EDITORIAL

A VENTILATORY STRATEGY FOR ACUTE RESPIRATORY FAILURE : WHERE "LESS IS MORE"

T K Lim

SINGAPORE MED J 1994; Vol 35: 343-344

Urgent management decisions regarding mode, timing, pace and level of support are often needed in patients with severe acute respiratory failure who are receiving mechanical ventilation in the critical care unit. These difficult decisions are made in the context of a plethora of information from blood tests, on-line monitoring devices, radiological images and bedside examination and during the course of rapidly evolving disease processes. An understanding of the basic physiological principles involved, target parameters, priorities and ways in which to reach these targets may facilitate decision making.

In the past decade we have witnessed rapid advances in theory as well as in practice, in the field of ventilatory support. However, controlled studies comparing different ventilator modes, regimens and techniques are not yet forthcoming and the critical information not incorporated into standard textbooks of medicine. Nevertheless, there is a large body of basic research in human and animal models coupled with a depth of experience which may form the basis for a strategic empiric approach to ventilator support in the critically ill patient. It may be appropriate at this time to make specific, practical recommendations with regards to ventilatory modalities and targets.

Some of the concepts which I will discuss have been addressed in detail by recent reviewers[1, 2]. The principles apply to restrictive as well as obstructive respiratory illnesses and are particularly relevant in patients who are severely ill and require a high level of ventilatory support. Hopefully, they might be of practical use to the frontline doctor in making a choice between therapeutic options.

Avoid Barotrauma

While barotrauma is traditionally associated with major air leaks and extensive subcutaneous or mediastinal emphysema, we have recognised in recent years that the consequences of barotrauma include impairment of cardiac pre-load, fall in cardiac output and net oxygen delivery plus further lung injury and aggravation of pulmonary edema[3,4]. Ventilatory strategies which use low airway pressures have been associated with improved survival in both status asthmaticus and the Adult Respiratory Distress Syndrome (ARDS)[5,6]. The peak airway pressure (Ppeak) should be kept below 40cm H_2O at all times, particularly during bag-ventilation by hand. This may require longer expiratory time (low respiratory rates), settings at low tidal volumes (Vt), low positive end-expiratory pressure (PEEP) and sedation with muscle paralysis. Both total PEEP (intrinsic PEEP is higher in severe airways obstruction) and mean airway pressure (Pmean) should also be monitored and kept as low as possible[7].

Department of Medicine
National University Hospital
Lower Kent Ridge Road
Singapore 0511

T K Lim, MBBS, M Med (Int Med), FAMS
Consultant Physician and Associate Professor

Use Appropriate PEEP

The level and pace of applied PEEP should be suited to the underlying cause of respiratory failure and not imposed on all patients in a uniform manner.

Applied PEEP is used in pulmonary edema (whether cardiogenic or non-cardiogenic ie in ARDS) to open up lung units which are available for further recruitment and useful function[8,9]. This reduces right to left intra-pulmonary shunting which should improve both lung compliance and oxygenation. In general the PEEP is optimal at around 10-15 cm H_2O and its use should be accompanied by clear improvement in oxygenation with mild increase in airway pressures and little change in haemodynamics. Continuous positive airway pressure applied via face mask may be so effective in treating hypoxemia that in some patients, it may be an alternative to tracheal intubation and full mechanical support[10].

Applied PEEP in patients with severe airways obstruction may reduce the trigger threshold load during inspiration on assist-control modes such as SIMV (synchronised intermittent mandatory ventilation). This should reduce work load and improve dyspnoea sensation[11-13]. Applied PEEP of 5 to 8 cm H_2O (always below 10cm H_2O) should be used only selectively in patients with severe airways obstruction who exhibit dynamic hyperinflation and are flow limited during tidal expiration[12, 13]. I found similarly that application of nasal CPAP at 5-8 cm H_2O achieved the best dyspnoea reduction in spontaneously breathing patients during acute exacerbation of chronic airways obstruction[14]. It is more likely to be helpful in chronic obstructive pulmonary disease and to be deleterious in status asthmaticus[11]. No changes in the degree of lung inflation, oxygenation and haemodynamics should result. Applying PEEP of above 10 cm H_2O will result in further hyperinflation, air trapping and fall in cardiac output.

Low FiO_2

Using supplemental oxygen with an FiO_2 of above 0.4 for prolonged periods may cause lung epithelial cell injury, perhaps via the activated inflammatory cascades and high energy oxygen radicals. The adjustment of oxygen supplement is most conveniently made with continuous monitoring by pulse oximetry (SpO_2). This may reduce both the need for repeat arterial gas measurements and delay time before oxygenation target is achieved. An SpO_2 of above 92% is probably safe and the patient need not be ventilated in order to target a "normal" saturation of > 95%[15]. During periods of decompensation and instability the FiO_2 might be increased for 6-12 hours with little risk. Nevertheless, concerted efforts

should be made to reduce FiO_2 to 0.4 as soon as the patient improves. If that is not possible then attempt to keep the FiO_2 as low as possible for a SpO_2 reading of >92%.

In case of a toss up, keeping a low airway distending pressure (Ppeak < 40 cm H_2O) takes precedence over keeping a low FiO_2 (<0.4). Major air leaks may be catastrophic and should be avoided at all costs even at the expense of increasing the FiO_2 to 1.0.

Low Vt

Current text books recommend volume preset ventilation and a Vt of above 10ml/kg body weight for mechanical ventilation in severe respiratory failure. Recent experimental work have shown that ventilation at even moderate Vt may be associated with lung edema[4]. Moreover, uncontrolled reports have linked low Vt ventilation to reduced mortality rates in both asthma and ARDS[5, 6]. Tidal volumes of as low as 5 ml/kg have been used, usually accompanied by carbon dioxide accumulation which should be monitored non-invasively via end-expired CO_2 capnography. This method of controlled or permissive hypercapnia is also consistent with the aim to keep a low Ppeak, Pmean and PEEP. The bonus is that low Vt ventilation may even improve cardiac output and tissue oxygen delivery (DO_2) in ARDS[16,17]. The safety of hypercapnia and its attendant acidosis needs to be further defined. A pH of > 7.25 is probably safe. Bicarbonate infusion for pH < 7.25 however may not improve either cardiac or respiratory function in this context[18].

Reversal of I:E ratio?

The reversal of the inspiratory:expiratory duty cycle (I:E ratio) from the normal of 1:3 to above 1:1 (Inverse ratio ventilation - IRV) have been advocated in patients with severe ARDS who need high distending airway pressures (Ppeak above 40-50 cmH_2O). This is an attempt to reduce Ppeak yet maintain oxygenation by keeping up the Pmean. The rationale is that the Ppeak may be more important for barotrauma and the Pmean for oxygenation[19]. It may spare the lung from further barotrauma and is an alternative to ventilating at high Ppeak and FiO_2. Inverse ratio ventilation usually require muscle paralysis and a controlled mode of ventilation. A high I:E ratio will result in autoPEEP and air trapping which is also not desirable. Moreover, controlled studies have not shown it to be superior to conventional ventilation. I find that Pmean and SpO_2 can usually be maintained by increasing the I:E ratio to 1:1 without actually reversing it. Fine tuning of the Pmean may then be achieved by altering the post inspiratory pause time with perhaps less increase in PEEP and thus air trapping than actual IRV.

A wide variety of new therapeutic modalities are under evaluation in the treatment of acute respiratory failure. They include biochemicals such as human surfactant and nitric oxide, physical techniques like tracheal insufflation and extra-corporeal membrane based gas renewal methods. Until these experimental treatments are proven effective in clinical trials and become widely available, we have to exploit ventilatory machine support as the primary treatment modality for most cases of respiratory failure. Application of a set of simple guidelines (Table I) may both facilitate therapeutic decision making and prevent complications.

Table I – This table summarises broad physiologic targets to be achieved when mechanical ventilation is delivered. Optimal goals have to be defined for individual patients and reviewed frequently.

Ventilatory Targets
1. SpO_2 > 92%
2. $FiO_2 \leqslant 0.4$
3. Ppeak $\leqslant$ 40 cm H_2O
4. Lowest Pmean & tPEEP
5. Low Vt 5-10 ml/kg
6. Respiratory rate 15-20/m
7. arterial pH $\geqslant$ 7.25
8. Best Bp, Ci, DO_2, MVO_2

Abbreviations:
SpO_2 - pulse O_2 saturation
FiO_2 - fractional inspired O_2
Ppeak, Pcam & tPEEP - peak, mean & total peep pressures
Vt - tidal volume
Bp - blood pressure
Ci - cardiac index
DO_2 - tissue O_2 delivery
MVO_2 - mixed venous O_2 saturation

References

1. Marini JJ. Patient-ventilator interaction:rational strategies for acute ventilatory management. Resp Care 1993; 38 : 482-93.
2. Kacmarek RM, Kickling KG. Permissive hypercapnia. Resp Care 1993; 38 : 372-87
3. Dreyfuss D, Soler P, Basset G, Saumon G. High inflation pressure pulmonary edema. Respective effects of high airway pressure, high tidal volume and PEEP. Am Rev Respir Dis 1988; 137 : 1159-64.
4. Corbridge TC, Wood LDH, Crawford GP, Chidoba MJ, Lanos T, Sznajol J. Adverse effect of large tidal volume ventilation with low PEEP in cannine acid aspiration. Am Rev Respir Dis 1990; 142 : 311-5.
5. Darioli R, Perret C. Mechanically controlled hypoventilation in status asthmaticus. Am Rev Respir Dis 1984; 129 : 385-7.
6. Hickling KG, Henderson SJ, Jackson J. Low mortality associated with low volume pressure limited ventilation with permissive hypercapnia in severe ARDS. Intensive Care Med 1990; 16 : 372-7.
7. Pepe PE, Marini JJ. Occult PEEP in mechanically ventilated patients with airflow obstruction. Am Rev Respir Dis 1982; 126 : 166-70.
8. Suter PM, Fairley HB, Isnderg MD. Optimal end-expiratory pressure in patients with acute respiratory failure. N Engl J Med 1975; 292 : 284-9.
9. Gattitoni L, Pesenti A, Avalli L, Rossi F. Pressure-volume curve of total respiratory system in acute respiratory failure. Computed tomography study. Am Rev Respir Dis 1987; 136 : 730-7.
10. Lee KH, Hui KP, Chin NK, Lim TK. Mask applied continuous positive airway Pressure: an alternative to intubation and mechanical ventilation. Third Congress of the Asia-Pacific Society of Respirology, Singapore 1993.
11. Tuxen DV. Detrimental effects of positive end-expiratory pressure during controlled mechanical ventilation of patients with severe airways obstruction. Am Rev Respir Dis 1989; 141 : 5-9.
12. Smith TC, Marini JJ. Impact of PEEP on lung mechanics and work of breathing in severe airflow obstruction. J Appl Physiol 1988; 65 : 1488-99.
13. Ranieri VM, Giuliani R, Cinnella G, Pesce C, Brienza N. Physiological effect of positive end-expiratory pressure in patients with chronic obstructive pulmonary disease during acute ventilatory failure and controlled mechanical ventilation. Am Rev Respir Dis 1993; 117 : 2-13.
14. Lim TK. Effect of nasal CPAP on patients with chronic obstructive pulmonary disease. Singapore Med J 1990; 31 : 233-7.
15. Lee KH, Hui KP, Lim TK. Factors influencing pulse oximetry compared to functional arterial saturation in multi-ethnic Singapore. Singapore Med J 1993; 34 : 385-7.
16. Kuski R, Takala J, Kari A, Milic-Emili J. Effect of tidal volume on gas exchange and oxygenation in ARDS. Am Rev Respir Dis 1992; 146 : 1131-5.
17. Tuxen DV, Williams TJ, Schienkestel CD, Czarny D, Bowles G. Use of measurement of pulmonary hyperinflation to control the level of mechanical ventilation in patients with acute severe asthma. Am Rev Respir Dis 1992; 146 : 1136-42.
18. Cooper DJ, Cailes JB, Scheinkestel CD, Tuxen DV. Does bicarbonate improve cardiac or respiratory function during respiratory acidosis in acute severe asthma - a prospective randomized study. Am Rev Respir Dis 1993; 147 : A614. (abstract)
19. Marcy TW, Marini JJ. Inverse ratio ventilation in ARDS: rationale and implementation. Chest 1991; 100 : 494-504.