MECHANICAL PULMONARY VENTILATION: INDICATIONS FOR ITS USES IN SURGICAL CASES*

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In the widest sense we may say that mechanical ventilation in the lungs is indicated by respiratory failure—the condition where the respiratory gas exchange between the lungs and the blood falls below that between the tissues and the blood.

PHYSIOLOGICAL CONSIDERATIONS

The manner in which respiratory function may fail is best considered in terms of normal respiratory physiology. The processes concerned with the uptake of oxygen and the elimination of carbon dioxide by the lungs—and certain aspects of acid-base balance—may be considered[1] as being (1) ventilation (the mass movement of air in and out of the lungs), (2) distribution (the distribution of the inspired air and the pulmonary blood flow within the lungs—these bear an optimal relationship to each other), (3) diffusion (the process by which O_2 and CO_2 are exchanged between alveolar air and the pulmonary capillary blood), (4) pulmonary blood flow (the amount of blood brought into contact with the respiratory epithelium in unit time), and (5) blood gas transport.

An understanding of the effects that derangements of these factors have on the arterial oxygen saturation and carbon-dioxide tension is essential for a clear understanding of respiratory failure. The first three are the mechanisms most relevant to clinical respiratory failure.

(1) *Ventilation*

If ventilation is impaired the amount of O_2 that can be taken up by the blood, and CO_2 excreted, is limited.

If the shape of the dissociation curves of oxygen and carbon dioxide, and the consequent effect of alveolar ventilation on the blood content of these gases is considered (Fig. 1), it will be realized that the CO_2 tension in the arterial blood will be the first to reflect any change in ventilation, rising or falling proportionately with change in ventilation. Reduced tension of O_2 in the alveolar air only leads to arterial oxygen desaturation of any moment where it has fallen quite markedly. But when it does reach this point where significant desaturation of arterial blood results it is on the slippery slope of the Eiger. Any further reduction in alveolar O_2 tension, as would result from a further decrease in ventilation, will result in gross and catastrophic arterial oxygen desaturation. This should serve to remind us that cyanosis is a sign of severe ventilatory impairment.

It must be noted also that hyperventilation while breathing air cannot increase the arterial oxygen saturation beyond 96 - 98%, its normal level. Remember, too, that with underventilation not only will there be the obvious respiratory acidosis, but the resultant tissue anoxia, leading to an increase in the fixed acids, will result also in a metabolic acidosis, thus adding fuel to the acidotic flame.

(2) and (3) *Distribution and Diffusion*

These two factors may be grouped together. Derangements often occur together, and the changes produced in the arterial oxygen saturation and CO_2 tension are in the same direction. Derangements in distribution result in disturbance of the over-all ventilation/perfusion ratios in the lung. The effects such changes have on the blood gas content are again dependent on the shape of the dissociation curves of the respective gases. Whereas the rise in CO_2 tension resulting from underventilation of a normally perfused lobule of lung may be compensated for by overventilation of another normally perfused lobule—thus preserving a normal total arterial CO_2 tension—the reduction in arterial oxygen saturation so resulting cannot be compensated for by this mechanism. It can be corrected only by raising the inspired, and so alveolar, O_2 tension. Derangements of any moment, therefore, in the ventilation/perfusion ratios in the lung always result in a lowered arterial oxygen saturation, possibly with a normal, not raised, arterial CO_2 tension, certainly in the early stages.

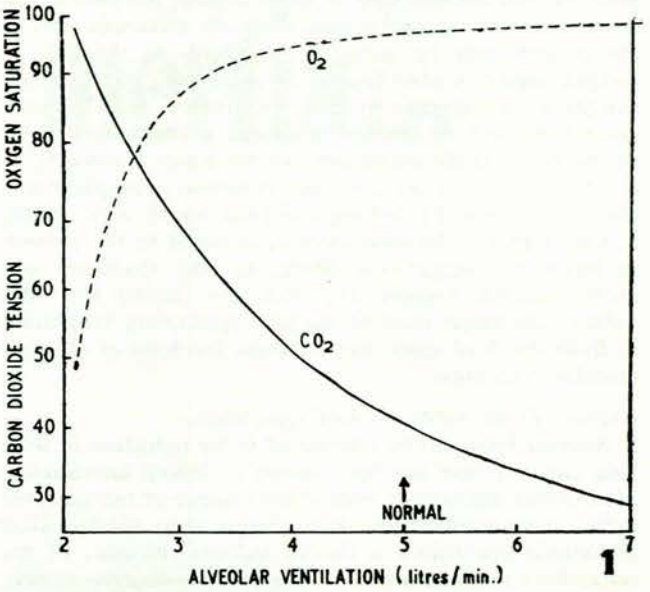

Fig. 1. Effect of the volume of pulmonary ventilation on arterial CO_2 tension (mm.Hg) and oxygen saturation (%).

With disturbances of diffusion, the differences in the relative solubilities of O_2 and CO_2 lead to an impairment of the O_2 uptake of the blood with no significant effect on the excretion of CO_2, and may thus lead to a similar blood gas picture of reduced arterial oxygen saturation with a normal CO_2 tension.

As recently shown by Nunn and Payne,[2] derangement of distribution produces detectable arterial oxygen desaturation in the immediate postanaesthetic phase more often than we realize.

The Work of Breathing

Another aspect of pulmonary physiology that must be

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considered is that of the work of breathing.[9, 15] The total mechanical work of breathing in normal subjects at rest is about 0·6 kg.-m. per min.,[1] with a metabolic cost to the patient of 1 - 3% of the total oxygen intake (i.e. about 0·5 ml. O_2 per litre of ventilation, or 4 ml. O_2 per min). In patients with chronic lung or heart disease, the mechanical work of breathing, and consequently the metabolic cost to the patient, may be increased 5 - 10fold. As the rate of oxygen intake is also limited in these patients, the consumption of oxygen by the respiratory muscles may severely restrict the amount of oxygen available to the rest of the body. If the compliance of the lungs is reduced, as it is for instance by postoperative pulmonary complications, the work involved in taking a normal breath is increased. It may therefore become more economical to the patient, in terms of energy expenditure, to take shallower and more frequent breaths. However, the smaller the tidal volume, the larger must be the total pulmonary ventilation to flush the dead space and maintain the level of effective alveolar ventilation.

Changes Produced by Surgical Operations

Another factor to be considered is the reduction in total lung capacity that has been shown to follow immediately after certain operations, even in the absence of recognizable pulmonary complications. This change, most marked after abdominal operations, is due to reduced efficiency of the respiratory musculature and pain in the operative wound. Anscombe and Buxton[3] found a mean reduction in total lung capacity after abdominal operation of 2·4 litres, with a mean reduction in vital capacity of 1·2 litres. These changes, for which a patient with normal pulmonary function compensates easily, may tip an emphysematous patient into respiratory insufficiency and failure.

OBSERVATIONS AND SPECIAL INVESTIGATIONS FOR ASSESSMENT AND MANAGEMENT OF MECHANICAL PULMONARY VENTILATION

Ideally, for assessing a patient as a candidate for mechanical pulmonary ventilation and for correct management of that treatment once it is instituted, there should be on the spot special facilities for measuring (1) volume ventilation as regards (a) rate, (b) minute volume, and (c) tidal volume; (2) arterial CO_2 tension; (3) arterial oxygen saturation; and (4) pH. Though it is possible to conduct the management of patients on ventilators without these facilities, if they are available they remove much of the guesswork and empiricism that result from their absence.

INDICATIONS FOR MECHANICAL PULMONARY VENTILATION

Correlating with the physiological factors mentioned above, two distinct types of respiratory failure may be recognized —distinct certainly in their early stages, though in later stages features of the first type mentioned are predominant. These two types are failure of respiratory function due to (1) a failure of ventilation, or (2) abnormalities of ventilation/perfusion and diffusion.

1. *Failure of Ventilation*

The cases that most often and most obviously call for the use of mechanical pulmonary ventilation are those in which the primary cause of respiratory insufficiency is a failure to move an adequate volume of air in and out c the lungs (i.e. failure of ventilation).

Clinically, this group of cases displays dyspnoea, swea ing, and tachycardia, with an initially raised bloc pressure. There is often skin vasodilation. Besides appea ing shallower than normal, the pattern of respiration often abnormal. For example, it may manifest a trache tug or paralysis of intercostal muscles or diaphragm. Late there will be cyanosis, mental confusion, large pupil twitching, low blood pressure, cardiac arrhythmia, and the end coma and death.

The four special investigations mentioned at the begi ning of this section will reveal:

(*a*) A reduced volume of effective pulmonary ventilatio

(*b*) A raised arterial CO_2 tension, usually very high.

(*c*) Arterial oxygen desaturation on breathing air, whic reverts to normal on breathing 100% O_2.

(*d*) In severe cases the pH will be lowered because (i) respiratory acidosis and later (ii) metabolic acidos from tissue anoxia.

In surgical practice this group of cases would include (1) Patients in whom there is a *functional disturbance the mechanism of ventilation*, viz.:

(*a*) Depression of the *respiratory centre itself*. This ma be due to cerebral trauma or to the effects of central depressant drugs such as the opiates and other analgesi or the barbiturates.

(*b*) Impairment of the *peripheral transmission mech nism* of the respiratory impulses. The prime example this circumstance, and the bogey of anaesthetists, is pr longed postoperative curarization from whatever cau Postoperative ventilatory impairment may also happen that *rara avis* the myasthenic patient and in patien suffering from certain myopathies and neuropathies. is seen in the patient with a fractured spine causing par lysis of the diaphragm and/or the intercostal muscl according to the level of the fracture. Metabolic acidos is becoming more obtrusive in surgical practice today the cause of functional ventilatory impairment.

(2) Patients in whom there is a *mechanical or anatomic disturbance of ventilatory capacity*, viz.:

(*a*) One of the commonest conditions that we must co sider in this group is chronic obstructive lung disease emphysema and chronic bronchitis. This is a compos disease that ultimately includes ventilatory/perfusi disturbances, but the basic pathology is bronchiolar o struction. What so often finally renders the ventilato capacity of the emphysematous patient inadequate is t association of one of the functional disturbances I ha mentioned with emphysema. (Remember that the asse ment of adequacy of ventilation from the measurement gross volume of ventilation alone may be fallacious view of the increased physiological dead space in patien suffering from emphysema.)

(*b*) A less common anatomical restriction of ventilato capacity is that which may follow pulmonary resection, where the remaining functioning lung tissue would be ju adequate, but is rendered inadequate in the immedia postoperative phase.

In groups (*a*) and (*b*) the possibility of postoperati respiratory difficulty may be anticipated by pre-operati

studies of respiratory function.

(*c*) Patients suffering from a critically crushed chest with multiple fractured ribs—an increasingly common phenomenon—are an important example of mechanical restriction of ventilatory capacity. In these cases there is gross disturbance of the mechanics of the thoracic cage together with pulmonary contusion, leading to a marked ventilatory impairment. Many methods are described of achieving surgical fixation of the unstable flail chest wall. However, in many centres, surgical methods of stabilizing crushed chests have been abandoned in favour of the use of mechanical pulmonary ventilation.[7-9]

2. *Abnormalities of Ventilation/Perfusion and Diffusion*

In this second group of cases, in which respiratory failure is due to ventilation/perfusion disturbances and diffusion abnormalities, the indications for the use of mechanical pulmonary ventilation are not as clear cut, nor are they as common, as those in the first group. Together with the ventilation/perfusion abnormalities and disturbances of diffusion, the increased work load of respiration that results from the lowered pulmonary compliance becomes an important aspect in these cases. An example of this type of respiratory failure is that which sometimes occurs after operations on the heart with cardiopulmonary bypass, particularly in patients who have had pulmonary hypertension. These patients manifest a respiratory distress[10, 11, 14] of which the most obvious sign is tachypnoea and mild cyanosis. There is an obvious increase in the respiratory work needed, and performed, by the patient. The four special investigations will show the following changes, in contrast with those characteristic of cases in which the primary cause of respiratory insufficiency is failure of ventilation:

(*a*) The volume of ventilation is increased.[10, 11] Though tidal volume is decreased, the physiological dead space is also decreased[12] and the frequency of respiration is markedly increased, with a resultant net increase in the total alveolar ventilation.[10, 13]

(*b*) The arterial CO_2 tension may be normal. It is often not raised.[10, 11, 15]

(*c*) The arterial oxygen saturation is reduced on breathing air. This will be increased on breathing 100% O_2, the degree depending on the amount of pulmonary veno-arterial shunting due to atelectasis.

(*d*) The pH may be normal.

Changes of this type are illustrated in Fig. 2. This illustrates daily estimations of ventilatory parameters and arterial oxygen saturation on breathing air and pure O_2 in the immediate postoperative period after operations on the heart with total body perfusion. This investigation was carried out at the British Postgraduate Medical School, Hammersmith Hospital, London.[10, 13] These changes are interesting in that they illustrate the failure of respiratory function without the failure, at least initially, of ventilation *per se*. Later, if the condition worsens, ventilation will begin to fail and the conditions of the common first type of respiratory failure will supervene.

In these cases, raising the inspired oxygen concentration, e.g. by use of an oxygen tent, is usually sufficient to increase the arterial oxygen saturation to adequate levels,

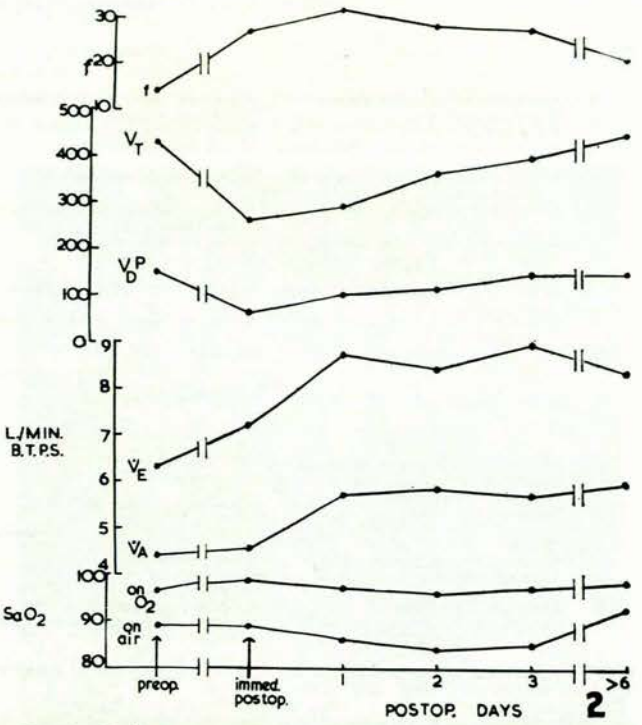

Fig. 2. Daily measurements of ventilatory parameters in the postoperative period after open heart surgery (means of 8 cases). These patients did not require mechanical pulmonary ventilation. (British Postgraduate Medical School, Hammersmith Hospital, London.[10, 13])
f = frequency (breaths per minute). V_T = tidal volume and V_{DP} = physiological dead space (both in ml. BTPS). $\dot{V}_E$ = minute volume and $\dot{V}_A$ = alveolar ventilation (both in litres per minute BTPS). SaO_2 = arterial oxygen saturation (%).

and the tachypnoea, with the increased work it demands, will decrease. However, in the severe cases where the patient is becoming exhausted, the institution of mechanical pulmonary ventilation with high inspired oxygen concentration will benefit the patient. Such benefit results not only from the improved ventilation and diffusion but also, as the work of breathing is taken over by the pump, from the diversion to the general body economy of the oxygen formerly utilized by the overworked respiratory muscles.

MANAGEMENT OF MECHANICAL PULMONARY VENTILATION

Recent advances in the design, sensitivity and response time of triggering mechanisms of some of the mechanical ventilators now available, make 'patient-triggered augmented ventilation' the method of choice in many cases requiring mechanical pulmonary ventilation today.

Patient triggered augmented (PTA) ventilation has the advantage of being virtually 'self-monitored'. The maintenance of the patient's spontaneous respiratory effort serves as a short-term safety mechanism for the emergencies that occur from time to time with this form of therapy. Further, the patient can communicate with his medical attendants by means of speech if the tracheotomy cuff is deflated. We regard PTA ventilation as indicated in all circumstances in which mechanical pulmonary ventilation is required in patients in whom some respiratory muscular activity is

TABLE I. PATIENTS TREATED WITH MECHANICAL PULMONARY VENTILATION

Patients	*Lesion*	*Operation*	*Indications for MPV*	*Type of MPV and make of ventilator*	*Duration of treatment (days)*	*Result*
			Open heart operations			
1	Aortic and mitral valve disease	Aortic and mitral valve prosthesis	Hypoventilation. Cold injury to phrenic	PTA. Bird	7	S
2	Mitral regurgitation	Mitral cusp extension	Pulmonary oedema	IPPR. Radcliffe	4	S
3	Atrial septal defect	Repair of atrial septal defect	Thoracic scoliosis. Hypoventilation	PTA. Bird	14	S
4	Mitral valve disease. Pulmonary hypertension	Mitral valve prosthesis	Respiratory distress	PTA. Bird	14	S
5	Mitral valve disease. Pulmonary hypertension	Mitral valve prosthesis	Respiratory distress	PTA. Bird	3	D
6	Mitral and tricuspid valve disease	Mitral valvotomy. Tricuspid valve prosthesis	Pulmonary oedema	IPPR followed by PTA. Bird	6	D
7	Aortic and mitral valve disease	Aortic valve prosthesis	Respiratory distress	PTA. Bird	5	D
8	Mitral valve disease. Pulmonary hypertension	Mitral valve prosthesis	Prolonged low output. Cerebral damage. Hypoventilation	PTA. Bird	1	D
9	Mitral and tricuspid valve disease	Mitral prosthesis. Tricuspid amuloplasty	Hypoventilation	PTA. Bird	4	S
10	Mitral valvotomy — acute mitral regurgitation	Mitral prosthesis (after 4 days)	Pulmonary oedema	IPPR. Bird	5	S
11	Left ventricular aneurysm	Excision	Pulmonary oedema	IPPR. Bird	4	S
			Closed heart operation			
12	Mitral stenosis	Mitral valvotomy	Pulmonary hypertension and emphysema. Hypoventilation	IPPR. Cyclator	7	S
			Crushed chest			
13	Multiple fractured ribs. Head injury	None	Flail chest	IPPR. Cyclator	5	D
14	Fractured ribs. Fractured sternum. Coronary thrombosis	None	Flail chest	IPPR. Cyclator	6	D
15	Neglected fractured ribs	None	Flail chest. Gross bronchopneumonia	IPPR. Bird	10 hrs.	D
			General surgery			
16	Ruptured abdominal aneurysm	Resection and grafting	Emphysema. Prolonged curarization	PTA. Bird	10	S
17	Gunshot wound	Laparotomy	Emphysema. Prolonged curarization	IPPR. Cyclator	6 hrs.	S
18	Pyelonephritis	Nephrectomy	Prolonged curarization	IPPR. Cyclator	2 hrs.	S
19	Abdominal aneurysm	Resection and grafting	Emphysema. Cardiac failure	IPPR. Cyclator	9	S
20	Bronchopleural fistula, pneumonia, and septicaemia	Insertion of thoracic under-water drain	Tachypnoea ++. Respiratory distress	PTA. Bird	2	D

MPV = mechanical pulmonary ventilation. PTA = patient-triggered augmented ventilation. IPPR = intermittent-positive-pressure ventilation. S = survived (see text). D = died.

present, provided that a negative-pressure phase (such as is necessary for triggering the ventilator) is not contraindicated by such conditions as pulmonary oedema or flail chest. For the patients with respiratory distress characterized by tachypnoea, to match the patient's respiratory efforts it is essential to have a ventilator that has a very rapid response time and is capable of delivering very high flow rates of gas. (For this we use the Bird Mk.8 ventilator.)

In the past 24 months we have had occasion to treat 20 surgical patients with mechanical ventilation for varying periods postoperatively at Groote Schuur Hospital (Table I). From this it may be noted that of these 20 patients, 9 were treated with PTA ventilation. Of the 11 treated by IPPR, 3 were so treated only because a ventilator considered adequate for PTA ventilation was not available at the time. In the column *Result,* 'survived' (S) means that the patient survived beyond the period for which respiratory assistance was necessary, while 'died' (D) means that the patient died while still having mechanical pulmonary ventilation.

As this paper is devoted to 'Indications', discussion of the results and complications encountered in these patients will be the subject of a future communication.

SUMMARY

Mechanical pulmonary ventilation is indicated by respiratory failure, viz.:

(1) That in which the prime failure is of *pulmonary ventilation.* In this type there is:

(a) Reduced pulmonary ventilation.
(b) Increased arterial CO_2 tension.
(c) Decreased arterial oxygen saturation on breathing air, which reverts to normal on breathing 100% O_2.
(d) In late cases a decrease in pH.

(2) Less commonly, that in which the primary failure is one of ventilation/perfusion ratios and diffusion abnormalities. This type may paradoxically show:

(a) Increased pulmonary ventilation.
(b) Normal arterial CO_2 tension.
(c) Arterial oxygen desaturation on breathing air, which may not necessarily be restored entirely to normal on breathing 100% O_2.
(d) The pH is usually normal.

Special reference is made in this type to the increased work demand of respiration.

Given a ventilator with the necessary sensitivity and rapid response time, patient-triggered augmented ventilation is considered the method of choice for providing mechanical pulmonary ventilation.

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