PULMONARY VENTILATION AND HYPOXIA*

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HYPOXIA, which is present in some patients with lung disease, may arise for the first time during a surgical operation; it can be inflicted upon an individual with normal lungs or superimposed by the anaesthetist upon one suffering from advanced lung disease.

To recognize the dangers before they develop is better than to have to counter them as they arise. Therefore, a system is desirable for revealing elements of pulmonary disorders likely to result in oxygen deficiency. Anaesthetists should find interest in such a system and in newer methods for testing the functions of the lungs upon which this is based.

HYPOXIA DUE TO PULMONARY DISEASE

Problems of conducting anaesthesia in patients with lung disease may be resolved, at least in part, by knowing which functions are upset and to what extent; this particularly applies to some of the commoner disorders. Unfortunately, physicians use the terms "chronic bronchitis," "asthma," and "emphysema" without general agreement. The phrase "chronic non-specified lung disease" has therefore been suggested for the group as a whole.¹ This includes patients with chronic‡ cough and expectoration, together with paroxysmal or persistent dyspnoea. The group excludes localized lung disease and generalized specific or infective diseases (for example, pneumonia, pneumoconiosis, collagen diseases, or primary cardiovascular renal disease).

Further differentiation of the non-specified group is made possible by lung function tests. Some of these may reveal abnormalities before clinical signs and symptoms appear; and some are simple enough for practical use.

LUNG FUNCTION

The "capacities" are not strictly tests of lung function; they roughly reflect the size of the patient and can be predicted from the height. Reduction in vital capacity (V.C.) occurs with diseases characterized by lesions encroaching upon the available space within the thorax, or which alter its shape, like kyphosclerosis. These "restrictive" disorders, since they diminish functioning lung tissue, are bound in one way or another, sooner or later, to affect respiration and inhalational procedures.

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‡By chronic is meant symptoms occurring on most days for at least three months in the year for at least two years.

"Obstructive" elements in the lower air passages can be as great a trial to the anaesthetist as obstruction in the upper airway. The vital spirogram gives measurements of both "restrictive" and "obstructive" disorders.² For this test a spirometer is needed which will not be affected by inertia or vibration in the fluid column. The instrument designed by Bernstein *et al.*³ overcomes these disadvantages by having a double pulley system for suspension, a light weight ball of reduced length, a wide bore breathing tube, and a reduced water surface. A record of time in seconds is also required.

The patient is asked to inhale as deeply as he can from the air and then exhales as deeply and as fast as he is able into the spirometer. (The forced vital capacity [F.V.C.] which is measured in this way is for all practical purposes the equivalent of the V.C.) That fraction of the V.C. which can be expelled in a given time (0.75 or 1 sec.) is termed the forced expiratory volume (F.E.V.). This test can be more easily applied than the maximum breathing capacity (M.B.C.) to which it is related ($F.E.V._{1.0} \times 35 = M.B.C.$ in Litres or $F.E.V._{0.75} \times 40 = M.B.C.$).

A normal subject can exhale 80 per cent of his V.C. in one second; a patient with obstruction in the lower air passages may only be able to exhale 40 per cent. If the F.E.V. is markedly increased after the inhalation of bronchodilator drugs (adrenalin and atropine compound spray, 1 per cent isoprenaline, or 2.5 per cent phenylephrine hydrochloride) for two minutes, then it would be reasonable to assume that the obstruction is due to muscular spasm such as occurs in asthma. An efficient nebulizer should be used and the test should be repeated one minute later.

Wright⁴ has devised an ingenious device which measures the peak flow of expiration. When this value is compared with the F.E.V., a very rough approximation between the two is found;⁵ unfortunately, some patients with obstructive disorders are able to exhale for a short period at a high velocity.

These tests do not, by themselves, allow of a specific diagnosis. A disorder characterized by a reduced V.C. with a normal F.E.V. indicates a restrictive process such as pleural effusion, a carcinoma, a pneumothorax, a pulmonary fibrosis, or a kyphosclerosis. A normal V.C. with a reduced F.E.V. unaffected by bronchodilator drugs suggests an obstructive process like emphysema.

The forced exhalation ratio $\frac{F.E.V.}{V.C.}$ changes in patients with respiratory disease but is unchanged in those with breathlessness of cardiac origin.⁶

It used to be held that the ratio of the functional residual air (F.R.C.) volume to the total lung volume (T.L.C.), expressed as a percentage, could be used as a measure of the severity of emphysema.⁷ Since emphysema cannot be defined precisely during life, this relation cannot be put to the test; furthermore, there is no linear relation between the ratio and the reduction in oxygen saturation of arterial blood in patients with pulmonary disease.⁸

A simple test is to cause the patient to step up and down a height of 20 cm. for two minutes at the rate of 30 times over a period of one minute (timed by metronome), and then to observe whether there is either cyanosis or breathlessness. An alternative procedure for a patient in bed is to cause him to raise and lower his legs with the knee joints extended at the rate of 60 times per minute

for two minutes. Undue breathlessness or cyanosis may suggest a diffusion defect in the lung and more complicated procedures like the carbon monoxide diffusion capacity are indicated.

If the patient is cyanosed at rest and the cyanosis remains after the inhalation of 100 per cent oxygen, then it may be assumed that there is a venous to arterial shunt. An oximeter is a better guide than relying upon visual impressions because, as Comroe and Botelho⁹ showed, clinicians may not recognize a state of cyanosis until the arterial blood is only 70 per cent saturated with oxygen. Intrapulmonary shunts may result from direct venous to arterial channels which have developed as a result of infective processes.¹⁰ When the disease process has developed so far as to cause respiratory failure, a raised carbon dioxide tension ($p\text{CO}_2$) can be measured. The normal range of $p\text{CO}_2$ is 40 mm. Hg, plus or minus 5 mm. Hg; 47 mm. Hg is considered the upper limit.

For many years $p\text{CO}_2$ measurements remained the province of respiratory physiologists because the methods were time-consuming and difficult. Recently a method has been introduced¹¹ and modified¹² so that $p\text{CO}_2$ can be measured with simple apparatus, and, furthermore, it comes within the capabilities of the resident anaesthetist or medical officer: to carry it out takes less than ten minutes.

The difference between the $p\text{CO}_2$ of mixed venous and arterial blood is 6 mm. Hg in the subject at rest¹³ and this difference remains even in the presence of pulmonary disease. The aim of the test is to measure the mixed venous $p\text{CO}_2$. Subtracting 6 mm. Hg from this value gives an estimate of arterial $p\text{CO}_2$. The subject breathes into a half-gallon bag filled with oxygen or air for one and a half minutes. At the end of this time the contents of the bag are nearly in equilibrium with the mixed venous (pulmonary artery) $p\text{CO}_2$. After a two-minute rest, the subject breathes into the bag again for 15 sec.; at the end of this time the bag $p\text{CO}_2$ is identical with the $p\text{CO}_2$ of mixed venous blood. If the 15-sec. period is exceeded then there is a further accumulation of CO_2 from a recirculation. The error of this method is of the order of 3 mm. Hg. Although erroneous results occur when there is a large intracardiac shunt, the method is reasonably accurate in the presence of pulmonary disease.

Moran Campbell¹⁴ has modified Haldane's apparatus to facilitate the measurement of $p\text{CO}_2$ and this has been achieved by removing the temperature compensatory burette and, instead of having to raise and lower the mercury bulb by hand, a syringe is used; all the operator has to do is to raise and depress the syringe barrel about fifteen times.

HYPOXIA MANIFESTED DURING OPERATIONS DUE TO PULMONARY DISEASE

Pathological lesions likely to influence anaesthesia are destruction of lung tissue, cyst formation, replacement of lung tissue by fibrosis, and reduction or thickening of the alveolar membrane.

Destruction of lung tissue reduces the extent of the vascular bed and eventually results in pulmonary hypertension. This leads to cardiac failure (*cor pulmonale*). Reduction in the extent of the alveolar membrane produces a diffusion defect. Where there are irregular patchy areas of lung destruction there may be some

alveoli without a vascular supply although they may be ventilated; this leads to an increase in the physiological deadspace; if alveoli are perfused but not ventilated then there is a venous admixture. Loss of supporting tissues around the bronchioles may result in airway obstruction during expiration, especially when expiration is an active process as may be the case after air trapping. Air trapping may occur when air passes into cystic areas of destroyed alveoli with valvular openings as described by Reid.¹⁵ Air may also be trapped distal to obstructed air passages. Replacement of lung tissue by fibrosis will result in a reduction of the capacities and will lower the reserve volumes. Restrictive disorders affect the rate at which gases are exchanged and hypoxia, when it appears, does so rapidly. Thus there is less time available for intubation and hypoxia develops fast during respiratory spasm.

Patients with obstructive diseases like chronic bronchitis and emphysema would be expected to develop respiratory spasm more easily because of the unduly sensitive state of the bronchi, and they are more likely to develop hypoxia from the obstructive effects of secretions which have not been coughed up. In some patients respiration is only maintained by a hypoxic drive and these present the anaesthetist with problems which may become acute in the immediate post-operative period. For instance, it is difficult to ascertain the extent to which suboxygenation may be allowed.

When there is an increase in the physiological dead space this must be allowed for in assessing the correct amount of increased artificial ventilation volumes.

In restrictive disease the inhalation of oxygen for a few minutes before anaesthesia is induced is a wise precaution. Speed in intubation comes from skill and adequate preparations.

The use of intermittent positive pressure (I.P.P.) techniques are preferable to techniques with spontaneous respiration because the effect of positive pressure is to open up alveoli which were previously closed; at least this appears to be the case from the work of Motley and Tomashefski¹⁶ in patients with pulmonary fibrosis. They showed how the oxygen difference between the alveoli and the arterial blood (Aa difference) decreased and the arterial oxygen tension rose with the patient breathing air with I.P.P. respiration to a value higher than it was when the patient breathed a high oxygen concentration spontaneously. Reducing the oxygen of inspired gases reduced the Aa difference; increasing the oxygen increased the difference.

In obstructive non-specific pulmonary disease it is important that the technique be arranged so that the upper airway is secure and all procedures and agents, such as ether and cyclopropane, likely to evoke spasm, should be avoided. To prevent any straining, the author's practice is to give the longer-acting relaxant before the patient begins to breathe as the effects of the short-acting relaxant (suxamethonium) wear off. Although contrary to current teaching, because of the danger of persistent curarization, this ensures a smooth transition without any straining.

Under full curarization there should be no active expirations and so air trapping from this cause is unlikely except during the recovery period as muscular power returns. Intermittent positive pressure respiration has much to

commend it in patients with pulmonary disease having surgical operations under anaesthesia, and not the least of the arguments in favour is that the patient can be well ventilated. If a negative phase is employed during expiration, this should be used with care; in some instances hypoxia will develop because the thorax ceases to expand, presumably from air trapping during the positive phase.¹⁷ Mixtures of helium and anaesthetic gases are rarely used in these patients.

HYPOXIA DUE TO ANAESTHESIA

Using the wrong gas in place of oxygen is now fortunately rare, but although there is a dual supply on every anaesthetic machine, it is still possible for the cylinder to become empty and for the anaesthetist to fail to realize it. With spontaneous breathing the odds are that the anaesthetist will notice the decrease in the bag size, but with artificial respiration with a pulmoflator, the error may not be so obvious and the patient may receive many respiration with pure nitrous oxide. There are devices (such as the Bosun)* which will sound an alarm when the oxygen runs out, but unfortunately the anaesthetist may have forgotten to set the instrument into action.

The use of profound respiratory depression from the anaesthetic agent is likely to add the histotoxic form to anoxic anoxia and this combined state is most undesirable. Anaesthesia with spontaneous respiration increases the liability of anoxic anoxia, but this is to some extent avoided by the use of high oxygen mixtures as when halothane is used. Whatever form the respiration takes, it is desirable for the anaesthetist to know the extent of the respiration volumes, and there are a number of devices^{4,18} to assist him.

Although the tidal and minute volumes can be readily measured, it is less easy to estimate alveolar ventilation, which is much more important. This is computed by subtracting the dead space (anatomical and physiological, whichever is the larger) from the tidal volume. The dead space is altered by intubation and by anaesthetic equipment.

An example of the way in which the dead space is affected by intubation may be taken from the case of a man with a 2-cm. diameter trachea which is 10 cm long. Assume that the lower end of the tube reaches the mid-point of the trachea, then the dead space will amount to that volume contained in the tube (15 ml.), half the trachea (15 ml.), and the air passages below the carina. (Rohrer¹⁹ computes this to be of the order of 70 ml.) Thus the dead space will amount to some 100 ml. With the patient breathing air through the upper end of the tube, the dead space would be reduced by some 50 ml. if it be assumed that the man's anatomical dead space is 150 ml. (Such would be the case in a man weighing 150 lb.) In anaesthetic practice, however, the upper end of the tube would be connected to a circle CO₂ absorption apparatus with an additional dead space of about 10 ml. A mask and a circle system would add 160 ml.; a mask and a Water's canister would add some 230 ml. (if the volume to the absorbing soda lime surface is reckoned at 80 ml.); a Water's soda lime canister without a mask would add some 80 ml. In the light of these findings it is interesting to compute

*Obtained from the British Oxygen Company Ltd., Deer Park Road, London.

the possible alveolar ventilation in a man inhaling oxygen and cyclopropane without intubation. If the patient's dead space is reckoned at 150 ml., then the total dead space will be of the order of 380 ml. If the tidal volume is depressed below the normal lower limit of 350 ml., then the alveolar ventilation will be 30 ml. multiplied by the respiratory rate; even if this were as much as 20/min., then the alveolar ventilation would only amount to 0.6 L./min. which is a marked reduction from the normal lower limit of about 3.5 L./min.

In some pulmoflators, provision is made so that the whole of each expiration is removed without rebreathing and without CO₂ absorption; in this type of equipment the increase of dead space due to the apparatus would amount to about 10 ml. only.

What happens to the physiological dead space during anaesthesia is more difficult to compute. Work in our own department²⁰ has shown that there is a difference between the pCO₂ in arterial blood and in alveolar air during anaesthesia with either spontaneous or artificial respiration. This order of difference (5 mm. Hg) was not found in conscious subjects where there is a difference of less than 2 mm. Hg which is almost within the error of the measurements. Other workers^{21,22} have confirmed that this gradient exists which suggests that there is an increase in the physiological dead space during anaesthesia.

Diffusion anoxia²³ after nitrous oxide and oxygen anaesthesia may be countered by close supervision and efficient oxygen therapy in the immediate recovery period. The anoxia is intensified by an outpouring of secretions as might occur when neostigmine has been given after insufficient atropine at the end of operation.

The hypoxic dangers of respiratory spasm are becoming less with the increasing use of relaxant techniques or those which include non-irritating agents such as halothane.

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