THE MEANING OF RESPIRATORY FAILURE*

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There is nothing mystical about the concept of respiratory failure today. It is no less understandable, no less measurable, no less assessable by laboratory and clinical means, and no less rationally treated (even if not always successfully) than congestive cardiac failure or renal failure. In all failing organs, normal function can serve as a basis for the estimation of the disorders of physiology that accompany failing function, for the recognition of such disorders, and for their treatment.

The function of respiration is responsible for:

(*i*) The saturation of arterial blood with oxygen.

(*ii*) The elimination of the waste product of metabolism, carbon dioxide.

(*iii*) The elimination of water vapour and heat.

Failure of arterial oxygen saturation results in central cyanosis, failure of elimination of carbon dioxide results in hypercapnia and carbon dioxide narcosis, and failure of elimination of water vapour and heat may result in alarming and sometimes catastrophic rises of body temperature.

Hyperventilation, i.e. the act of over-breathing, may, by increasing alveolar ventilation, compensate for threatening failure, to which state of compensation the term 'respiratory insufficiency' is conventionally applied; and it is important to remember that this hyperventilation maintains respiratory function at a normal level at a cost, namely the extra cost of the work of breathing entailed in the hyperventilatory effort, which uses up sorely needed oxygen.

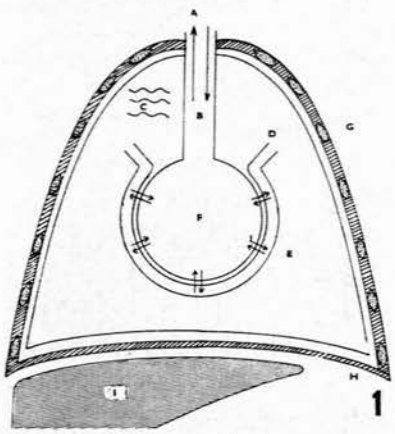

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Fig. 1. Respiratory function ensures (*a*) arterial O saturation, (*b*) CO_2 elimination and (*c*) H_2O and heat elimination.

Muscle and elastic tissue each offer resistance and contribute to contraction. Non-elastic resistance is offered by airways deadspace, chest wall (including fat), and liver mass.

A=ventilation, B=deadspace, C=elastic, D=perfusion, E=diffusion, F=alveoli, G=non-elastic chest wall, H=diaphragm, I=liver.

The maintenance of respiratory function is dependent upon:

(*i*) Ventilation, which conveys heat out and air in and out of the lung.

(*ii*) Circulatory perfusion, which carries blood to and from the functioning unit of the alveolus.

(*iii*) Gaseous diffusion, which transfers oxygen and carbon dioxide across the alveolar membrane.

Failure of one or more of the above mechanisms may lead to respiratory failure.

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The Mechanics of Ventilation

Fig. 1 illustrates diagrammatically the fact that air must be carried to the alveoli through the anatomical deadspace (about 150 ml.), i.e. the space between the lips and the terminal bronchioles, which is physiologically functionless and which offers resistance to the passage of air — a resistance which is grossly increased in bronchospasm, bronchitis and foreign body in the bronchial tree. This airway resistance is an important factor in the genesis of some cases of respiratory failure. It is a form of 'non-elastic' resistance.

Inspiration is effected by the muscles of respiration in the chest wall and the diaphragm, and paralysis of these muscles is another cause of respiratory failure, for example in poliomyelitis. Inspiration has to overcome normal elastic resistance offered by the elastic tissue of the lung, and it also has to overcome non-elastic resistance offered by the structures of the chest wall, for example the ribs and muscles as well as the airways which have already been mentioned. Elastic resistance is increased in diffuse pulmonary fibrosis, and reduced in emphysema, as will be illustrated in Fig. 7. Non-elastic resistance, other than that offered by airways, may be increased in conditions of the chest wall and so impede normal respiratory function. For example, this happens in acute conditions such as trauma with fractured ribs, postoperative immobilization after abdominal and thoracic operations, immobilization associated with pleural pain from any cause, and chronic conditions such as kyphoscoliosis and, most important, obesity as it affects the chest wall and abdomen. We attach great importance to obesity as a cause of increased resistance to respiration.

Expiration is mainly effected by the elastic tissue of the lung. This fails in emphysema, and the lung gradually balloons to assume a permanently increased volume. Expiration, too, is impeded by airway resistance, as in asthma and chest-wall resistance, as specified earlier, and also by hepatomegaly from any cause. The enlargement of the liver in congestive cardiac failure is one of the factors causing cardiac dyspnoea, and in a condition such as Gaucher's disease, in which the liver may be 20% of the total body weight, dyspnoea may have no other obvious cause.

Both inspiration and expiration are inhibited by sedative drugs, which may be a very potent cause of precipitation of acute respiratory failure, and by disease or trauma affecting the respiratory centre.

Perfusion may fail, as in emphysema.

Diffusion may fail, as in pneumonia, massive collapse, and 'alveolar-capillary block' syndromes.

Failure of Ventilatory Function

Failure of ventilatory function is always associated with alveolar hypoventilation, even though there may be a tremendously increased output of energy and apparent hyper-

ventilation by the overaction of the usual and the accessory muscles of respiration in a vain effort to improve alveolar ventilation — a phenomenon seen particularly in severe status asthmaticus. Furthermore, alveolar hypoventilation results in reduction in the amount of heat normally lost by the respiratory route (about 15% of the total heat loss in the normal nude subject), and if to this disability, which in itself is slight, is added a reduction or loss of other heat-losing mechanisms, as may occur when a patient is covered with blankets (thus reducing heat loss from the skin) or in an overheated operating theatre or ward (which reduces heat loss by radiation on account of the high environmental temperature), the body temperature may rise to hyperpyrexial levels, sometimes catastrophically rapidly, with fatal results.

It is clear, therefore, that respiratory failure may occur owing to intrinsic diseases of the respiratory system — emphysema, diffuse fibrosis, pneumonia, massive collapse — and also owing to a number of conditions in which there is no intrinsic lung pathology. In many cases more than one factor plays a part.

Lung Volumes or Capacities

Fig. 2 illustrates some features of lung volumes. On the left of the figure is shown a more or less scale drawing

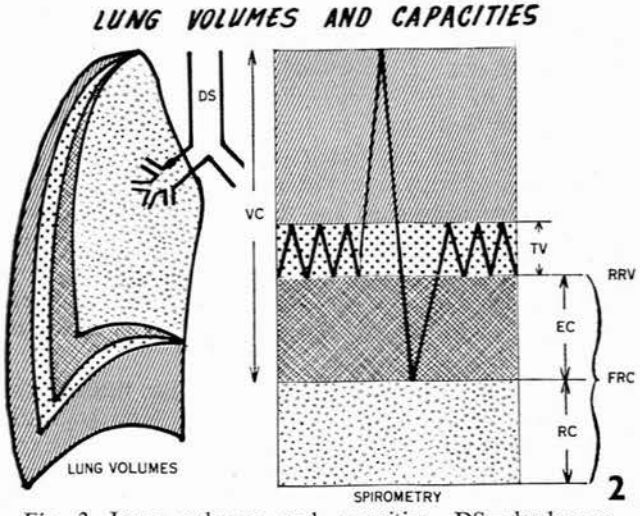

Fig. 2. Lung volumes and capacities. DS=deadspace. VC=vital capacity. TV=tidal volume. RRV=resting respiratory volume. FRC=functional residual capacity, made up by expiratory capacity (EC) and residual capacity (RC).

of the anatomical size of the normal lung in various phases of respiration, and on the right are seen the corresponding spirometry readings, appropriately shaded. The tidal volume is the volume used when sitting quietly at rest; plenty of reserve is left in the normal lung. Then comes deep inspiration to the full capacity of the lung, followed by full expiration to the very end of expiration, the difference being the vital capacity. At the end of expiration there remains a considerable volume of lung that can never be emptied by expiration — the residual capacity — which is measured by special inert gas 'mixing techniques'. The deadspace is shown in the anatomical drawing of the lung and is calculated by formulae — the normal for an adult being about 150 ml.

In Fig. 3 there is added to the spirometry tracing a tracing of a 'maximum breathing capacity', i.e. the maxi-

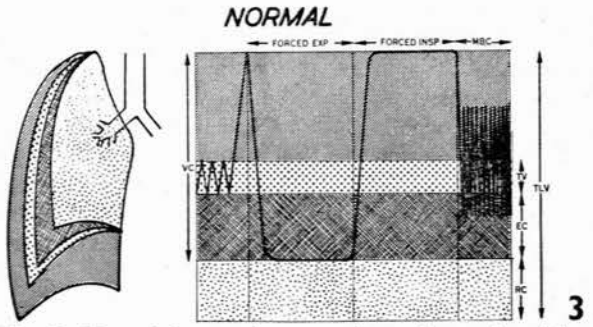

Fig. 3. Normal lung volumes and maximum breathing capacity (MBC). TLV=total lung volume.

mum volume of air that can be moved in and out of the lungs, using maximum inspiratory and expiratory effort. The effort is recorded for 15 seconds and then, by convention, calculated as volume of air moved per minute. This is a useful test, but, of course, it tests a number of lung functions. A normal test is dependent on the strength and efficiency of the respiratory muscles, upon the resistance of the chest wall, upon the elastic power and elastic resistance of the lungs, and upon the airway resistance which may be grossly increased, for example, in bronchospasm. The test, therefore, reflects the overall capacity of the subject to move air in and out of the lungs, and as such it is a useful test, but other tests are required to break down the result into the various causal components.

What happens to the lung volumes in abnormal conditions?

In Fig. 4 are shown the findings in diffuse pulmonary fibrosis. Both in the drawing of the lung and in the

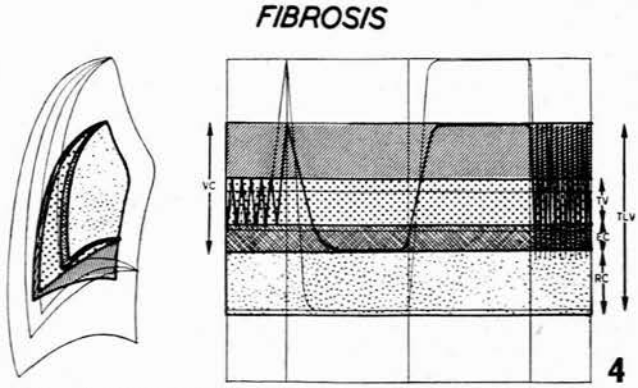

Fig. 4. Lung volumes and maximum breathing capacity in pulmonary fibrosis.

spirometry tracings the faint outline of the normal findings, as shown in Fig. 3, appear for comparison. It is noted that the total lung volume is small, therefore the reserves are low, the tidal volume is about normal, and maximum breathing capacity is near normal, but closely approximates the use of the full vital capacity, which is greatly reduced.

In Fig. 5 are shown the findings in emphysema. While the total lung volume is greatly increased, the tidal volume,

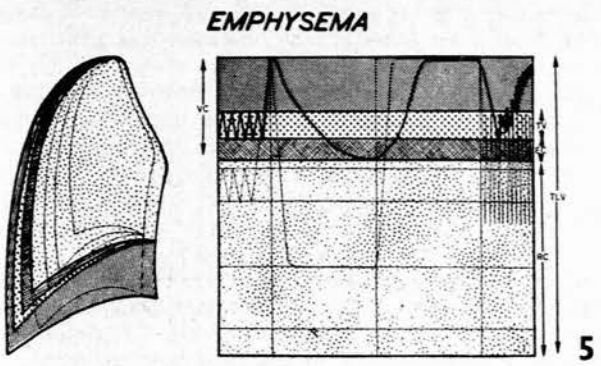

Fig. 5. Lung volumes and maximum breathing capacity in emphysema.

vital capacity, and maximum breathing capacity are all greatly decreased, since the lungs are in a permanently distended state. There is a very large residual capacity and very little room for ventilatory movement. Because of the reduced elastic tissue, which is characteristic of emphysema and which therefore inhibits the pushing out of air, and because of the frequently associated bronchospasm, which offers non-elastic airway resistance, 'air trapping' occurs during the maximum breathing capacity test. Lung function is clearly gravely impeded and readily fails with the additional strain of such conditions as bronchospasm, pneumonia, massive collapse from bronchial blockage by viscid secretions of bronchitis, over-sedation, etc.

The Work of Breathing

Increased work, as was pointed out above, may compensate for reduction of respiratory function, but, as was also pointed out in an earlier paragraph, the increased work may be quite ineffective in its efforts to increase alveolar ventilation, and these vain efforts use up much-needed oxygen. Increased work of breathing is one of the factors causing the subjective sensation and the objective sign of dyspnoea.

It is a matter of elementary mathematics and physics that when a specified amount of force is applied to an object to move it through a certain distance, the *work* done is the product of the *force* and the *distance*, as shown in the formula:

Work = force × distance.

Translating this simple physical principle to the work of breathing, *force* can be substituted by *intrathoracic pressure*, which is a force and which is measurable by intraoesophageal balloon techniques, and *distance* can be substituted by the *volume* of air moved in and out of the lungs in one inspiratory and expiratory cycle of respiration, measured by spirometry techniques. The *work of breathing* can be estimated by the formula:

Work of breathing = pressure × volume.

The bellows simile, illustrated in Fig. 6, is useful for understanding the concept of the 'work of breathing'. The spring, A, represents elastic force and resistance; the bellows fabric, B, represents non-elastic resistance excluding that caused by the airways; and the nozzle, C, represents non-elastic resistance arising from deadspace airways. The hands applied to work the bellows represent the power of the muscles of respiration during inspiration. On the left

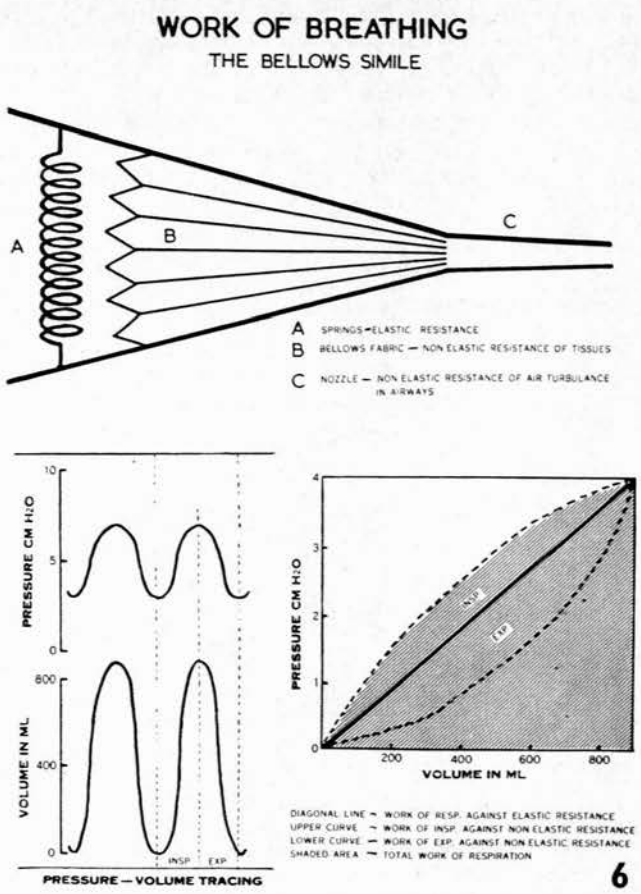

Fig. 6. Work of breathing — the bellows simile.

is shown a 'pressure-volume' tracing, and on the right the same tracing translated to graphic form. From the pressure-volume tracing it is seen that for an intrathoracic pressure change of 4 cm. of water, 900 ml. of air are moved in and out of the lung. The pressure changes are represented on the vertical line of the graph, and the volume changes on the horizontal line. The straight diagonal line in the graph represents the linear relationship that exists between volume change and pressure change, if the volume change takes place against elastic resistance and relaxation only. In the case of the lung, there is however non-elastic resistance as well, and the effect of the pressure change on the sum of the elastic and non-elastic resistance results in an upwardly curved line for inspiration and a downwardly curved line for expiration. At the beginning of inspiration, greater pressure is required to produce a given volume change, and as inspiration progresses, relatively less pressure is required to produce the same volume change. On expiration, the first phase requires a greater fall of pressure to produce a given volume change; and in the later phases, less pressure change results in the same given amount of volume change. The net result is an elliptical 'hoop' which represents the effect of the elastic and non-elastic resistance. The greater the elastic factor, absolute or relative, the narrower the 'hoop' and the nearer the 'hoop' approximates to a straight line; and the more the non-elastic factor,

the more eggshaped the 'hoop'. The total shaded area in the figure can be proved mathematically to represent the total amount of work done in the inspiratory and expiratory phases of one respiration.

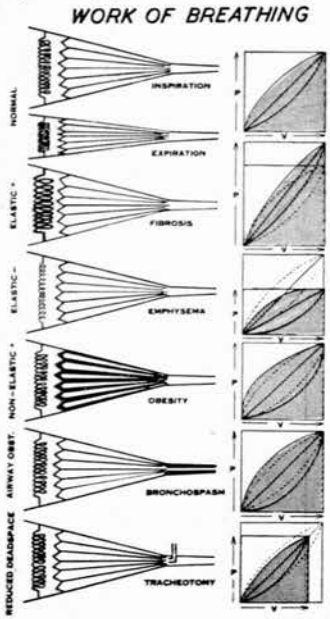

Fig. 7. Pressure-volume graphs for fibrosis, emphysema, obesity, bronchospasm and tracheotomy are each shown on a light background drawing of the normal. P=pressure in centimetres of water. V=volume of air in millilitres.

Fig. 7 illustrates what happens to the amount of the work of breathing in certain abnormal states. The figure shows a series of bellows similes representing different pathological states, with corresponding pressure-volume curves. For simplicity, the volume of air moved per respiration is the same in all examples, namely 900 ml., with the exception of the 'tracheotomy' example in which there must obviously be a smaller volume to move because of the exclusion of the nozzle 'deadspace'. In addition, for simplicity, only one changing factor is shown in each diagram. This obviously cannot apply in life, since in all cases multiple factors play a part.

The first two bellows similes represent normal inspiration and expiration with the normal pressure-volume curve, as in Fig. 6.

A heavy thick spring represents increased elastic resistance such as may occur in diffuse pulmonary fibrosis, other resistances in the diagram remaining normal. The 'hoop' flattens out towards becoming a straight line, since the elastic factor alone, uncomplicated by non-elastic factors, produces a straight pressure-volume line. But the pressure change associated with the movement of the standard 900 ml. of air is greater; therefore, the 'hoop' is steeper. The total shaded area indicates that the total work of one respiration is greater than normal.

A thin light spring represents reduced elastic resistance, as in emphysema. Because the non-elastic resistance factor dominates, the 'hoop' becomes more ovate; because the elastic resistance is less than normal, less pressure change moves a given amount of air, and the slope of the 'hoop' becomes flatter. The total work of one respiration is actually less than normal in this example, but it must yet again be emphasized that in emphysema the advantage of there being less elastic resistance is more than offset by a number of disadvantageous factors.

A normal spring with thickened bellows fabric representing non-elastic tissue resistance increases the work needed to overcome non-elastic resistance, and the result is a more ovate 'hoop' and a greater than normal amount of total work for one respiration. This situation arises, e.g. in the very common condition of obesity.

A narrowed nozzle represents bronchospasm, which causes increased non-elastic resistance and results in a curve much the same as that seen in other causes of increased non-elastic resistance such as obesity, kyphoscoliosis, or fractured ribs.

Lastly, if the length of the nozzle is excluded by inserting a 'tracheotomy' tube near its attachment to the bellows fabric, there is much less non-elastic resistance from airway deadspace, and the volume of air to be moved during each respiration is less by the volume of the end of the nozzle. The pressure-volume curve shows the lesser volume of air moved, and the 'hoop' is narrowed since elastic resistance now plays a relatively greater role in total resistance. The total work of one respiration, as shown by the total shaded area, is clearly less than normal. Tracheotomy, in other words, reduces the work of breathing.

The concept of the work of breathing is a most important one, since an increase uses more oxygen without necessarily increasing ventilation. Increased work is accompanied by dyspnoea; reduction of work reduces dyspnoea.

CONCLUSION

An understanding of the mechanics of breathing enables us to appreciate the rationale of the clinical and laboratory recognition of respiratory failure and of its treatment.

The basic work on which this paper is based, was carried out in the Cardio-pulmonary Unit of the Council for Scientific and Industrial Research in the Department of Medicine of the University of the Witwatersrand and the Johannesburg General Hospital. Thanks are due to the photographic unit of the Department of Medicine for making the diagrams.