The purpose of breathing is to ventilate the alveoli efficiently, but this cannot be accomplished unless the rate and depth of respiration are variable within wide limits. In normal lungs the limits between which the minute ventilation can vary are very wide, but in certain diseases the maximum voluntary minute ventilation may be grossly reduced.

This paper is a brief commentary on some of the results which have been obtained from studies on the mechanical properties of the lungs. The references quoted are to a few papers which illustrate the physiological principles discussed.

**Factors producing movement of the lungs**

At the end of a normal expiration the lungs are in a state of partial inflation. They are held in this condition as the result of the operation of two opposing forces, namely, the elastic recoil of the lungs tending to collapse them and the resistance of the chest wall opposing further passive movement. The elastic recoil of the lungs can readily be shown to exist by the introduction of air into the pleural space when the lung parenchyma shrinks away from the chest wall.

During inspiration, an outwardly directed force is applied to the lung surfaces. This overcomes the elastic and viscous forces (cf. below) opposing expansion of the lungs, and inspiration occurs. The inflating force applied to the pleura can be measured directly in the pleural space or by measuring the intra-oesophageal pressure (Dornhorst and Leathart, 1952), which is a measure of the intrapleural pressure. Expiration is a passive process.

Campbell (1955) made an electromyographic study of the behaviour of the intercostal muscles in man during quiet breathing and during slight hyperventilation. The lower intercostal muscles contracted during inspiration, but were relaxed during expiration in quiet breathing as well as during hyperpnoea. The evidence concerning the activity of the upper intercostal muscles was not clear, probably because there was considerable technical difficulty in obtaining adequate records from them. Campbell and Green (1955) have studied, similarly, the behaviour of the abdominal muscles. The pattern in this case was complex as it was related to posture as well as to the phases of respiration. In general the muscles were less active during inspiration than during expiration, and, the greater the minute ventilation, the greater was this difference in activity.

**Factors opposing movement of the lungs**

To understand the nature of the factors which oppose inspiration the model described by Christie (1953) is of value (fig. 1). When the bellows are expanded (inspiration) the spring is stretched and work is needed to accomplish this. This is exactly analogous to the work required during inspiration to overcome the elastic properties of the lung. In the model the material of which the bellows are made resists movement because of its viscosity. This has its counterpart in the viscosity of the lung tissue. During inflation of the bellows air flows through the nozzle and its flow is resisted, as is the flow of all fluids through tubes. This resistance is analogous to that which hinders the passage of air along the airways of the lungs.
During expiration the lungs become progressively less distended and so the elastic work done during inspiration becomes available to assist expiration. The viscous resistance of the lung tissue to deflation and the viscous resistance due to airflow through the lung airways operate as in inspiration.

Otis, Fenn and Rahn (1950) have shown that when a subject is ventilated artificially in Drinker's machine, the work involved in moving the lungs and chest wall is about 0.4 kg.m per minute. Of this, about 60 per cent is expended in overcoming the elastic resistance of the lung and chest wall and 40 per cent in overcoming viscous resistances. Marshall, McIlroy and Christie (1954) have shown that the work of moving the lungs alone is about 0.3 kg.m per minute. Of this, 70 per cent is used in overcoming the elastic resistance of the lung and 30 per cent in overcoming viscous resistances.

The laws governing the flow of gases or fluids through tubes have been known for many years. For any given pressure difference between the ends of a tube the volume of air which passes through the tube in one second depends upon its length and diameter. By halving the diameter the volume of flow per second is reduced to one-sixteenth, whereas, by doubling the length, the volume of flow per second is only halved. These statements are true only under certain conditions of rate of flow and tube geometry which result in the flow being streamlined or laminar. The movement of the gas through the tube under these conditions may be regarded as the telescopic sliding of adjacent concentric layers of gas over one another without mixing. In any one tube, laminar flow is more likely to occur when the velocity of the gas is slow. When the rate of airflow is increased, a stage is reached at which the streamlined flow becomes unstable, and, at even faster rates of flow, turbulent. In streamlined and in turbulent flow two different mathematical expressions relate flow to pressure and tube geometry.

All this basic information has been obtained in laboratory experiments made under conditions of constant flow through smooth straight tubes. The question arises as to whether these physical laws, determined under carefully controlled conditions, can be applied to the airflow through the respiratory passages. During breathing the rate of airflow changes from moment to moment, and the inside of the bronchi and trachea cannot be regarded as smooth. Besides, the air passages branch and alter in diameter in a more or less unknown manner. It is true that a great deal is known about the branchings of the larger airways, but no details could ever become available about the narrower tubes. We are justified in assuming, however, that the physics of gas flow through the air passages of the lungs must be very complex and that the flow of gas is resisted by the narrowness of the tubes.

The work done in moving the lungs during each respiratory cycle is the direct result of the viscous hindrance offered by the tissue of the lungs to inflation and deflation and by the flow of gas along the airways. If the lungs and airways offered no viscous hindrance to inflation and deflation, but were merely perfectly elastic, the simultaneous measurement of intra-oesophageal pressure (which is equivalent to intra-pleural pressure) and tidal volume would show that the most "negative" intra-oesophageal pressure was observed at the summit of inspiration and the least "negative" pressure at the end of expiration. Such, however, is not the case. Figure 2 shows that the intra-oesophageal pressure and tidal volume are a little out of step. The greater this distance, the greater is the amount of work performed during a respiratory cycle.

![Figure 2](https://example.com/figure2.png)

Simultaneous tracings of intra-oesophageal pressure and tidal volume (above) and the pressure-volume diagram corresponding to one breath (Modified from Marshall et al. (1954).)
Simultaneous tracings of pressure and volume (fig. 2) enable a pressure-volume diagram to be constructed. This relates the changes in intraoesophageal pressure to the simultaneous change in the volume inspired or expired. The diagram resembles an ellipse. Had the lungs been perfectly elastic and without viscous hindrance, the pressure-volume diagram would have been represented by the major axis of the ellipse. As things are, during inspiration, work (i.e. pressure \times volume) is required to distend the elastic lungs. The amount of "elastic work" done in inspiration is represented by the area of the triangle OAN. In addition to the "elastic work" done in inspiration, work is done against viscous resistances and this is represented by the area OIA.

During expiration the energy stored in the lungs as a result of the work done during inspiration against elastic resistance is liberated and is more than sufficient to overcome the viscous hindrance to expiration. The work done against the viscous hindrance to expiration is given by the area OEA, which is clearly less than the "elastic work" (area OAN) recovered from the deflating lungs.

The total work done in one respiratory cycle against viscous resistances is therefore represented by the area OIAE.

The changes in the amount of work necessary to move the lungs have been studied in a number of conditions. Marshall, Mcllroy and Christie (1954) and Mcllroy and Christie (1954), for example, have applied this method to the study of patients with mitral stenosis and emphysema. In patients with heart failure, dyspnoea and orthopnoea are associated with a substantially greater amount of work than normal because the physical properties of the lungs are changed. Christie (1953) has reported in summary the interesting finding that the rate and depth of breathing are adjusted at rest or during exercise, so that the subject performs the minimum amount of work in moving the lungs by automatic adjustment of the rate and depth of breathing.

**THE SUBDIVISIONS OF THE LUNG CAPACITY**

The most commonly measured subdivision of the lung capacity is the vital capacity which was first studied by Hutchinson after he invented the spirometer. It soon became known that the vital capacity of normal subjects varied widely and that in certain diseases the maximum amount of air which could be drawn into the lungs and then expelled was small. We now know that after a maximal expiration a considerable volume of air remains in the lungs.

If a spirometric tracing of quiet breathing is made, it will be found that the depth of successive respirations is approximately the same (fig. 3); this is the *tidal volume*. The lower level of such a tracing, which represents the limit of normal expiration, is fairly constant and is referred to as the *resting respiratory (or end-expiratory) level*.

![Diagrammatic representation of a spirometer tracing showing vital capacity (V.C), inspiratory capacity (I.C.), tidal volume (T.V.), expiratory reserve volume (E.R.V.), and resting respiratory level (R.R.L.)*](https://academic.oup.com/bja/article-abstract/28/12/536/289674)

The maximal volume of air which can be drawn into the lungs from the resting respiratory level is called the *inspiratory capacity*. The volume of air which can be expired after a normal expiration is called the *expiratory reserve volume*. The air which remains in the lungs after a maximal voluntary expiration is called the *residual capacity*, which is not shown in figure 3. The vital capacity is the sum of the inspiratory capacity and the expiratory reserve volume. The *total lung capacity* is the sum of the vital capacity and the residual capacity. The *functional residual capacity* is the...
volume occupied by the expiratory reserve volume and the residual capacity.

The vital capacity is so easy to measure and is affected in so many pulmonary diseases that it has been extensively studied.

A measure of vital capacity can be used in two ways: either (1) to compare the subject's performance on one occasion with that on a subsequent occasion so that he acts as his own control, or (2) to relate the subject's performance to that of comparable normal individuals. Many attempts have been made to find a means of predicting the vital capacity, but none has been entirely successful.

Table I is taken from the work of Hurtado and Fray (1933) (with medical students) and of Aslett, Hart and McMichael (1939) (with coalminers) and shows the difficulty of prediction. They studied the degree of correlation between the vital capacity and certain easily determined body measurements.

The nearer the correlation coefficient is to unity the more closely can the vital capacity be predicted. Thus, the vital capacity had a high degree of correlation (+0.72) with radiological lung volume on inspiration (R.L.V. Insp.) in Hurtado’s series and an appreciably lower correlation (+0.63) in McMichael’s series. Differences are much more marked when the correlation of other pairs of variates are considered. For vital capacity and body surface area, for example, the figures were +0.28 and +0.67, which means that in the former series there was little correlation between these two variates, whereas in the latter correlation was moderately good.

The explanation of these divergent results is not far to seek. The two sets of measurements were made on different samples of men; both groups were clinically “normal,” but their members were quite different in build, occupation and so on. A correlation coefficient between the vital capacity and another variate determined on a group of medical students cannot, therefore, be used to help in predicting the vital capacity of coalminers or any group of individuals other than medical students.

**Table I**

<table>
<thead>
<tr>
<th>Characteristics correlated</th>
<th>Correlation coefficient</th>
</tr>
</thead>
<tbody>
<tr>
<td>V.C. and R.L.V. Insp.</td>
<td>+0.72</td>
</tr>
<tr>
<td>*V.C. and R.L.V.</td>
<td>+0.63</td>
</tr>
<tr>
<td>V.C. and body-height</td>
<td>+0.55</td>
</tr>
<tr>
<td>*V.C. and standing-height</td>
<td>+0.70</td>
</tr>
<tr>
<td>*V.C. and stem-height</td>
<td>+0.78</td>
</tr>
<tr>
<td>V.C. and body-surface area</td>
<td>+0.28</td>
</tr>
<tr>
<td>*V.C. and body-surface area</td>
<td>+0.67</td>
</tr>
</tbody>
</table>

The correlations marked with an asterisk have been taken from the paper by Aslett, Hart and McMichael (1939); the others are from Hurtado and Fray (1933).
volume of the expiratory reserve volume is sub-
tracted from the functional residual capacity the
volume of the residual air can be calculated.

A wide range of “normal” values is found for
the subdivisions of the lung volume (table II).
This means that a suspicion of abnormality is not
aroused unless the volume of one or more of the
subdivisions in a patient is grossly different from
to ventilate their lungs at the rate of 200 or more
litres a minute for short periods of time. Patients
with emphysema, on the other hand, may achieve
a voluntary minute ventilation of no more than
20 litres. Hermannsen introduced the maximum
breathing capacity test to provide a quantitative
measure of differences in the power of voluntary
ventilation.

<table>
<thead>
<tr>
<th>Fraction of lung capacity</th>
<th>Mean from the mean</th>
<th>Standard deviation from the mean</th>
<th>Confidence limits* 67% 95%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vital capacity</td>
<td>78.0 ± 4.3</td>
<td>73.7-82.3 69.4-86.6</td>
<td></td>
</tr>
<tr>
<td>Inspiratory capacity</td>
<td>61.9 ± 5.3</td>
<td>56.6-67.2 51.3-72.5</td>
<td></td>
</tr>
<tr>
<td>Expiratory reserve volume</td>
<td>16.2 ± 4.1</td>
<td>12.1-20.3 8.0-24.4</td>
<td></td>
</tr>
<tr>
<td>Residual capacity</td>
<td>22.0 ± 4.3</td>
<td>17.7-26.3 13.4-30.6</td>
<td></td>
</tr>
</tbody>
</table>

Columns 1, 2, and 3 have been taken from Hurtado and Boller (1933, their table 3).
*Confidence limits: 67%, means that 67% of normal young males would be found to have ratios within the limits shown in the table.

the corresponding mean value obtained from
appropriate normal subjects used as controls. It
is evident, therefore, that measurements of the
subdivisions of the lung volume are, taken alone,
unlikely to reveal small changes in lung pathology.

The following are references to some examples
of groups of individuals who may be used as standards of “normality”: Hurtado and Boller
(1933) and Hurtado and Fray (1933) used medical
students aged 18–30 years; Kaltreider, Fray and
Hyde (1938) used males aged 18–30, males aged
38–63, and females aged 18–34 years. Whitfield,
Waterhouse and Arnott (1950) studied subjects
aged 10–70 years.

**DYNAMIC MEASURES OF LUNG FUNCTION**

**Maximum breathing capacity.**

It is usual when testing the function of any
organ to subject it to some stress and measure
the way in which it stands up to the strain. The
exercise tolerance test and the urea concentra-
tion test are examples. So far as the lungs are
concerned, the interest lies in the minute volume
of ventilation which the subject can call upon
and the efficiency of alveolar ventilation.

It is possible for normal individuals voluntarily
to perform the test he used an ordinary spiro-
meter from which the valves and carbon dioxide
absorbing canister had been removed. Subjects
were instructed to breathe into the apparatus as
deeply as possible and as rapidly as possible for
about 20 seconds, and a tracing of the movements
of the spirometer bell was obtained. The volume
of air, calculated in litres per minute, which the
subject breathed out was referred to as his maxi-
mum breathing capacity (M.B.C.). Experience has
shown that this test is a sensitive index of the
mechanical function of the lungs.

D’Silva and Mendel (1950) reported that
measurements of M.B.C. were not possible with
their group of normal medical students using a
spirometer of conventional pattern, because the
inertia of the instrument was too great. Bernstein,
D’Silva and Mendel (1952) published the design
of a new spirometer which overcomes these
difficulties and found that the M.B.C. of normal
subjects is not reached at respiratory rates below
70 breaths a minute.

Various investigators have used specially
designed valves, through which the subject’s expired air during maximum breathing was passed
into a Douglas bag for a known time. The volume
of gas in the bag was measured and the M.B.C. calculated. The success of the determination by this method largely depends upon the low resistance offered by the valves to the passage through them of rapidly moving gas. They are designed so that the maximal rate of gas flow (which may be well over 300 litres a minute) is hindered as little as possible. The greater the resistance offered by the valves, the poorer is the subject's performance in the test. The disadvantage of the method is that no graphic record of the subject's performance is obtained.

When the M.B.C. test was performed at a controlled respiratory rate, D'Silva and Kazantzis (1954) found that the tidal volume during maximum breathing in normal subjects was a fairly constant fraction of the subject's vital capacity and depended on the rate of breathing. Thus, in a series of 44 subjects breathing at 70 breaths a minute this fraction (called the swept fraction) was 52.2% ± S.D.7.22. At 30 breaths a minute it was 79.5% ± S.D.8.34. The value of relating a subject's performance in the M.B.C. test to his vital capacity is shown by comparing the performance of young normals with that of young patients with ankylosing spondylitis (D'Silva, Freeland, and Kazantzis, 1953). Although the M.B.C. of the two groups was widely different, their swept fractions were the same. This result means that in these patients the ease with which the lungs could be moved was the same as in normal subjects, though the magnitude of the excursion was limited by mechanical factors operating outside the lungs. The results obtained from patients were reproduced in normal subjects in whom the movements of the thoracic cage were restricted by a tightly laced waistcoat.

The prediction formula suggested by Wright and his colleagues (1949) which related a subject's performance in the test to his age and surface area, only approximately represented the performance of 14 normal medical students studied by Mendel (1949). He found that the M.B.C. of the men ranged from 140 to 320 (mean 203) litres a minute, whereas their predicted M.B.C. ranged from 154 to 171 (mean 165) litres a minute. Baldwin, Cournand and Richards (1948) have also published a prediction formula which gave figures for the same group ranging from 120 to 159 (mean 132) litres a minute. It is apparent that it is not possible to predict the M.B.C. of an individual with any useful degree of accuracy from his age and measurements of his physical proportions.

"Fast vital capacity" curves.

If a subject makes a maximal inspiration, and then a record is made of the rate at which this is breathed out when he exerts a maximum of effort, the curves, drawn from the original record and shown in figure 4, are obtained.

Curve A is from a normal subject, B is from a patient. Subject A breathed out his vital capacity volume in one-quarter of the time taken by B, as the curves show. The vertical lines a and b represent those portions of the respective vital capacities which the two subjects breathed out tidally at a respiratory rate of 30 breaths a minute. Subject A breathed out 73 per cent of his vital capacity compared with 32 per cent by B. As their respective vital capacities were 4.36 and 3.15 litres, it is obvious that A's performance in the test was much better than B's.

It might be, therefore, that accurate forecasts of M.B.C. would be possible from a study of "fast vital capacity" curves.

Gaensler (1951) has found that the percentage of the total vital capacity breathed out in the first second of a single maximal breath, exhaled as rapidly as possible, was, on the average, 83 per cent, and that the performances of normal subjects in the fast vital capacity test were closely correlated with their M.B.C.s. It is known, however, that the fast expiratory and the fast inspiratory curves are of quite different shape; Gaensler's method of prediction neglects the fast inspiratory curve. It is necessary, therefore, to extend Gaensler's observations to take into account both the fast inspiratory and the fast expiratory curves.

If a record of maximum breathing is made on a fast moving drum the inspiratory and expiratory portions of the tracing can be superimposed upon parts of the fast inspiratory and fast expiratory curves respectively. When the rate of maximum voluntary breathing is increased the tidal air is reduced, but the inspiratory and expiratory portions of the tracing can still be superimposed upon
Two fast expiratory curves (A and B) drawn on an arbitrary time scale; a and b are the tidal volumes of the two subjects breathing as deeply as possible at 30 r.p.m.

CONCLUSION

The stage which has already been reached in research on the mechanical function of the lungs enables one to obtain moderately reliable quantitative data about the function of normal lungs as a whole. First steps in the extension of these investigations to a study of each lung have been possible since bronchspirometry was introduced. So far very little work has been done on the study of single lobes or of even smaller portions of the lung. Should this become possible, then physiological techniques may be expected to provide useful information about the magnitude of the disorders of mechanical function. At present the disorder must be considerable to be revealed by tests of lung function. The solution to the problem seems to lie in parallel studies of normal and diseased lungs. The purely "academic" studies on the former will lead to a more accurate interpretation of the results obtained on patients.

Needless to say, these various tests of pulmonary function can give no information about the
aetiology of lung disease. They may, however, be utilized in animals in which aetiological studies are being made.

REFERENCES


CORRESPONDENCE

THE ADMINISTRATION OF NEOSTIGMINE

Sir,—I welcome Dr. Lawson's report of cardiac arrest following the use of neostigmine (Brit. J. Anaesth., 1956, 28, 336). It once more draws our attention to the dangers of this drug. I believe that neostigmine is administered far too casually by many of us. Like so many faults in anaesthesia, we get away with it most of the time.

Dr. Boulton in his letter (Brit. J. Anaesth., 1956, 28, 449) mentions the value of the pulse rate as an indication of vagal tone present. Based on this observation I have adopted, for the last few years, a very rigid routine in the administration of this drug. It is as follows:

The patient's pre-operative pulse rate is recorded. If muscle tone is considered inadequate at the end of the operation, intravenous atropine sulphate is given, the dose depending upon the pulse rate at this time. If the pulse is below 80 per minute, 1/75–1/50 grain (0.5–1.3 mg) is given; if it is above this rate, 1/100 grain (0.65 mg) is given. The pulse rate is then taken at intervals until the maximum acceleration has been produced. Neostigmine (diluted to 2.5 mg in 5 ml) is injected slowly, in divided doses, until either adequate muscle tone has returned or the pulse rate has fallen to the pre-operative level. Should the muscle tone still be unsatisfactory when the pulse has slowed to this level, a further dose of atropine 1/100 grain (0.65 mg) is given before more neostigmine is administered.

This technique may sound tedious, but I believe it is safe, and, besides preventing the rare tragedy, it cuts down the incidence of salivation and abdominal colic.

I now teach this method to the students.

RONALD GREEN
Royal Free Hospital, London.