Pathophysiology and Treatment of Acute Respiratory Failure in the Postoperative Patient

R. J. JEDEIKIN

SUMMARY

The pathophysiology and treatment of acute respiratory failure in the postoperative patient is discussed. Special emphasis is placed on the concept of closing volumes, which forms the basis for the understanding of the pathophysiology, and is used as an index for patient assessment.

In the treatment of acute respiratory failure postoperatively, the discussion has been confined to prophylaxis, use of intravenous analgesia, intermittent positive pressure ventilation and the dramatic use of positive end-respiratory pressure.

S. Afr. Med. J., **48**, 1257 (1974).

The recent advances in anaesthesia and intensive care have resulted in a better understanding of the pathophysiology of acute respiratory failure (ARF) and consequent improvement in its management. The aim of this review is to briefly discuss these two aspects of ARF, because it is a serious postoperative complication that can usually be avoided.

Department of Anaesthetics, University of Cape Town

R. J. JEDEIKIN, B.SC., M.B. CH.B., F.F.A. (S.A.) (Present address: University of Vermont, Department of Anesthesiology, Mary Fletcher Hospital, Burlington, Vermont, USA)

Date received: 11 February 1974.

Ventilation *per se* is extremely difficult to monitor properly, while cyanosis is a notoriously unreliable physical sign. For these reasons there is no recognised clinical definition of respiratory failure. The diagnosis of ARF is made solely on blood gas estimation: 'acute respiratory failure is a state where the arterial oxygen tension (p_aO_2) is 60 mmHg or less, and/or the arterial carbon dioxide tension (p_aCO_2) is 49 mmHg or higher'.[1]

In essence, this is a measure of the ability of the lungs to carry out their primary function in life—gaseous exchange.

PATHOPHYSIOLOGY

During normal tidal-volume breathing, the volume of air left in the lungs at the end of expiration is ± 3 000 ml, and is known as the functional residual capacity (FRC). Following maximal expiration there is usually a residual volume (RV) of ± 1 200 ml in the alveoli that cannot be exhaled. However, during forceful exhalation, as the volume of the lungs is reduced, small airways (less than 2-mm diameter) tend to collapse.[2] Furthermore, it is the small airways in the dependent lung regions (where there is less negative pressure) that tend towards early closure. The volume of air left in the lungs when airway closure occurs

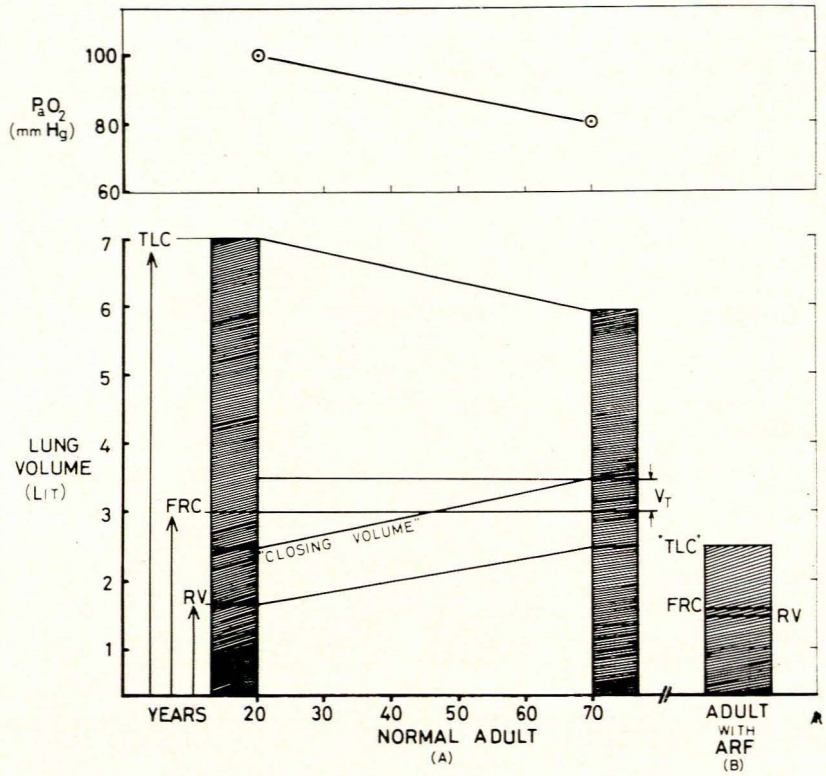

Fig. 1. Increasing closing volume shown in relation to age.

is known as the closing volume, and will vary according to position and age.[3,4] The older the person the earlier small airway closure during expiration will occur, and therefore the larger will be the closing volume (Fig. 1).

In the young person, airway closure only tends to occur during forced expiration at volumes approximately equal to RV, while in the elderly, probably because of loss of elasticity, airway closure may occur during tidal volume expiration. The above holds true for the upright position. In the recumbent position, the diaphragm will encroach on the expiratory volume resulting in earlier airway closure.

Normally, those small airways that have closed will open on inspiration, sighing or yawning, and at the end of inspiration, regardless of age, all regions of the lung are open. If, however, they should fail to open due to an abnormality, air which is trapped behind the collapsed passages is absorbed, resulting in alveoli collapse and atelectasis. Pulmonary capillaries continue to perfuse this non-ventilated area of lung tissue, resulting in ventilation-perfusion imbalance, which manifests clinically as a drop in the partial pressure of oxygen (p_aO_2).

This is exactly what can happen postoperatively and explains why the obese, elderly patient so often develops postoperative basal atelectasis. The lung bases are particularly prone because there is less negative pressure and the alveoli are already small. As the patient is recumbent postoperatively, the diaphragm is raised and the lung volume decreased. In the immediate postoperative period following a general anaesthetic, the patient is usually unable to take a deep breath, sigh or cough in order to reopen collapsed or closed alveoli.

If the patient is bronchitic, or even has just a plain cold, the airways may already be partially closed with mucus as well as being congested. This results in collapse and consolidation, and the patient is on the way to respiratory failure.

When a patient is already in acute respiratory failure his lung volumes are usually decreased and there is a drop in the FRC. The cycle perpetuates itself, and with further diminution of FRC there is an increased tendency for early airway closure in expiration. The extent to which this occurs will be influenced by 3 factors: (*a*) the nature of the acute process, e.g. mucous plugging, pulmonary emboli, Mendelsohn's syndrome, etc; (*b*) integrity of the surfactant system, e.g. the presence of pulmonary oedema will, for instance, increase the surface tension by destruction and surfactant; and (*c*) low compliance and/or increased airway resistance as can occur in chronic obstructive airway disease, kyphoscoliosis, etc.

PATIENTS AT RISK

There are basically 2 groups of factors (explained in terms of airway closure) which, unless attended to, will precipitate ARF. Awareness of these factors and anticipation of the suspect patient can lead to better preoperative workup, followed by a drop in the incidence of postoperative ARF, more thoughtful anaesthesia, and early recognition of respiratory problems postoperatively, resulting in early, vigorous therapy.

Group A (Non-Pulmonary Factors)

The obese patient[5] or the patient with abdominal distension is a poor risk. Anaesthesia *per se*,[6] the supine and head-down position,[7] as well as inadequate turning of patients, potentiate airway closure, as do too vigorous airway suction and expiratory efforts. Paralysis, debility, narcotic drugs and pain result in poor inspiratory effort and failure to open closed airways.[8]

Group B (Pulmonary Factors)

There are basically 3 pathological factors that are associated with most pulmonary conditions: increased surface tension; interstitial oedema[9] (e.g. a patient in congestive cardiac failure); and inflammation (including the common cold), resulting in swelling of the bronchial mucosa and the intrabronchial tree.

PATIENT ASSESSMENT

A full clinical history and examination is mandatory, and only from a sound clinical basis can the decision be made as to what further sophisticated tests should be done. It is, however, rare for a patient about to undergo surgery today, not to have a pre-operative chest X-ray film taken, and if he is over 40 years of age, an ECG. The respiratory and cardiovasculature status of the patient is of extreme importance. Where a patient with chronic obstructive airway disease is coughing copious amounts of sputum, it is often of more consequence than his lung function studies. It is the patient with undiagnosed myocardial ischaemia who is likely to be tipped into pulmonary oedema by the injudicious administration of intravenous fluid. Furthermore, the type of operative procedure in conjunction with the patient's clinical state may influence the clinician in having the patient artificially ventilated during the immediate postoperative period.

The basic lung function tests are the vital capacity (VC) and the forced expiratory volume in the first second (FEV_1), which are indicative of the ventilatory reserve and mechanics. Occasionally FEV_1 and VC are used in deciding whether a patient may have the respiratory reserve necessary in order to recover from a pneumonectomy or lobectomy.

It is occasionally necessary to do full pulmonary volume studies including single-breath and steady-state measurements. Assessment of ventilation is done by assessing the p_aCO_2 while the p_aO_2 and inspired oxygen concentration (F_iO_2) are for assessing oxygenation of the patient. It is important that the p_aO_2 be related to the F_iO_2 in order to estimate the efficiency of oxygenation.

TREATMENT

Prophylaxis is the cornerstone of all medical treatments. The importance of early recognition of risk factors and subsequently the timely treatment of these patients pre-operatively, will result in a marked clinical and laboratory improvement with low morbidity and low mortality post-operatively.

Adequate treatment of underlying disease is well appreciated, as is the ever-increasing and important role of the chest physiotherapist, before as well as after operation.

Perhaps the most difficult problem facing the clinician in the postoperative period is the adequate treatment of pain. Too little analgesia can result in hypoventilation, ineffectual coughing with poor clearance of secretions, and subsequent atelectasis and infection. Over-sedation or too much analgesia, especially in the at-risk patient with emphysema, by depressing the respiratory centre, will result in the same sequence of events as those that follow on too little analgesia.

Regional anaesthesia and local anaesthetic blocks are commonly used in some centres and are an excellent way of treating pain. However, not all parts of the body are accessible to this form of treatment, and opiates still present an important method of pain relief. Moreover, it is thought that judicious small amounts of opiates can be used in the treatment of pain if given by intravenous injection.

The aim of intravenous analgesia is to titrate small doses of either morphine or pethidine against the patient's pain threshold. In other words, following an initial 2 - 3 mg of morphine intravenously (depending on the size and the age of the patient), intravenous increments of 1 mg morphine may be repeated at half-hourly intervals until there is adequate pain relief without the unwanted sequel of respiratory depression. Such patients, however, will require careful monitoring of the respiratory rate, depth of respiration, blood pressure and pulse rate, and ideally this should be practised in an intensive care environment. Similarly, intravenous morphine is often of great advantage in alleviating the pain of vigorous chest physiotherapy when given 15 minutes beforehand.

ARTIFICIAL VENTILATION

In the past, intermittent positive pressure ventilation (IPPV) was associated with serious complications and a high mortality. With the exception of cases of poliomyelitis, IPPV was the last resort when all conservative measures had failed.

Today IPPV is not only more frequently used in the treatment of acute respiratory failure, but has a place in the prevention of ARF. When ARF is anticipated postoperatively, IPPV may be continued from the operating theatre to the ward. As a rule, in most centres, patients who have had an open-heart operation are ventilated for a minimum of 12 hours afterwards. The practice of artificial ventilation during the postoperative period has now extended to the general surgical wards, and postoperative IPPV is mainly indicated for those patients with poor lung function studies pre-operatively, certain neurosurgical operations, certain upper abdominal operations, especially where it is thought the patient will have much pain (abdominal thoracic incisions, etc.), and fixed cardiac output states.

It is usually unnecessary to ventilate these patients for over 48 hours, 12 - 24 hours being the mean extubation time.[10]

Equally vexing is the problem of when to initiate artificial ventilation in the patient already in respiratory failure. Respiratory failure is not usually a precipitous event, but more commonly the result of inadequate care and monitoring. There is no doubt that the morbidity and mortality of ARF increases with the severity, despite intervention with IPPV. The longer the delay in starting treatment, the more likely that IPPV at a late stage may in fact add to the damage, owing to the need for higher inspiratory oxygen concentration (F_iO_2) and the use of high tidal volumes and high airway pressure. As a guideline it is suggested that ventilatory support be initiated in adults when the respiratory rate increases to over 35/min, the pO_2 drops to under 70 mmHg while on oxygen, and the p_aCO_2 is over 55 mmHg.[11] These are, however, not absolute figures and it is very important to note the trend, e.g. regular monitoring of blood gases may show a progressive drop in pO_2 associated with an ever-increasing F_iO_2. The clinician's clinical judgement should be linked with the laboratory information.

It has recently been shown that the pattern of ventilation may profoundly affect the oxygenation and ventilation of the patient. During prolonged IPPV with a constant volume ventilator, it was noted that there was a progressive drop in p_aO_2 which was reversed by intermittent hyperventilation and/or sighs.[12] This has, however, been refuted by some observers.[13]

What does appear to be accepted is that ventilation with low tidal volumes tends to drop the p_aO_2, while larger tidal volumes (10 - 15 ml/kg) are able to maintain a better p_aO_2. No lung damage is noted at these high tidal volumes. Excessive low p_aCO_2 is treated by means of an external deadspace.[14]

The most important advance in the management of acute respiratory failure during the last 5 years has been the advent of PEEP (positive end expiratory pressure).[15,16] PEEP enables the patient to keep a positive and expiratory pressure in his lungs. It has been shown that if a patient exhales against a resistance this tends to keep small dependent airways open, and will therefore increase his FRC.[17] Thus, during inspiration more alveoli are kept open and the arterial pO_2 is improved. It is also thought that PEEP, by virtue of the increased pressure in the lungs, will push fluid out of the alveoli into the interstitial space and perhaps even into the pulmonary vasculature.[18] PEEP is usually introduced when, despite ever-increasing inspired oxygen content, the p_aO_2 continues to decrease. Absolute figures that may be used as a guide are a pO_2 of under 70 when the patient is on 50% oxygen and intermittent positive pressure ventilation.[15] Initially, PEEP was only used when a situation with the above figures had been reached, but there is an increasing tendency to use PEEP earlier in anticipation of further deterioration.

PEEP or constant positive pressure breathing (CPPB) can be used either with artificial ventilation with a ventilator, or by spontaneous breathing,[19] with PEEP ranging anywhere from 2 - 10 cm of H_2O. Levels of 15 cm and more have been recorded. PEEP may be simply applied to a ventilator by attaching the expiratory limb of the ventilator to a calibrated tube immersed in water.

In some patients PEEP may not improve the p_aO_2, and may even worsen it, and it may also result in two major complications: increased tendency to tension pneumothorax, and hypotension, usually in patients already dehydrated or hypovolaemic. According to Deane *et al.*[20] the incidence of tension pneumothorax has been markedly decreased by the use of non-depolarising agents on patients on CPPB, while deleterious drops in blood pressure can be avoided by adequate hydration of the patient.

I should like to thank Professor A. B. Bull, Head of the Department of Anaesthetics, University of Cape Town and Groote Schuur Hospital, for his encouragement, critical review and help with this manuscript.

REFERENCES

1. Bates, D. V., Macklem, P. T. and Christie, R. V. (1971): *Respiratory Function in Disease. An Introduction to the Integrated Study of the Lung*, 2nd ed., p. 442. Philadelphia: W. B. Saunders.
2. Burger, E. J. J. and Macklem, P. T. (1968): J. Appl. Physiol., **25**, 139.
3. Holland, J., Milic-Emili, J. and Macklem, P. T. (1968): J. Clin. Invest., **47**, 81.
4. Leblanc, P., Rutt, F. and Milic-Emili, J. (1970): J. Appl. Physiol., **28**, 448.
5. Holley, H. S., Milic-Emili, J. and Becklate, M. V. (1967): J. Clin. Invest., **46**, 475.
6. Don, H. F., Wahba, W. M. and Cuadrado, L. (1970): Anesthesiology, **32**, 521.
7. Don, H. F., Craig, D. B. and Wahba, W. M. (1971): *Ibid.*, **35**, 582.
8. Bendixen, H. H., Egbert, L. O. and Hedley-Whyte, J. (1965): *Respiratory Care*. St. Louis: C. V. Mosby.
9. Hughes, J. M. B. and Rosenzweig, D. Y. (1970): J. Appl. Physiol., **29**, 332.
10. Pontoppidan, H., Laver, M. B. and Geffin, B. (1970): Advanc. Surg., **4**, 163.
11. Pontoppidan, H., Geffin, B. and Lowenstein, E. (1972):: N. Engl. J. Med., **287**, 799.
12. Bendixen, H. H., Hedley-Whyte, J. and Laver, M. B. (1963): *Ibid.*, **269**, 991.
13. Bergman, N. A. (1970): Anesthesiology, **32**, 297.
14. Suwa, K., Geffin, B. and Pontoppidan, H. (1968): *Ibid.*, **29**, 1206.
15. Kumar, A., Falke, K. J. and Geffin, B. (1970): New Engl. J. Med., **283**, 1430.
16. Ashbaugh, D. G., Petty, T. L. and Bigelow, D. B. (1969): J. Thorac. Cardiovasc. Surg., **57**, 31.
17. McIntyre, R. W., Laws, A. K. and Ramachandran, P. R. (1969): Canad. Anaesth. Soc. J., **16**, 477.
18. Staub, N. C. (1970): Hum. Path., **1**, 419.
19. Gregory, G. A., Kitterman, J. A. and Phibbs, R. H. (1971): New Engl. J. Med., **284**, 1333.
20. Deane, R., Morgan, J. G. and Shinosaki, T. Personal communication.