REMARKS

ON

THE ELASTICITY OF THE LUNGS AND CHEST-WALLS: WITH REFERENCE TO SOME PHENOMENA OF CHEST-DISEASE.*

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MR. PRESIDENT AND GENTLEMEN,—I must ask for your kind indulgence, and that of the members present, if, in my endeavour to condense my remarks within the prescribed limit of time (twenty minutes), I become a little disconnected, if not incoherent. A few preliminary considerations of a physiological kind are necessary, inasmuch as the matter before us hangs very much upon our estimation of the forces concerned in respiration.

In order to illustrate the current views respecting the mechanism of respiration, I have taken these diagrams (Fig. 1) from one of our best and most modern text-books of physiology. They are copied from Funke, but they also represent the essential features shown in Donders's well-known schema, with the addition that here a small piece of India-rubber (6) is inserted to represent the elasticity of the chest-walls.

Fig. 1.—The left hand figure represents a flask, through the cork of which a tube is passed, having attached to it two India-rubber bags, to represent the lungs; 3 is a mercurial manometer communicating with interior of flask; 4 is a piece of India-rubber to represent the diaphragm; at 6, a small piece of India-rubber closes an opening in the flask. On pulling downwards the diaphragm by means of knob 5, the condition of inspiration is represented, the lungs expanded, the mercury of manometer and the elastic lamina (6) pressed inwards by atmospheric pressure. (Copied from Dr. McKendrick's *Outlines of Physiology*.)

These diagrams figure fairly well the views of the mechanism of respiration which have been held since Hutchinson, more than thirty years ago, published his valuable paper: viz., that inspiration is a wholly muscular act; the muscles, in expanding the thorax, having to contend against, first, the elasticity of the lungs, second, the inertia and elasticity of the chest-walls. Expiration, on the other hand, is effected by the elastic recoil of the lungs and of the chest wall.

Now, I want to bring forward certain facts, which have most of them been long known, to show—first, respecting inspiration, that in calm breathing there is no inertia or elasticity of the chest-walls to be overcome, but, on the contrary, that the thoracic elasticity is a force of appreciable power, constantly tending to enlarge the thorax, and therefore acting in favour of inspiration.

That it is a matter of some importance whether our chest-wall elasticity is for or against us in inspiration will be clear, when I remind you that Hutchinson estimated the elastic resistance to each inspiration arising from the chest walls alone at more than 100 lbs., and, in deep inspiration, at 400 even to 1,000 lbs. (*Medico-Chirurgical Transactions*, vol. 29, pp. 208-211.) It may be interesting to some of us, already oppressed by a sense of hard work, to know that our mere breath-fetching is not so laborious a proceeding as this. The rectification of this error also enables us satisfactorily to explain many morbid chest-conditions which would be otherwise obscure. What, then, are

the facts? The late Dr. Salter was, I believe, the first, eighteen years ago, to point out that, at the commencement of breathing, the elasticity of the chest-walls should be placed as an inspiratory force. By a simple experiment, he showed that, in the dead subject, the thorax, when relieved from the traction of the lungs by making an opening into the pleura, expanded to the extent of $\frac{1}{10}$ th inch. This appears but a small result, but it was full of significance. Unfortunately, Salter did not repeat his experiments. This has been done since, however, by different methods and with additional precautions. Traube found, many

Fig. 2.—Schema arranged to represent the conditions present in the chest at the period of respiratory pause—i.e., at the end of calm expiration. Inspiration is effected by exhausting with the syringe. The balance of mercury (H I H') is now observed to be in favour of inspiration. If the exhausting by syringe be carried beyond the point at which the mercurial columns (H K H' K') become level, deep inspiration will be represented, during which the balance of mercury (i.e., the thoracic elasticity) will be in favour of expiration. (For a description, see text.)

years later* (*Gesammelte Beiträge*, Band i, *Experimentelle Untersuchungen*, page 141, 1871), in the living dog, that the chest-walls, on being relieved from the traction of the lungs, expanded in the manner indicated by Salter; but he does not seem to have been aware of Salter's experiment, and he asserted that the same result was not obtained in the dead animal, and he regarded it, therefore, as a phenomenon depending upon muscular action. Paul Bert (*Leçons sur la Respiration*, 1870, page 359), confirmed Salter's observations, by recording simultaneously the elastic pressure of the lungs and the expansion of the chest-wall at the moment of opening the pleura of a recently killed dog. I have myself repeated them, with additional precautions, in several cases, and have found that in the healthy chest the expansion is from 1.6 to 3.19 millimètres.† It would appear, then, that in this respect the mechanical conditions of the chest were erroneously estimated by Hutchinson, and are erroneously depicted in these diagrams (Fig. 1), inasmuch as the chest-walls, being elastic—albeit stiffly so—are drawn inwards by the lungs, until the elasticity of the one counterbalances the elasticity of the other. Hence, in the position of ordinary expiration, the lungs should be represented as partially expanded, the chest-wall as slightly drawn inwards, and, let me add, the mercury of the manometers as rising towards the chest, in accordance with the negative pressure there. The diaphragm is, of course, amenable to the same influence, and, as has been pointed out by Mr. Le Gros Clark (*Proceedings of Royal Society*, May 1871), is drawn upwards into a position of passive tension. If, in the right-hand diagram of Fig. 1, intended to represent the chest during inspiration, we were to represent the diaphragm in the position of dotted line 5', we should

* Traube also observes the flattening of the diaphragm on the affected side. Dr. Ransome, from whose valuable work on stethometry, at page 95, I obtain the reference to Traube, adopts that author's view of the mechanism of these phenomena.

† For fuller description and details respecting these and other experiments, see *Medico-Chirurgical Transactions*, vol. lix, and author's recent work on *Consumption and Diseases of the Pleura*, page 272, et seq.

‡ Constructed by Mr. Hawksley of Oxford Street.

* Read in the Section of Medicine at the Annual Meeting of the British Medical Association in Bath, August 1878.

fairly depict the real conditions present in the chest during the respiratory pause, *i.e.*, at the end of ordinary expiration. I have here an instrument† which represents more accurately the statics of the chest, and which I will describe to you in a very few words.

This schema (Fig. 2) consists of a closed cylinder of glass, divided into two air-tight compartments (A A') by an elastic mediastinum, E F, made of thin sheet India-rubber. Each compartment represents one-half of the chest consisting of a pleural cavity, A (A') containing an elastic bag or lung c (c'). Each lung is provided with a tube D (D') or trachea. A second tube B (B') communicates with the pleura and is closed by a stopcock, and a third tube, G (G'), also opening into the pleura is attached externally to a manometer.

By opening the stopcocks of the trachea-tubes D, D' on each side, and then by partially exhausting the air from the pleural chambers through the tubes B B', and shutting their stopcocks, the bags c c' are partially expanded C C' to represent the condition of the lungs before inspiration. It will be observed also that there is a rise of mercury H I H' I' towards the chest-cavity; this rise represents the recession of the chest wall and diaphragm in obedience to the traction of the lungs, and is equivalent therefore to elastic laminæ 6, 6, of diagrams (Fig. 1). The extremities of the manometers remote from the chest are connected by tubing with the nozzle of an ordinary syringe. The hand of the operator drawing the piston of the syringe represents the muscles of inspiration. Thus, when I withdraw the piston, I cause the mercurial columns to become level, and the lungs C C' to further expand. Thus far the weight of mercury H I, H' I', representing thoracic elasticity, has helped me as an inspiratory force. If now I exhaust still further, so as to represent a deep inspiration, the mercury mounts up in the opposite limb of the manometers, and the balance of mercury (*i.e.*, thoracic elasticity) becomes more and more against me, and ready to act as an expiratory force.

It must be remembered that the columns of mercury H I, H' I' represent on each side the sums total of but a small expansion of from 1 to 3.5 millimètres, distributed over the surface of the chest-walls. Careful observation, however, shows that these figures, small as they are, indicate pretty well the limits of expansion of the ribs during calm breathing. Hutchinson calculated the costal movement in health at $\frac{1}{10}$ to $\frac{1}{2}$ line = 1 to 2 millimètres; Sanderson gives 1.6 millimètres. My own measurement would give rather a higher figure—2 to 3 millimètres. But, from some careful tape measurements, I have found the total circumferential expansion in health not to exceed $\frac{1}{2}$ to $\frac{1}{4}$ inch. It would appear, then—and this is the point to which I wish especially to direct attention—that in health, throughout ordinary inspiration, the limit of thoracic recoil, which we may call the reserve capacity of the thorax, is barely reached; and, therefore, that the sole resistance to be overcome is that of the lungs. I will not dwell upon the beauty of the mechanism, by which our respiration ceaselessly and without conscious toil proceeds from the earliest breath of infancy to old age. The secondary effects of these elastic forces upon the circulation through the heart is also a tempting subject which I must pass by. It is the adaptation of this mechanism to diseased conditions to which I wish now briefly to refer.

The capacity and willingness (if I may so say) of the thorax to enlarge within certain limits, without muscular effort from without or pressure from within, is a most valuable safeguard in some diseases, serving to protect the lungs from pressure during temporary conditions of inflammation or engorgement. If we look at a case of pneumonia, we find that, whereas on the healthy side the play of the chest-wall is natural or increased, on the other side it is annulled. There is no retreat with expiration, and inspiration is checked by pain. On *post mortem* inspection, we find the fine granulations of lymph evenly spread upon the pleural surfaces, showing no sign of pressure. The patient suffers little pain, except when he coughs or attempts to draw a deep breath. In fact, as the inflamed lung increases in bulk, the thoracic wall retreats to the position of inspiration; and it is probable that this reserve capacity of the thorax is not exhausted in any case of pneumonia, so that in no case is the swollen and tender lung compressed.

In temporary engorgement and oedema of the lungs, the consequences would be much more serious were it not for the reserve capacity of the thorax, which protects the organs from compression, save perhaps in very extreme cases. Again, it is only in very extreme cases of emphysema that the excentric thoracic resilience is entirely neutralised. I have directed my attention especially to this point in many cases, and I believe that the lungs are never enlarged so as to be compressed by the ribs.

With regard to emphysema, the loss of the elastic spring of the chest-wall is one of the first important features of the disease. In the normal chest, a deep breath is drawn by first making a deep expiration, and then a deep inspiration. In emphysema, the power of making a

deep expiration is partially or entirely lost; but the whole reserve capacity of the thorax is taken up, and it is in this direction that the trouble of emphysematous people lies. Regarding, as I think we most of us do, the emphysema as in many cases essentially a primary disease of the lungs of a degenerative kind, I think we must observe in the constant traction of the thorax upon the lungs a determining cause of the disease. If, as is often the case in early life, the chest-walls be soft and feeble, they cannot properly expand, and we get a small, narrow, or distorted chest. On the other hand, if the elasticity of the lungs be relaxed by disease, the chest expands gradually to the limits of its reserve capacity.

In dealing with the subject of pleuritic effusions, so far as it concerns the question before us, I must refer to the effects of effusion—1. Upon the position of the heart; 2. Upon the position of the thoracic wall; 3. Upon the position of the diaphragm; 4. Upon the position of the lung. A very brief remark upon each of the headings is all that I shall attempt to-day.

We must premise that we are now dealing with uncomplicated cases of effusion, in which there are no disturbing phenomena of pulmonary consolidation or pleuritic adhesions.

It matters not, save as a mere question of rapidity with which it is poured out, whether the effusion be gaseous or liquid. The heart is immediately displaced *pari passu* with the effusion (as you perceive in the schema, where I produce right pneumothorax by opening stopcock B), and quite independently of any intrathoracic pressure, until the lung has collapsed to one-third of its bulk.

The practical point, and one of considerable clinical importance, is, that you cannot remove the effusion without reinducing the negative pressure, or without admitting air to replace it. By the suction from an aspirator or a syphon, you may remove fluid by overcoming the elastic resistance of the lung and causing it to re-expand as the fluid is withdrawn, and restore the heart to its natural position, as I do now by this aspirator-syringe applied to tube B. But, if a plain trocar be inserted with the view of removing such an effusion, it will not do so until air is admitted to take its place. I have tested this practically by several simple experiments, taking the intrathoracic pressure at different stages of paracentesis; and, after the first few minutes of the escape of fluid, whilst there are still, perhaps, three pints or so of fluid in the chest, the pressure becomes *nil*—the mercurial columns being level.*

The position of the thoracic wall in effusion into the pleura can be equally well shown by this schema. As the lungs recede and their elasticity becomes relaxed, the thorax expands by virtue of its resilience; *i.e.*, in the schema, the mercurial columns become level. This process of expansion is instant in the case of pneumothorax; gradual in that of liquid effusion. It will be remarked that both sides expand simultaneously, but the affected side in slight excess. Beyond this, no further enlargement takes place until the lung is thoroughly collapsed *i.e.*, until two-thirds of the pleura is occupied by fluid. Hence it follows that, up to this very considerable degree of effusion, we can get little or no information from comparative measurements of the two sides of the chest. I have repeatedly found much—two or three pints—of fluid in the pleura, with positive diminution of measurement on the affected side.

On the other hand, in simple cases, a decided increase in the circumference on the affected side is clear evidence of an excessive effusion causing positive intrathoracic pressure.

With regard to the diaphragm, the same thing holds good. We get no depression of the diaphragm beyond that slight downward movement due to its elastic tension being gradually neutralised, until the elasticity of the lungs is completely relaxed and positive intrathoracic pressure commences. Depression of diaphragm is also, then, an urgent signal for paracentesis. Time now fails me; and I will, therefore, only further draw attention to some admirable investigations by Dr. G. M. Garland of Harvard University upon the elasticity of the lungs as it influences their position and the physical signs in pleuritic effusions. (*On Pneumodynamics*, New York, 1878.)

* In a clinical lecture by Dr. W. H. Stone on "Pleural Tension", published in the *Medical Examiner* for January 24th, and in a more recent pamphlet, will also be found some interesting remarks in connection with the subject above treated of.

MEDICAL FEES.—At a meeting of the Nenagh Board of Guardians last week, a communication was received from the Local Government Board for Ireland, fixing the salaries of medical officers attending as witnesses at Petty Sessions at £1 per day in town, and £2 in the country.

PRESENTATION TO MR. E. F. H. BURROUGHS.—On November 7th, a handsome silver kettle and stand were presented, at a largely attended public meeting, to Mr. Burroughs on his leaving Weston-Zoyland. The presentation was followed by a concert.