Ordinary expiration	{ Elasticity of lung Gravity of chest-wall	{	Elasticity of costal parietes (<i>ex-centric thoracic resilience</i>).
B. Ordinary inspiration	{ Elasticity of lung Elasticity of parietes Gravity of chest-wall		Muscles of ordinary inspiration, diaphragm principally.
C. Extreme inspiration	{ Elasticity of lung Physical reaction of parietes (<i>concentric thoracic resilience</i>) Gravitation of chest-wall		Muscles of extraordinary inspiration.
D. Extreme expiration	{ Elasticity of lung Gravity of chest-wall Muscles of extraordinary expiration		Elasticity of costal parietes, excentric thoracic resilience (<i>extreme</i>).

¹ Vide 'Lancet,' Aug. 5th, 1865.

The best modern physiologists now recognise the fact that in the normal position of thoracic repose the contractility of the lungs is exactly counterpoised by the elastic resilience of the chest-wall. [We may accurately name this elastic resilience of the chest-wall the *excentric thoracic resilience*.] Dr. Burdon Sanderson, in the 'Hand-book for the Physiological Laboratory,' p. 294, speaks thus of excentric thoracic resilience; he observes that "The muscular movements by which the chest is expanded must be studied in their relation to a certain definable position of the thorax which is called *the position of equilibrium*. It is the position assumed by it at the end of normal expiration. For as no muscle takes part in the normal expiratory act, the whole thoracic muscular apparatus is at that moment in a state of rest, the bones and cartilages assuming that position which results from the balance of the opposed elastic forces which act upon them from within and from without. Of these elastic forces the most important is that of the lungs, which organs, being contained in a cavity much larger than they are themselves, to the inner surface of which their external surface is inseparably applied, constantly draw together its walls with a force to be investigated in a future paragraph.

"Next in importance is the elasticity of the ribs and cartilages, by virtue of which the thoracic wall ever tends to be larger than it is, in opposition to the contractile influence of the lungs." That the thoracic parietes are drawn inwards by the elastic recoil of the lungs would scarcely seem to need demonstration; it is demonstrable, however, by an experiment of Dr. Salter's.¹ He took a capillary tube expanded at its lower end into a bell shape and closed at this extremity by a thin layer of caoutchouc. Filling the bell-end with a coloured fluid so as to include a few inches of the tube, he fixed the instrument vertically, so that the caoutchouc touched the cartilage of the third rib of a man recently dead. The integuments had pre-

¹ Loc. cit., p. 143.

viously been reflected from the chest. He now cautiously divided the cartilage, but no movement of the fluid took place. On penetrating further, however, into the pleural cavity air was heard to enter the chest, and the fluid mounted the tube to the height of one additional inch. This movement Dr. Salter estimated to be equivalent to an expansion of $\frac{1}{100}$ of an inch.

In May, 1871, Mr. Le Gros Clark related, in a paper read before the Royal Society, an experiment to demonstrate what he terms "the passive tension of the diaphragm." "The trachea of a sheep immediately after death was exposed in the neck, divided, and tied over a glass tube, which was put in communication with a graduated receiver placed under water and guarded by a stopcock. The pleuræ were then opened, and, as air entered, the diaphragm became flaccid, but the lungs remained unchanged in position and form." It is clear that the air in this experiment could only enter the pleura in consequence of the expansion of the chest by virtue of the *excentric thoracic resilience*. For, the lungs being secured, they could not collapse and so suck in the air. Mr. Le Gros Clark's experiment is further valuable to the present purpose inasmuch as it very clearly shows that the diaphragm also exercises some elastic traction upon the lung; indeed, it is Mr. Clark's object in his paper to point out that this *passive tension* is the means of retaining the supplemental air in the lungs.

My principal object in bringing the present paper before the Society is to endeavour to point out some of the bearings of these physiological facts in clinical medicine, and to indicate how they serve to afford us a better insight as to the true mechanism and relative value in diagnosis of some of the signs of chest disease. Let me, however, venture first to observe that, so far as I am aware, no physiologist has fully pointed out the importance of the thoracic resilience as a conservator of force in respiration. Mr. Hutchinson and all who have followed him place thoracic elasticity as a force, the chief force indeed, against

which the inspiratory muscles have to contend, and which is wholly in favour of expiration. Even Dr. Salter, who observes that thoracic elasticity is in favour of ordinary inspiration at its very commencement, places it as the opponent of the latter portion of that act. It is obvious, however, on very slight reflection, that the elasticity of the chest-wall is a force, not only in favour of inspiration at the commencement, but against expiration at the termination of the respiratory act. It renders easier the expansion of the chest by neutralising the first resistance and inertia of the lungs, and in the final contraction of the chest in expiration exercises a buffer-like action in taking off the shock of recoil. This elastic *help* at the beginning and elastic *kindrance* at the end of the respiratory act is a spring-like function of the chest-wall, the importance of which has not been duly acknowledged. It is only necessary, however, to glance at the emphysematous chest to note the uneasiness entailed by its loss. The lungs in emphysema have not lost all their elasticity, but they have lost so much of it that the excentric thoracic and diaphragmatic resilience has nothing to oppose it, the chest boundaries are no longer therefore drawn inwards by the lungs in expiration beyond the position they would of their own accord adopt. Every physician must have repeatedly remarked the peculiar jerk or tug with which inspiration commences in extreme emphysema, a *tug* which communicates a shock to the stethoscope very appreciable to the ear.

It is important to know whether in calm breathing the chest-wall expands beyond the point to which its resilience would, if unopposed by the lung-elasticity, carry it. Mr. Hutchinson calculated the costal movement in health not to exceed from $\frac{1}{10}$ to $\frac{1}{4}$ of a line, *i. e.* from 1—2 millimètres in quiet breathing. Taking the average of Dr. Sanderson's measurements in the case of a healthy man aged 22, we find the expansion to be 1.6 millimètre.

Dr. Sibson gives the thoracic movement during tranquil

inspiration as $\frac{1}{16}$ inch; in very robust people rather less, in weakly persons considerably more.

Dr. Ransome, in his valuable paper on the respiratory movements read before the Society in 1872, has, unfortunately, not recorded the thoracic movement of calm breathing.

I have myself made some observations to ascertain the extent of thoracic expansion in calm breathing by means of a very simple but accurate instrument made for me by Mr. Hawksley. This instrument may be briefly described as consisting of a convenient chest-piece connected with a straight rod made of a light material by means of an elastic joint. The other end of the rod has a metallic writer affixed which marks upon a horizontal plate that can be shifted by ordinary clockwork. This instrument simply records the forward movement of the chest-wall in any given direction, without reference to the mechanism in such expansion which does not, for the moment, concern us.

The mean of nineteen measurements of expansion taken from myself, instrument being applied at mid-sternum level of fifth cartilage was 2.75 mm., least expansion being 2.5, greatest 4 mm. Observations upon others have shown considerable variations, but within this range.

Let us now compare these movements with the expansion of the chest as ascertained *post mortem* to be the result of its resilience alone. Dr. Salter, in his experiment upon the dead subject already quoted, measured an expansion of the chest on permitting air to enter the pleura of 1 millimètre. I have repeated Dr. Salter's experiment at different points of the chest, avoiding, however, in all cases dividing any of the cartilages.

The instrument used in these experiments is similar to that employed by Dr. Salter, and consists of a fine tube (A, B) expanded at its lower end into a bell shape, across which is stretched a piece of caoutchouc (B) having attached to its centre a projecting button (c). The bell is filled with a coloured fluid, which occupies also a few inches of

FIG. 1.

the fine tube. Diameter of base of tube B is found to be 22 millimètres. Diameter of tube 1·5 mm., their proportion to one another being therefore as 14·66 to 1.

Experiment 1 (*vide* Table No. I).—Male subject, aged 33, death one week after double amputation for gangrene.

Post-mortem, twenty-four hours.—Rigor mortis still present in arms, slight tinge of commencing decomposition at root of neck. Right side of chest perceptibly more prominent than left.

Integuments reflected from chest, trachea cut across below cricoid cartilage, to ensure no obstruction from any mucous collection at the glottis. The third cartilage exposed on each side of the sternum. Instrument fixed by suitable holder vertically over the third left cartilage, so that button of bulb rested lightly upon it, causing the level of fluid in the tube to rise a few inches. The level of the

fluid D in the stem then accurately noted from scale attached, and found to be quite sensitive to any expansion or recession of the chest-wall. The pleura was then cautiously opened by an incision between the second and third left cartilages, and the fluid in the stem (observed by Dr. Coupland) steadily but somewhat slowly rose $1\frac{1}{4}$ inch, or 3·15 centimètres, which, divided by 14·6, gives 2·143 millimètres of actual expansion. On opening the right pleura in similar way, no further expansion. On examining the chest, the cartilages were not ossified, both lungs were somewhat emphysematous, right more so than left.

My results are briefly recorded in the following table (see Table No 1).

Insufficient as these observations are to justify any very positive conclusions, they yet furnish strong evidence in favour of the two following propositions :

1. That in health the excentric thoracic resilience favours inspiration throughout the normal respiratory act in calm breathing.

2. That in emphysema or œdema, as the lungs enlarge, the thoracic walls yield outwards, but that up to an expansion of probably between 3 and 4 millimètres there is no intrathoracic pressure.

I must not omit here to thank Dr. Coupland for his kind help in giving me opportunities for my experiments at the Middlesex Hospital, and for co-operating with me in making the observations.

Ordinary inspiration being mainly, however, effected by the descent of the diaphragm, the resilience of that muscular membrane, its "passive tension" according to Mr. Le Gros Clark, is in favour of inspiration at the commencement of that act. This resilience of the diaphragm is doubtless derived from its attachment to the costal margin. It is usually said that the diaphragm is the only muscle used in calm inspiration; it is obvious, however, that if this were so the chest-walls would yield inwards with the descent of this muscle; whereas, on the contrary, the chest expands a little, albeit a very little, and

TABLE I.—*Giving observations on thoracic resilience in 10 cases post mortem.*
All observations made with the co-operation of Dr. Coupland at the Middlesex Hospital.

No	Sex.	Age.	P. M.	Case.	Point of application of bulb.	Pleura opened.	Rise of fluid in stem.		Equivalent expansion of chest. Millim.	Remarks.
							Centim.	Total.		
1	M.	33	24 hrs.	Amputation	3rd L. cart.	a—2nd L. space b—2nd R. space	3·15 0	3·15	2·143	Right lung adherent, emphysematous; left free, slightly emphysematous; abdomen flaccid.
2	F.	38	21 hrs.	Tumour of jaw	3rd mid. st.	a—3rd space b—3rd space		3·5	2·39	Slight further rise on making two fresh openings at 5th space; slight bronchitis.
3	M.	54	27 hrs.	Epithelioma of penis	4th mid. st.	a—3rd R. space b—2nd R. space c—3rd L. space	0 0 2·4	2·4	1·63	Right lung adherent, side more prominent; some emphysema of both lungs, mostly of the right.
4	F.	44	20 hrs.	Carcinoma of uterus	5th mid. st.	a—2nd R. space b—2nd L. space	2·3 2·3	4·6	3·19	Lungs quite healthy, no adhesions; collapsed to about 1/4th of bulk; rigor mortis, no decomposition; abdomen flaccid.
5	M.	27	23 hrs.	Del. tremens	4th mid. st.	a—3rd R. space b—2nd L. space	0 0·3	0·3	0	Edema of both lungs, recent pleural adhesions both sides.
6	F.	36	...	Comp. fract., bruised strum	5th mid. st.	a—2nd R. space b—2nd L. space	0 0	0	0	Bronchitis; adhesions both sides.
7	M.	60	...	Fract. ribs, left side	4th mid. st.	a—2nd R. space b—2nd L. space	0 0	0	0	Cartilages ossified; right lung emphysematous; three pints serum in left pleura.
8	M.	64	...	Strangulated hernia	4th mid. st.	a—2nd R. space b—2nd L. space	0·3 0	0·3	0	Lungs emphysematous and cedematous.
9	M.	41	12 hrs.	Fractured skull	4th mid. st.	a—2nd R. space b—2nd L. space	0 0	0	0	Lungs highly cedematous; dynamometer connected with trachea registered no pressure.
10	F.	56	40 hrs.	Carcinoma uteri	5th mid. st.	a—3rd L. space b—3rd R. space	0 0·4	0·4	0	Rigor mortis passing off; abdomen distended; chest-walls rigid, lungs emphysematous, but collapsed half an inch.

GENERAL REMARKS ON TABLE.—The first 4 cases give positive results, viz. an actual expansion of chest on opening the pleural cavities = from 1·6 mm. to 3·19 mm. Case 4, giving the greatest expansion, was the only case in which the lungs were quite healthy. The last 6 cases give practically negative results, viz. no expansion or contraction in 3 cases, a small fractional expansion in 1 case, and a small fractional contraction in 2 cases. In all these latter 6 cases the lungs were notably affected by disease.

this must be by the aid of muscular action. The intercostals especially, as pointed out by Mr. Clark, acting from above downwards, *i. e.* from the less to the more movable ribs, support and even expand the costal margin whilst it endures the muscular traction of the diaphragm. It is the function of the excentric thoracic resilience to reduce the muscular power thus used in calm inspiration to a minimum.

A word or two must now be said as to the manner in which the mediastinum is affected by the elastic tension of the lungs. Dividing the thoracic cavity into two halves, the mediastinum presents a surface to each, and between these two surfaces are included the heart and the great vessels proceeding to and from it, the main bronchi, glands, &c. The mediastinum is movable from side to side within certain limits. Now, just as the elastic tension of the lung draws upon the thoracic wall, and has as its counterpoise the resilience of the ribs and cartilage, so, in the median line, the elastic lung on one side draws upon the mediastinum and has as its counterpoise an equal traction of the opposite lung. This traction upon the mediastinum from either side is *constant* in health, existing even in extreme expiration, but it is vastly increased with the expansion of the lungs. Its tendency is to draw apart the two layers, if we may so think of them, of the mediastinum and to exercise a suction power upon the blood towards the heart, arteries, and veins which they include. This constant determination of blood towards the heart, increased with each inspiration, has attracted much attention. It is beyond our purpose now to consider its multiple effects and counter-effects. It is obvious, however, and the stethograph records the fact, that every contraction of the heart modifies the respiratory curve, and, contrariwise, the respiration modifies the pulse-trace. We need go no further to find the chief cause of the constant tendency to the entry of air into a vein opened at the root of the neck.

The above considerations enable us better to understand

some phenomena of disease which would be otherwise obscure, and which have been in some instances inaccurately or inadequately explained.

Among the diseases which affect the lung- or thoracic elasticity we must first speak of that common pulmonary disease *vesicular emphysema* of the so-called hypertrophic kind. It has been currently taught since the time of Stokes that the characteristic signs of emphysema—the enlarged chest, the displaced heart, the lowered diaphragm and abdominal organs, and the troubled circulation—are due to the pressure of the expanded lungs. Now, it would be unwise to assert that such pressure by the lungs upon their surroundings is *impossible*, but both physiological and clinical observation, I think, concur in showing that it almost never occurs. It is singular that Stokes'—who regarded the lungs in emphysema as being compressed by the chest-walls, and as in their turn pressing upon other parts—should have at the same time given it as his constant experience that the intercostal spaces, so far from being bulged, are, in fact, more deeply marked in this disease than in health.² Without going so far as Dr. Stokes, whose assertion in this latter respect has been earnestly combated by others, one may safely affirm that only in very marked cases of emphysema do the intercostal spaces become *level* in expiration, that save in forced expiration these spaces never puff outwards, and that they invariably deepen at the first moment of inspiration. Similarly do all other soft parts deepen with inspiration even in the most extreme cases of emphysema. This is very notably the case with the supra-clavicular and supra-sternal fossæ. These facts prove that although the lungs in emphysema are expanded from loss of their elasticity, according to the degree of emphysema, the chest is similarly and to an equal extent expanded, not by any *pressure of the lungs*, but by the *excentric thoracic resilience* being less and less

¹ 'Dis. of Chest,' 1837, part i, p. 187.

² Dr. Stokes observes that, even in fluid effusions, the intercostal grooves are not obliterated, except in very severe cases or in cases of long duration.

opposed as the emphysema increases. Any further enlargement of the chest during inspiration, however, is opposed, as it is in health, by the elasticity of the lungs and, I may add, by the *concentric* thoracic resilience. In fact, as has been pointed out by Dr. C. J. B. Williams and others, inspiration in great emphysema begins at the level at which normal inspiration would terminate, *i. e.* between the levels B and C of the table of coefficients. The diaphragm is in emphysema similarly lowered, not being drawn up to its normal degree of convexity with expiration; it is hence disabled for inspiration, which is mainly accomplished by thoracic expansion in this disease in its more advanced stages. The extraordinary muscles of inspiration are habitually used in emphysema, and the peculiar jerk with which they come into action, at once to overcome the inertia and resistance of the thoracic parietes and the lungs, has already been referred to as illustrating, by contrast, the important part elasticity plays in facilitating the normal respiratory act. The position of the heart and liver are necessarily lowered with the lowered position of the diaphragm. It is inaccurate, therefore, to say that these organs are pushed downwards by the pressure of the enlarged lungs. It must, too, be only in very rare instances that the heart is compressed between the lungs. These organs are sometimes, however, described as, in emphysema, bulging forth after death on removing the sternum. My own observation would lead me to doubt this occurrence except where some fluid is present in the pleura, or some obstruction such as inhaled blood-clot occludes bronchi immediately before death. The whole venous circulation is in emphysema impeded by the extinction of that *constant* aspiration towards the heart which obtains in health, and which in this disease is only present in an enfeebled degree during inspiration. Sir William Jenner has, however, clearly pointed out that the obstruction in the pulmonary capillaries is the chief cause of the disordered circulation and dropsy that ensue in this disease.

In another condition of things the opposite to that of

emphysema, viz. in which, while the lung elasticity remains normal, the thoracic wall has suffered loss of resilience through impairment in the nutrition of its bony and muscular structures, we observe recession of the least resisting parts, increased during inspiration, and consequent deformity of the chest.

It would appear from some interesting observations by M. Woillez¹ that in the pyrexial stage of acute diseases an appreciable enlargement of the thoracic circumference takes place, gradually to subside as the fever abates. M. Woillez has taken careful measurements in cases of variola, idiopathic erysipelas, scarlatina, articular rheumatism, pneumonia, hæmoptysis, bronchitis, and pericarditis, and he finds the enlargement to take place within the first few hours of the onset of fever, and to more gradually subside to the normal as the disease retrogresses. He considers the increased circumference of the chest to be due to a "diminished general elasticity of the chest," which he traces mainly to loss of elasticity of the lungs in consequence of their temporary engorgement in fever. I do not know that any one has repeated these observations of M. Woillez.

The ready expansion of the chest-walls by virtue of their elastic resilience to the increased capacity needed in all cases of acute and rapid enlargement of one or both lungs, *e. g.* in pneumonia, bronchitis, whooping-cough, &c., is a beautiful instance of adaptation in the human mechanism.

In cases of effusion into the pleura, whether of air or fluid, there are two phenomena to be always observed, unless their occurrence be prevented in some exceptional manner, viz. displacement of heart and enlargement of the circumference of the chest, especially of that half of it corresponding with the affected side.

Displacement of the heart in pneumothorax.—On the occurrence of pneumothorax the condition of the air within the pleural cavity as regards pressure depends upon the nature of the perforation, whether it be a free or a

¹ "Recherches sur les Variations de la Capacité thoracique dans les Maladies Aigües," 'Mémoires de la Société Médicale d'Observation.'

valvular opening. If the communication with the bronchus be free and patent, the air passes to and fro through the lung with inspiration and expiration. In this case it is obvious, and it can be readily shown, that there is no compression of the air within the pleura. If, however, the perforation be not free, but more or less valvular, the entry of additional air into the pleura readily takes place, but its escape with expiration is imperfect or impossible, and consequently there is in these cases air pent up in the pleura and exercising pressure upon all surrounding parts.

Now, as a matter of clinical experience, we find in both cases, *i. e.* when there is free circulation of air in the pleura and when the air is confined and pent up there, that displacement of the heart is one of the first and most necessary signs of the pneumothorax.

I have tabulated some notes of seventeen cases of pneumothorax to give information with regard to the degree of pressure and the position of the heart. Of these 17 cases 9 were males, 6 females, and in 2 instances the sex is not stated. The pneumothorax was in 5 cases on the right side, in 10 on the left, and in 1 case it was double-sided. In 12 of the cases tabulated there was found *post mortem* an air pressure within the pleura varying in degree:—the highest pressures being 7 inches, $5\frac{1}{2}$ inches, and $5\frac{3}{10}$ inches of *water* respectively, and the lowest pressures $1\frac{1}{4}$ inch and 2 inches. In all these cases the heart was displaced, although in four of them I do not find the fact definitely stated in my notes. Woodcuts Nos. 2 and 3 are diagrams of a case, No. 11, in which the intrapleural pressure was the highest. One diagram (woodcut No. 2) was taken during life and illustrates the clinical features of the case; the other (No. 3) delineates the position of parts found at the *post-mortem* examination.

In 4 out of the 17 cases there was no intrapleural pressure, yet in 3 of these cases the heart was very greatly displaced, and in the fourth case, in which the heart was not displaced, this was accounted for by the unruptured lung being so consolidated as not to permit of its collapse.

TABLE II.—Cases of *pneumothorax tabulated*.

No. Age.	Sex.	Side affected.	Duration of pneumothorax.	P.-M., hours.	Air pressure in pleura.	Position of heart.	Physician.	Remarks.
1	...	...	...	30	4 in. of water	...	...	No further p.m. notes.
2	M.	Right	2 days	12	5 $\frac{1}{10}$ in. "	Displaced to left	Dr. Sanderson	Two openings in pleura; no decomposition or distension of abdomen; $\frac{3}{4}$ pint of turbid fluid in pleura.
3	F.	Left	A few days	14	2 in. "	Displaced to right	Dr. Powell	No examination allowed beyond testing pressure.
4	M.	Left	2 days	16	3 $\frac{1}{4}$ in. "	Not stated	Dr. Sanderson	Opening valvular.
5	M.	Right	30 hours	30	2 in. "	Ditto	Ditto	Base of right lung collapsed—the only healthy portion of lungs.
6	M.	Right	36 hours	30	4 in. "	Displaced to left	Dr. Quain	Liver displaced downwards; opening perfectly valvular. <i>Vide</i> 'Path. Trans., vol. xix, p. 77.
7	M.	Left	3 days	20	1 $\frac{1}{4}$ in. "	Displaced backwards	Ditto	Air from left pleura had entered into pericardium. 'Path. Trans., vol. xx, p. 99.
8	F.	Both	Left old, Rt. 10 min.	24	L. 3 $\frac{1}{2}$ in. " R. 2 $\frac{3}{4}$ in. "	On admission displaced to right	Dr. Pollock	Two quarts of pus in left pleura.
9	M.	Left	4 $\frac{1}{2}$ months	30	Nil	Displaced to right of sternum	Dr. Cotton	Left pleura contained a small quantity of purulent fluid. 'Med. Times,' Aug. 21st, 1869.
10	F.	Left	13 days	30	Nil	Ditto	Dr. Alison	Left pleura contained about a pint of purulent fluid. Loc. cit.
11	M.	Left	11 days	18	7 in. "	Ditto	Dr. Pollock	A valvular opening.
12	M.	Left	...	...	2 in. "	Not stated	...	Opening valvular.
13	F.	Left	...	30	3 $\frac{3}{8}$ in. "	Displaced to right	Dr. Pollock	Opening could not be discovered.
14	F.	Right	7 days	...	Nil	Not displaced	Dr. Cotton	Recent consolidation of left lung.
15	F.	Left	15 hours	...	Not noted	Ditto	Dr. Pollock	Opening free, so pressure probably <i>nil</i> ; right lung consolidated throughout.
16	M.	Right	...	40	5 $\frac{1}{2}$ in. "	Displaced to left	...	Small valvular opening.
17	M.	Left	...	...	Nil	Apex at left ensiform	Dr. Cotton	Opening free.

SUMMARY.—17 cases { 9 M. } 5 Right { 8 heart displaced } Probably in all cases displaced.
 { 6 F. } 10 Left { 4 not stated }
 { 2 F. } 1 Double { 3 heart displaced }
 4 no pressure . . . { 1 not displaced, accounted for by consolidation of left lung.
 1 not noted . . . Heart not displaced, right lung consolidated.

Figs. 2, 3.—Diagrams of Case 11, Table II. Left pneumothorax ; intra-pleural pressure = 7 inches of water.

Diag. 2, taken during life.—*a*. Outline of heart. *b*. Position of undulating impulse. *c*. Apex beat. *e*. Some dulness and moist crackling rhonchus. Left side enlarged and hyperresonant ; no respiratory sound save at *d*, where inspiration has some hollow quality. Diag. 3 indicates exact position of parts as found *post mortem*. *a*. Tube inserted into left pleura to ascertain intra-thoracic pressure. The shading at the upper part on this side indicates the compressed left lung.

The following diagrams illustrate these three cases. It will be observed from the remarks attached to two of these cases in the Table that a small quantity of purulent fluid was found in the pleura of one and about a pint of purulent fluid in that of the other. It must, however, I think, be granted that this amount of fluid was quite insufficient to account for any appreciable displacement, especially when it is remembered that it did not cause any compression of the air within the pleura. In the third case there was no fluid present.

Some seven or eight years ago I endeavoured to explain and to illustrate the essential cause of the cardiac displacement in pneumothorax by some simple experiments which were related to this Society.¹ I need only briefly mention them now.

¹ 'Medical Times and Gazette,' Jan. and Feb., 1869.

Figs. 4, 5.—Case No. 9, Table II. Left pneumothorax; no intra-thoracic pressure.

Diag. 4.—Percussion limits marked out before opening thorax. *a, b.* Boundary of left pleura. *a.* Displaced cardiac dulness. Diag. 5.—Exact position of heart ascertained on removing sternum. A small puncture is indicated at fourth space, through which a stilette was thrust to fix the heart *in situ* before removing sternum.

Figs. 6, 7.—Case No. 10, Table II. Left pneumothorax; no intra-thoracic pressure.

Diag. 6, taken before removing sternum, showing displaced cardiac dulness.—*st.* Point at which stilette inserted to transfix heart *in situ*. *a, b.* Margin of left pleural cavity. Diag. 7 shows position of heart and collapsed left lung as seen on removing cartilages.

NOTE.—The roughness of the above diagrams is due to their being exact copies, on a reduced scale, from my clinical and post-mortem note-books.

FIG. 8.

Case No. 17, Table II. Left pneumothorax; no intra-thoracic pressure. *ll*. Collapsed left lung, communicating with pleura by free opening at *a*.

1. In the dead subject, the chest being healthy, a long and light needle was thrust vertically into the heart to serve as an index (*vide* woodcuts Nos. 9 and 10), and on the left pleura being then cautiously opened so as to freely admit air, the needle became slightly deflected, so as to indicate a movement of the heart towards the right. An inspiration was now imitated by evenly raising both arms above the head, and the displacement became more marked.

2. A similar experiment was made upon a living dog whilst under chloroform. The deflection of the needle was still more marked.

The inference from these experiments was that the elastic tension of one lung, when unopposed by that of the other, was sufficient to draw aside the mediastinum and with it the heart. In the case of the dog the displacement was still more marked, because the lungs being in action their tension was naturally increased with inspiration.

The chief conclusions which seem to follow from the facts recorded in the table are—

1. That displacement of the heart is an immediate and a most important sign of pneumothorax, depending

Figs. 9, 10.—Diagrams of sections of the chest to illustrate experiments showing the effect upon the position of the heart of admitting air into one pleural cavity. Fig. 10 shows deflection of an arrow thrust vertically into the heart on making a free communication between the left pleural cavity (*pc*) and the external air. (Adapted from Pirogoff's Anatomical Plates.)

upon the mere presence of air in the pleura and upon the contractility of the unruptured lung.

2. That the cardiac displacement is by no means necessarily a sign of intra-pleural pressure, since, as will be seen on glancing at the diagrams of Cases 9 and 10 (*vide* woodcuts Nos. 4 to 7), the heart may be displaced to the right of the sternum without there being any pressure.

3. Hence, in discussing the question of paracentesis in any given case of pneumothorax we must take into consideration other things besides the position of the heart.

The above views are, however, by no means generally accepted even by those who are acquainted with them. Dr. Hayden, of Dublin, in his recent elaborate work on 'Diseases of the Heart,' at p. 100, after very fully and fairly stating my experiments and deductions, gives what he conceives to be good reasons for adhering to the doctrine of lateral displacement of the heart by pressure, "as promulgated by Bertin, Laennec, Hope, Stokes, and Walshe, and almost universally held at the present time." In the first place, Dr. Hayden thinks that in my experiments the deflection of the needle was "directly and exclusively due to pressure upon the mediastinum of the air admitted into the pleura. This air being free, and its pressure therefore not being neutralised by the elastic reaction of the lung, as was the case with that included within the pulmonary tissue of the opposite lung." If Dr. Hayden's objections be valid they will not fail to be ably urged by some of the Fellows present, and it would be taking up too much of the Society's time to refer at length to his arguments now. I must say, however, that it is difficult to understand how there can be any difference between the atmospheric pressure within the lung and that outside the thorax; and the atmospheric pressure being the same, therefore, on both sides of the balance, but the elastic tension on the one side being annulled, the elastic tension on the other side seems to be the only force which, thus unopposed, can rightly be considered as disturbing the equilibrium. I would beg further to remark respecting

two points in Dr. Hayden's experiment undertaken in order to test the accuracy of my views. Firstly, that he apparently does not consider that the thoracic wall has anything to do with the question ; and, secondly, he seems to think that by inflating the lung he can imitate its normal expansion. Thus, Dr. Hayden¹ commences his experiment by removing the anterior wall of the chest of a subject brought in for dissection, except the sternum, which he divides in the median line with a saw. He then detaches the root of the left lung and corks up its main bronchus. He then further proceeds to inflate the right lung by blowing into the trachea to ascertain the effect upon the heart's position. Is it possible to conceive any experiment performed under conditions more diverse from those which are natural ? Yet it is from such an experiment that Dr. Hayden draws conclusions favorable to the views of the distinguished observers already quoted.

Whilst contending that the *primary* cause of displacement of the heart in pneumothorax is not air-pressure acting from the diseased side, but lung-traction acting upon the healthy side, I am, of course, fully aware that such displacement must necessarily be increased should air accumulate in the pleura.

The mechanism of displacement of the heart in cases of fluid effusion into the pleura is essentially the same, but the occupation of the pleura by the effusion being gradual instead of sudden, as is its occupation by air, the displacement of the heart is slow instead of being instantaneous.

Cardiac dislocation occurs in pleurisy *pari passu* with the effusion, whereas if its occurrence were a matter of *pressure* it should not take place so long as the fluid no more than occupied the space left by the contracted lung.

I would mention, by the way, one little point of clinical interest to be observed in cases of moderate effusion into the right pleura, viz. that in such cases the heart may appear to beat more to the right than natural. This is

¹ P. 102.

due to the right lung as it collapses uncovering the right side of the heart, the impulse of which is much more readily transmitted through fluid than through the spongy lung. Thus, whilst the heart is really displaced slightly to the left, it may seem not to be displaced at all or even to be somewhat more to the right than natural.

In cases of considerable fluid effusion into the pleura there is, of course, always pressure upon the heart. I have measured this intra-pleural pressure in several cases at the various periods of removal of the fluid by thoracocentesis, and have found the fluid pressure to vary from half an inch to an inch and a half of *mercury* at the moment of puncturing the chest. I subjoin a table giving particulars of three such cases, but need not further refer to them.

It may, however, at first appear somewhat surprising that with the considerable intra-pleural pressure here recorded there is not more difference between the chest measurements on the two sides in cases of effusion into the pleura. Sometimes these measurements are equal; there is rarely an inch or an inch and a half of difference between them. A little consideration, however, points out the reason of this, and it is the last matter I have to touch upon. When an effusion, whether of air or fluid, takes place into the pleura on one side, in proportion as the mediastinum encroaches upon the healthy side the thoracic parietes on that side expand in consequence of the relaxed lung setting free their excentric resilience. This tends to equalise the measurements of the two sides, but to increase the total circumference of the chest.

NOTE.—I have only been able briefly to glance at the elaborate work just published by Dr. Ransome on stethometry. As in his paper referred to, at page 170, however, the thoracic movements recorded in Dr. Ransome's present tables appear to be those of forced breathing, with which the observations in the present paper do not deal. At page 96 Dr. Ransome makes an interesting observation with reference to forced breathing, which, as I have shown, should also (proportionately) hold good with ordinary respiration, viz. that in measuring the movements of forced breathing by means of his three-planned stethometer, "the index of the *forward* dial generally moves first, a forward push of 0.25 to 0.30 inch being often noticed before any decided action of the other indices takes place. . . . This fact," observes Dr. Ransome, "would probably be sufficient to prove that there must be some preliminary indrawing of the chest-wall in the expiratory act."

TABLE III.—*Showing intra-pleural pressure in cases of empyema.*

Case.	Sex.	Age.	Side.	Duration.	Intra-pleural pressure (mercury).	Fluid removed.	Chest measurements before operation.
T. W.	M.	30	Left	10 months	<p>Commencement of operation + 1 in. ... $\frac{1}{4}$th in.</p> <p>Termination of operation + $\frac{1}{4}$ in. ... $\frac{3}{4}$th in.</p> <p>Movement } $\frac{1}{2}$ in.</p>	Thin pus, $4\frac{1}{2}$ pints	<p>Left semicircumference ... $18\frac{1}{2}$ in.</p> <p>Right " ... $18\frac{1}{2}$ in.</p> <p>Difference ... $\frac{1}{4}$</p>
T. B.	M.	28	Right	5 years	<p>Commencement of operation + $\frac{1}{4}$ in.</p> <p>Termination of operation — 1 in.</p>	Pus, 4 pints	<p>Right semicircumference... $18\frac{1}{2}$ in.</p> <p>Left " ... $17\frac{3}{8}$ in.</p> <p>Difference ... $\frac{1}{8}$ in.</p>
E. M.	M.	22	Left	...	<p>Commencement of operation —</p> <p>During inspiration + $1\frac{1}{4}$ in.</p> <p>During expiration + $1\frac{1}{2}$ in.</p> <p>Termination of operation $\pm$ level</p>	Pus, $4\frac{1}{2}$ pints	<p>Left semicircumference ... $17\frac{3}{8}$ in.</p> <p>Right " ... $16\frac{3}{8}$ in.</p> <p>Difference ... $1\frac{1}{4}$ in.</p>