DISORDERED PULMONARY FUNCTION IN EMPHYSEMA

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Introduction

The remarkable advances in pulmonary physiology during the last decade have provided new techniques with which to analyse the complex disorders present in lung disease. The increased precision of these new methods has led to the appearance of symbols, equations, graphs and the frightening paraphernalia of mathematics. The main aim of this article is to show how these measurements have improved our understanding of emphysema by discussing the data obtained from one patient who has been extensively studied. Although the various disorders have been separated into groups for the purposes of discussion it is important to realize that they are all interrelated.

A complete assessment of emphysema (or any other lung disease) requires many measurements. However, as so often happens, improved physiological knowledge has led to improved ability to interpret clinical evidence. Therefore, at the end, I have suggested a simple scheme for the evaluation of emphysema making use of clinical information and tests most of which are so simple as to be within the capabilities of any reasonably equipped hospital.

In the various tables and illustrations reasonable normal values for a man of 60 years have been given for comparison. No technical details are given in the text. For the further elucidation of the underlying pulmonary physiology reference should be made to 'The Lung' (Comroe, Forster, Dubois, Briscoe and Carlsen, 1955). References are only given to aspects of the subject not covered in that work.

Clinical Details

The patient (Mr. F.H.T.), a motor engineer, is aged 59, 5 ft. 9 in. tall and 1.8 sq.m. in surface area. He has suffered from increasingly severe dyspnoea for 33 years. There is no history of longstanding cough or sputum preceding the onset of dyspnoea. He has been increasingly incapacitated for the last seven years and before admission could only walk 25 yards. He has continued to work by driving his car from the door

of his house to the desk in the garage where he is employed. Climbing the 14 stairs in his house causes intense distress lasting 10 to 15 minutes and often forcing him to kneel on the stairs. He has not walked out of the front gate of his house for ten years nor had a pair of shoes repaired for seven years. He has great difficulty in dressing himself and in eating his food. In recent years he has had a variable amount of mucoid sputum which causes bouts of coughing leading to intense dyspnoea, and interrupting his sleep every few hours. He sleeps more comfortably lying flat than when propped up. Clinically and radiologically he has the changes of severe emphysema. He is cyanosed at rest and becomes very cyanosed on slight exertion. There is no clinical evidence of heart failure. On screening the right ventricle is moderately enlarged and the main pulmonary arteries are prominent. E.C.G. shows an incomplete right bundle branch block. Repeated bacteriological examination of the sputum has not shown any pathogenic organism to be persistently present. The Hb concentration is 19 g. per 100 ml. and the P.C.V. 63 per cent.

By way of introduction some personal data and the results of some simple well-known pulmonary function tests performed on him are shown in Table 1.

TABLE 1

	Mr. F.H.T.	Normal
Age (year)	59	
Height (in.)	69	
Weight (lb.)	135	
Surface area (sq. m.)	1.8	
Vital capacity (L./B.T.P.)	2.4	4
Forced exp. vol. in 1 sec. (L./B.T.P.)	0.75	3.2
Max. vol. ventilation (M.B.C.) (L./min.)	24	> 100
Arterial CO ₂ tension (mm. Hg.)	58	40
Arterial O ₂ saturation (per cent.)	82	> 95

Evidence of Emphysema

The lungs in emphysema are voluminous, inelastic and there is destruction of alveoli, alveolar walls and lobular septa, and of pulmonary capillaries. This sentence briefly summarizes the

pathological description of the condition which, in the absence of knowledge of a specific lesion, is the only way in which the disease can be identified. The first physiological studies to be described are those which satisfy these three criteria: increase in volume, inelasticity, loss of functioning lung surface.

TABLE 2
LUNG VOLUMES

	Mr. F.H.T.	Normal
Total lung capacity, L./B.T.P.	10.3	7.0
Residual volume	7.9	3.0
Vital capacity	2.4	4.0
Inspiratory capacity	1.7	3.0
Functional residual capacity ..	8.6	4.0

FIG. 1.—The lung volumes and the forced vital capacity. V.C., vital capacity; F.E.V.₁ forced expired volume in 1 sec.

The Volume of the Lungs (Table 2 and Fig. 1)

There is increase in the resting volume of the lungs (the Functional Residual Capacity, F.R.C.). There is also increase in the volume of air which cannot be expelled by a voluntary expiratory effort so that this residual volume represents 77 per cent. of the total lung capacity. At the age of 60 the residual volume should only be 45 per cent. of total lung capacity.

The Elastic Properties

Before considering the elastic properties of the lungs the term ‘elasticity’ must be discussed because as commonly used it has not the same meaning as it has to the physicist or physiologist. Strictly, elasticity is the property of a substance or

structure by virtue of which it returns to its original shape and dimensions when a deforming force has been removed. Thus a rubber balloon is elastic if after it has been inflated it will empty itself to its original volume. If it remains partially inflated then it has partially lost its elasticity. No unit of measurement is employed. If a physicist wishes to describe how much a substance will change its dimensions in response to a deforming force he determines the modulus of elasticity which relates stress to strain. When a physiologist wants to describe the distensibility of the lungs he measures their compliance, that is the change in volume (the strain) produced by a given change in pressure (the stress). In Fig. 2 the

FIG. 2.—The elastic properties of the lungs. This diagram relates the volume of the lungs to their elastic recoil pressure as measured in the pleura or oesophagus.

volume of the lungs is plotted against the oesophageal pressure. (The oesophageal pressure closely resembles intrapleural pressure and therefore may be used to study the pressure developed when the lungs are distended.) It can be seen firstly, that when the oesophageal pressure is atmospheric (that is when the lungs are relaxed) their volume is 8 l. instead of 3 l. That is to say they have partially lost their elasticity. Secondly, for any given change in oesophageal pressure the lungs change in volume more than they ought to. Their compliance (900 ml./cm. H₂O) is greater than normal (250 ml./cm.). Conversely, for any given volume of distension they exert less recoil pressure (1.1 cm. H₂O/l. instead of 4.0 cm. H₂O/l.).

The Area and Permeability of the Alveolar-capillary Membrane

The volume of O_2 or CO_2 which diffuses across the alveolar-capillary membrane per minute depends upon the following factors: the difference in the partial pressure of the gas between the alveolar air and the pulmonary capillary blood; the solubility of the gas; the area and thickness of the membranes and fluid barriers between the alveolar air and the red blood corpuscles; the rate of certain physico-chemical processes within the red cell. Of these factors the most important physiologically is the solubility of the gas. CO_2 is much more soluble than O_2 and consequently diffuses 20 times more rapidly. The important consequence of this difference between the two gases is that disease processes which destroy the alveolar-capillary surface or make it less permeable have a much greater effect on O_2 uptake by the blood than on CO_2 output. So great is the difference, in fact, that O_2 uptake becomes inadequate to support life before a significant CO_2 pressure difference between the pulmonary capillary blood and the alveolar air appears.

In an attempt to study changes in the alveolar-capillary membrane physiologists measure the oxygen diffusing capacity, which is defined as the volume of O_2 which crosses from the alveolar air to the pulmonary capillary blood per unit pressure gradient per unit time. The units of diffusing capacity are therefore ml. of O_2 /mm. Hg./min.

The O_2 diffusing capacity (DO_2) is difficult to measure for a number of physiological and technical reasons. Therefore carbon monoxide is now widely used for the measurement of the diffusing capacity (Dco) because its diffusion properties are similar to those of O_2 , and because its affinity for haemoglobin removes some of the difficulties.

Apart from disease the diffusing capacity can be affected by other factors notably physical exercise which raises it by increasing the number of functioning pulmonary capillaries. It is customary, therefore, to specify the level of activity at which the measurement was made. The conditions of most clinical interest are the resting diffusing capacity and the maximum diffusing capacity.

In Table 3 it can be seen that the diffusing capacity measured by three different methods was found to be very low. If we take the value for the maximum diffusing capacity we can calculate the significance of this measurement. The DO_2 (Max) is 8 ml. O_2 per min. per mm. Hg. tension difference between the alveolar air and the pulmonary capillary blood. If the alveolar O_2 tension was normal (about 100 mm. Hg.) and the mean O_2 tension in the pulmonary capillaries was 0 mm. Hg. (which is impossible) the greatest

TABLE 3
THE DIFFUSING CAPACITY

	Mr. F.H.T.	Normal
Resting diffusing capacity, ml./CO/ min. mm. Hg.:		
CO (single breath method) ..	2.7	15
CO (steady state method) ..	4.2	15
Maximal diffusing capacity (O_2 method), ml./ O_2 /min. mm. Hg. ..	8	40

The single breath Dco was measured by the method of Ogilvie *et al.* (1957). The steady state Dco was measured by Method II of Bates *et al.* (1955). The DO_2 was measured by Riley's method. The values obtained for Dco and DO_2 are not directly comparable because CO and O_2 have different physical properties. They can be approximately compared by using the following ratio:

$$DO_2 = 1.3 \times Dco$$

volume of O_2 that could be taken up by his lungs would be 800 ml./min. In fact, as his alveolar PO_2 is about 80 mm. Hg. and the mean pulmonary capillary PO_2 is about 15 mm. Hg. on exercise, then the maximum O_2 uptake he can achieve is only about 520 ml./min. Such a severe reduction in diffusing capacity is unusual.

The Mechanics of Breathing

Evidence has already been presented that the lungs have partially lost their elasticity and have become abnormally distensible. These changes, together with changes in the walls of the airways, are probably of dominant importance in most cases of emphysema.

Airway Obstruction

It is well known that patients with emphysema have difficulty in expelling the air during expiration. In Fig. 1 it can be seen that the volume of air expelled by Mr. F.H.T. in 1 sec. (Forced Expired Volume in 1 sec., F.E.V.₁) is only 0.75 instead of 3.2 l. A normal subject expels all his vital capacity in less than 4 secs., whereas expiration was still proceeding after 8 sec. in the emphysematous patient when he had to give up and take another breath.

Expiratory obstruction in emphysema is usually attributed to 'bronchospasm' or other processes in the walls of the airways, whereas in fact most of the airway obstruction is due to loss of elasticity and there may be relatively little organic narrowing of the airways.

The resistance of the respiratory passages to airflow can be estimated by measuring the pressure difference between the mouth and the alveolar air and dividing it by the rate of airflow at the instant the measurement was made. The value obtained is called the flow, non-elastic, or viscous resistance.

TABLE 4
THE MECHANICAL PROPERTIES OF THE LUNGS

	Mr. F.H.T.	Normal
Lung compliance (zero frequency), ml./cm. H ₂ O ..	900	250
Elastic resistance of lungs (elastance), cm. H ₂ O/l:		
During slow inspiration	1.1	4.0
Breathing frequency 19 per min.	7.9	4.0
Non-elastic Resistance (flow—or viscous—resistance), cm. H ₂ O/l./sec.:		
Interrupter method	3.7	< 3.0
Oesophageal pressure method	4.7	< 2.5
Maximum effective intrathoracic pressure, cm. H ₂ O ..	2	> 25

The units are usually cm. H₂O/l./sec. [There are several methods by which one can attempt to measure the pressure of the air in the alveoli (see Comroe *et al.*, chap. 7). There are theoretical and technical difficulties about each of them, but these difficulties detract little from their value in clinical studies of chronic lung disease.] In Table 4 the resistance to airflow *during inspiration* is shown to be less than twice the normal value whereas in asthma the flow resistance is usually five to ten times the normal value. The flow resistance was measured during inspiration because during expiration the airways are narrowed by external pressure and the flow resistance measured under these conditions is very variable and does not represent the resistance due to organic narrowing of the airways. In most patients with emphysema the resistance to airflow during inspiration is increased, presumably because there is associated bronchitis. This increase in resistance is however, small compared with that occurring in asthma and may, as in the present case, be relatively slight. The obstruction to expiration in emphysema is not due to 'bronchospasm' but to the loss of lung elastic recoil. Expiration during natural breathing both at rest and on exercise is produced by the passive recoil of the lungs (Campbell, 1958). The recoil pressure of the lungs, as it drives the air up the airways, maintains the pressure in them above that in the surrounding intrapleural space. This mechanism is sufficient for all physiological levels of ventilation in health but not in emphysema. In emphysema the elastic recoil of the lungs is largely lost. If an attempt is made to accelerate the rate of airflow by using expiratory muscles then the intrapleural pressure narrows the airways as well as increasing alveolar pressure. The flow resistance may then rise to very high levels. In the present case it exceeded 80 cm. H₂O/l./sec. during a forced expiration. One can measure the level of intrapleural pressure at which the airways are so compressed that their resistance is greatly increased to the point at which no further increase in flow rate occurs with increasing effort. This

maximum effective intrathoracic pressure (Campbell, Martin and Riley, 1957) in normal subjects is approximately 30 cm. H₂O. In Mr. F.H.T. it is 2 cm. H₂O.

These factors which cause obstruction to expiration also impair the efficiency of coughing. The linear velocity of the airflow in the trachea of Mr. F.H.T. during coughing is probably about 100 miles per hour instead of about 600 m.p.h.

The Mechanics of Breathing and the Maximum Breathing Capacity and Forced Vital Capacity Tests

A patient's performance in these very useful tests of ventilatory function is limited by the flow resistance of his airways during expiration. Unfortunately these tests do not distinguish between the mechanisms which may be responsible for the resistance. Thus results may be obtained in a patient during an attack of asthma, whose lungs are not affected by emphysema, which are similar to those found in a patient with airway obstruction due to loss of elastic support in whom there is little 'bronchospasm.'

It is to be hoped that the application of more refined techniques will enable the influence of the different mechanisms to be separately evaluated in individual patients.

The Mechanics of Breathing and the Distribution of Ventilation

The respiratory exchanges between the alveolar air and the pulmonary capillary blood should ideally take place with the minimum of ventilation and pulmonary capillary blood flow and with the maintenance of normal arterial blood composition. If perfection is to be achieved, the distribution of ventilation relative to blood flow must be the same in all parts of the lungs. To take an extreme example, the ventilation of one lung and the perfusion of the other will not result in adequate gaseous exchange whatever the volume of ventilation or pulmonary blood flow. Similar reasoning applies at the alveolar level. If every alternate alveolus is overventilated and every other one is

FIG. 3A

FIG. 3B

FIG. 3.—The mechanical properties of the lungs and the distribution of ventilation.

overperfused then the arterial blood will not be normal. In Fig. 3a a simple situation is shown diagrammatically to illustrate how the mechanical properties of the lungs affect the distribution of air. Two 'lung units' (e.g. alveoli, lobules, lobes) are shown which are assumed to have equal volume (say, 10 c.mm.). One of these (A) is less distensible than the other so that when the pressure around them is lowered to -5 cm. H_2O it enlarges to 15 c.mm. while the other (B) enlarges to 20 c.mm.

Therefore, if they both have the same capillary blood flow, then B will be overventilated relative to A. Of course, compensation can occur by adjusting the capillary blood flow so that more goes to B than to A. However, this compensation may be difficult to achieve particularly if the following factors are also considered. The airway leading to A is shown to be wide and offering little resistance to airflow whereas that to B is

shown to be narrow. In Fig. 3b the effect of this difference on the ventilation of A and B is considered. It is assumed that at point X the pressure round them is suddenly reduced to -5 cm. H_2O . Air will rapidly enter A which enlarges to 15 c.mm. Air enters B more slowly and it does not reach its final volume of 20 c.mm. for some time. If the surrounding pressure returned to its resting value at point Y then A would have been more ventilated than B, whereas if it did not return until Z, then B would have been more ventilated than A. Thus, at a high rate of breathing A would be more ventilated than B, and at a low rate of breathing B would be more ventilated than A.

The effect of the rate of breathing on the distribution of ventilation cannot yet be shown directly but, following the more advanced reasoning of Otis *et al.* (1956), we can strongly suspect that it exists in Mr. F.H.T. because the apparent elastic recoil pressure exerted by his lungs increases at higher rates of breathing. [In Table 4 it can be seen that the apparent elastic resistance when breathing at 19 per minute in a normal subject is the same as the true static elastic resistance; whereas in Mr. F.H.T. the apparent elastic resistance of the lungs at 19 breaths per min. was 7.9 cm. $H_2O/l.$ compared with a true elastic resistance of 1.1 cm. $H_2O/l.$ This difference is explained by the fact that there is a true cessation of airflow throughout the lungs of the normal subject at the end of inspiration, whatever the rate of breathing. In the patient with emphysema, on the other hand, although airflow through the mouth stops at the end of inspiration there are still pressure differences between different parts of the lungs which cause airflow to continue within the lungs themselves. These factors cause the pressure difference between the pleura and the mouth to be greater than it is under truly static conditions.]

The Distribution of Ventilation and Pulmonary Blood Flow

If all alveoli receive the same amounts of air and of blood then the concentration of the gases in all the alveoli will be the same. Even in normal subjects these perfect conditions of distribution are not achieved and in emphysema distribution is far from perfect. This imperfection can be analysed by using the concept of 'ideal' alveolar air.

In any subject with a given alveolar ventilation and a given pulmonary capillary blood flow with mixed venous blood of a given composition, the composition of the air in each alveolus will vary depending upon the ratio of ventilation to blood flow. The 'ideal' alveolar air composition for

TABLE 5
DISTRIBUTION OF VENTILATION AND PULMONARY BLOOD FLOW

	Mr. F.H.T.	Normal
Physiological dead space, ml.	330	160
Physiological dead space as per cent. of tidal volume . .	66	30
Alveolar-arterial O ₂ tension difference, mm. Hg. . .	50	< 10
Venous-admixture or 'shunt effect,' per cent. of cardiac output	30	< 5
Mixing efficiency, per cent.	44	> 76
Alveolar N ₂ per cent. after 7 min. O ₂ breathing, per cent.	6.5	< 2.5

any subject under given conditions is that composition which every alveolus would have if they all had the same ventilation in relation to blood flow. Alveoli which have too great a ventilation will have a lower CO₂ tension and a higher O₂ tension than the 'ideal' value. Alveoli which have too great a volume of blood flow will have a higher CO₂ tension and a lower O₂ tension than the 'ideal' value. Now, let us assume that a particular alveolus has 0.5 of the 'ideal' CO₂ concentration and that it contributes x ml. of alveolar air to the expired air. This x ml. can be regarded as being equivalent to x/2 ml. of 'ideal' alveolar air, and x/2 of unchanged, inspired or dead space air. Let us now turn to the pulmonary circulation and assume that a particular alveolus has too much blood flow so that the blood only takes up 0.5 of the amount of O₂ which is required to bring it to the 'ideal' value. If this alveolus has y ml. of blood flow then its contribution to the arterial blood can be regarded as equivalent to y/2 ml. of 'ideal' blood and y/2 ml. of mixed venous or shunted blood.

In a normal subject the volume of the pulmonary dead space is approximately equal to the volume of the conducting airways. In Table 5 it can be seen that the dead space in Mr. F.H.T. is 170 ml. greater than normal. Most of this increase is due to ventilation of parts of the lung where the capillary circulation is relatively inadequate.

In normal subjects there is a slightly uneven distribution of pulmonary blood flow which, as explained above, causes a 'shunt-like' effect. There are also small volumes of 'true' shunt through bronchial and Thebesian veins. Together the 'true' shunt and 'shunt-like' effects cause the admixture of a volume of mixed venous blood equal to about 2 to 5 per cent. of the cardiac output. In table 4 the apparent shunt in Mr. F.H.T. will be seen to equal 30 per cent. of the cardiac output. (The great bulk of this apparent shunt must be due to the impaired distribution of blood flow, because his arterial blood becomes fully saturated if he breathes 100 per cent O₂. If he had much 'true' shunt, breathing O₂ would not produce full saturation.)

Distribution of Inspired Air

In the above account the term 'distribution' is used to describe ventilation in relation to pulmonary blood flow. It can also be used in the sense of spatial distribution of the air within the lungs. Using the term in this sense, perfect distribution implies that all alveoli during inspiration receive at the same time air of the same composition and in an amount proportional to their volume. There are several techniques by which the degree of perfection of the distribution of air in this sense can be estimated. Most of them depend upon suddenly changing the composition of the inspired air and studying the changes in the composition of the expired air in relation to time. Thus, if there are parts of the lungs which receive too small a proportion of the inspired air they will take longer to reach a new steady level. The unevenness of distribution of the inspired air in Mr. F.H.T. was assessed by two methods. First, he breathed 100 per cent. O₂ for 7 mins. and at the end of that time his alveolar air contained 6.5 per cent. N₂ instead of the expected value of less than 2.5 per cent. This finding shows that there are large parts of his lungs which are poorly ventilated and from which the N₂ was not 'washed out.' Secondly, we used the method of Bates and Christie (1950), which depends upon the rate of mixing of a foreign gas (helium) in the lungs. The normal 'mixing efficiency' by this technique is over 70 per cent. In Mr. F.H.T. the value was 44 per cent.

The Control of Breathing

In normal subjects and even in patients with severe disease of the lungs the arterial CO₂ tension is maintained at 40 mm. Hg. (± 5 mm.). This stability is achieved because the respiratory centre is very sensitive to changes in the arterial CO₂ tension and adjusts the ventilation accordingly. In some patients with very severe lung disease the arterial CO₂ tension is elevated. In the present case that value is about 60 mm. Hg. showing that the alveolar ventilation is only 2/3 of that required to keep the tension normal. Initially this raised PCO₂ would seem to suggest that the respiratory

TABLE 6
THE WORK OF BREATHING

	Mr. F.H.T.	Normal
At Rest (ventilation 9.1/min.):		
Mechanical work, KgM./min.	0.7	0.4
Oxygen cost, ml. O ₂ /min.	20-30	5
Efficiency, per cent.	1-2	5-10
Exercise (ventilation 16.1/min.):		
Total O ₂ consumption, ml./min.	500	500
O ₂ consumption of respiratory muscles, ml./min.	80-100	10

The data were obtained by the methods of Campbell, Westlake, and Charnick (1957).
The normal values for a man of 60 are not known with any confidence.

centre has lost some of its sensitivity to CO₂. However, most of the CO₂ retention can probably be explained by the mechanical difficulty in ventilating the lungs. (The mechanical difficulty requires greater muscular force. Greater muscular force required a greater discharge from the respiratory centre. The greater discharge by the respiratory centre demands a greater stimulation—a higher arterial CO₂ tension.) It is not yet known whether or not there is, in addition to the mechanical limitation, a true diminution of the sensitivity of the respiratory centre to PCO₂ in emphysema. The excessive reduction of ventilation when anoxia is relieved suggests that there probably is some alteration in sensitivity to CO₂ tension. When breathing 100 per cent O₂ our patient's arterial PCO₂ rose from 60 to 70 mm. Hg. showing that the alveolar ventilation had decreased by about 15 per cent. This is only a moderate change compared with those seen in patients who develop CO₂ narcosis.

The Respiratory Muscles

The intercostal, external oblique and sternomastoid muscles were examined electromyographically (Campbell, 1958). The lower intercostals and the sternomastoids were found to contract during inspiration even at rest. They relaxed completely during expiration. There was no expiratory muscle activity even when he was breathless. The diaphragm cannot be examined electromyographically but on clinical examination and screening it was found to be very low and flattened so that the base of the thorax was constricted instead of expanded by its contraction; the vertical excursion during quiet breathing was less than 1 cm. (normal 1.5 cm.); during forced expiration the diaphragm initially was depressed instead of elevated and its subsequent ascent was slow and jerky.

The Work of Breathing

The *mechanical* work performed by the respiratory muscles could be calculated if the pressure exerted by them and the volume of air displaced

by this pressure were measured. Unfortunately such measurements are difficult to make. The *metabolic* work performed by the respiratory muscles can be calculated by estimating their O₂ consumption. Table 6 shows that the work of breathing and the O₂ consumption of the respiratory muscles is somewhat increased in Mr. F.H.T. at rest and greatly increased at higher levels of ventilation. These studies of the work of breathing were made with the subject seated and with his back, shoulders and head supported to obtain maximum relaxation of the accessory muscles. When standing without such support there was clearly much more effort involved and the O₂ cost of breathing must have been even greater. It appeared, in fact, that at 12 to 14 l./min. (his ventilation when standing) he had no O₂ to spare for exercise and if he increased his ventilation the O₂ cost of breathing was greater than the extra O₂ intake.

The Arterial Blood

In Table 7 the O₂ tension at different levels in the pathway from inspired air to arterial blood at rest and during exercise are shown for Mr. F.H.T. and a normal subject. The level of exercise in Mr. F.H.T. was sufficient to double his O₂ consumption. (In a normal subject the values quoted would remain substantially the same up to much greater levels of exercise.) The relative importance of the various abnormalities can be appreciated. Thus the severe fall in arterial O₂ tension on exercise can be seen to be due to the low diffusing capacity. Underventilation could have played no part in this arterial desaturation because the alveolar air composition did not change.

The inter-relationships between the arterial pH, CO₂ tension, total CO₂ content and bicarbonate concentration are very important in the understanding of the acidosis of pulmonary emphysema. They are, however, problems which demand an account of acid base regulation which space does not permit. For an understanding of

TABLE 7
OXYGEN TRANSPORT

	Mr. Rest	F.H.T. Exercise	Normal Rest and Exercise	Physiological Explanation of Difference
Arterial CO ₂ tension, mm. Hg.	60	60	40	Alveolar underventilation at rest. Not more marked during exercise
Inspired O ₂ tension (room air), mm. Hg.	150	150	150	
Alveolar O ₂ tension, mm. Hg.	80	80	100	Alveolar underventilation
Pulmonary end capillary O ₂ tension:				
mm. Hg.	75	35	99.9	} Low diffusing capacity
Saturation, per cent.	93	60	98.5	
Arterial O ₂ Tension:				
mm. Hg.	50	25	95	} Venous admixture effect
Saturation, per cent.	82	40	98	

Rounded figures are given to simplify comparisons.

them, reference should be made to Comroe *et al.*, or to Davenport (1950).

The Assessment of a Patient with Emphysema

Studies such as have been described in this article are outside the range of any but a few centres. It is, however, desirable that much more attention be paid to the assessment of patients with chronic lung disease of all types. At the present time the attention paid to the assessment of these patients compares very unfavourably with that devoted to those with heart, liver or kidney disease.

The individual patient benefits from such studies because diagnosis, progress and the response to treatment are all much better assessed when objective measurements as well as the usual clinical data are available. Furthermore, our understanding of this very neglected group of conditions—asthma, bronchitis, emphysema—will improve if we can advance beyond the purely descriptive clinical criteria which at present are all that we have for defining these conditions.

Unfortunately, those who are tempted to employ pulmonary function tests are often dismayed by the array that face them. They hope for a single simple test that will give them a figure or a value which will tell them how 'good' the pulmonary function is. It should be clear from this article that there is no single test that can adequately assess all patients with chronic lung disease. However, provided one has an appreciation of the elements of pulmonary physiology, a very good assessment can be made of any patient using clinical data and tests that are all within the capacity of a reasonably equipped hospital. I recommend that when faced with a patient with chronic lung disease one should ask three questions:

(1) How good is the mechanical or bellows function of the lungs?

(2) Is ventilation adequate to maintain a normal arterial CO₂ tension?

(3) Are the processes affecting O₂ uptake in the lungs adequate for the maintenance of the normal arterial O₂ concentration?

I will now suggest how these questions can be answered at three levels of precision.

I. These questions can often be simply answered at least partially by physical examination alone:

(1) The bellows function can be assessed by looking for the signs of reduced chest movement or airway obstruction.

(2) Ventilation is probably inadequate if the patient has a raised pulse rate and other evidence of vasodilation. Unfortunately, ventilation is most likely to be inadequate when there is an acute respiratory infection in a patient with chronic lung disease. In those circumstances the signs of underventilation and CO₂ retention are often obscured by those of infection and heart failure. It must be stressed that the absence of cyanosis is not evidence of adequate ventilation. Alveolar ventilation must be reduced almost by half before significant arterial desaturation occurs.

(3) The adequacy of O₂ uptake in the lungs can be assessed by exercising a patient to the limit of tolerance. If cyanosis does not develop, then the diffusing capacity and the distribution of pulmonary blood flow are adequate, and the limit to respiratory exchange is being set by the ventilation.

II. Very simple tests which will suffice in the majority of patients are as follows:

(1) The mechanical function can be assessed by any of the following: fluoroscopy; the F.E.V.₁ test (Forced Expired volume in 1 sec.); the M.B.C.; or (less satisfactory) the Vital Capacity.

(2) The adequacy of ventilation can be assessed by measuring the plasma CO₂ content, bicarbonate concentration or (less satisfactory) the alkali reserve.

(3) The adequacy of O₂ uptake in the lungs can be assessed by measuring the arterial O₂ saturation

(for clinical purposes an ear oximeter is adequate) at rest and exercise.

III. More complex measurements, but which should be well within the capabilities of any centre taking a special interest in these conditions are as follows:

- (1) The elastic properties of the lungs and their resistance to airflow.
- (2) The arterial pH and CO₂ tension.
- (3) The alveolar-arterial O₂ tension difference and, possibly, the diffusing capacity.

If facilities for such studies are available then the more advanced techniques referred to in this article will be within range.

It is preferable that the development of facilities should be as even as possible. It is unbalanced, say, to spend large sums of money on an apparatus for measuring differential lung function or the CO diffusing capacity in a laboratory where the arterial blood CO₂ tension cannot be measured.

Summary

The disordered pulmonary function in emphysema is described using data obtained from one patient.

An approach to the problem of pulmonary function assessment is suggested.

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