EXTRACTS FROM THE JOURNALS

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Mechanical Ventilation in Acute Lung Injury (ALI) and Acute Respiratory Distress Syndrome (ARDS)[1]

ARDS develops in 25% of patients with sepsis, and in 8.7% of mechanically ventilated patients. Mortality in patients with ARDS is approximately 35% although death is usually due to multiple organ failure rather than to pulmonary failure.

There is increasing clinical and experimental evidence that poor ventilatory technique is harmful to the lungs. Ventilator induced lung injury (VILI) is thought to be caused by the application of physiological tidal volumes to the area of nonconsolidated alveoli. Delivering standard tidal volumes of 10 -12ml/kg inevitably causes over-distension of these alveoli. Only tidal volume limitation has been shown to improve survival in a randomised controlled trial [2]. Similarly, repeated opening and closing of alveoli during ventilation may exert substantial damaging shear forces on epithelial cells. All this evidence has stimulated the development of so-called protective strategies for mechanical ventilation that minimise further damage to the lung[3].

Protective strategies include:

- **Inspired oxygen concentration** - ventilation using high concentrations of inspired oxygen rapidly causes absorption atelectasis and possibly pulmonary cytotoxicity. Standard practice is to titrate the FIO_2 to an arterial oxygen saturation of 90%. However, patients who are treated with low tidal volumes will require a higher FIO_2 (0.56% versus 0.51%[2]) compared with those receiving traditional tidal volumes.

- **End-inspiratory volume/pressure**. In patients with ARDS mechanical ventilation with a lower tidal volume than is traditionally used, results in decreased mortality and increases the number of days without ventilator use. Therefore, standard practice in these severely ill patients should be to ventilate them with a tidal volume of 6 ml/kg and end-inspiratory pressures around 25cmH_2O. To accomplish a sufficient arterial oxygen saturation, respiratory rate has to be increased. These low tidal volumes may compromise alveolar ventilation and result in "permissive" hypercapnia, but this is generally felt to be preferable to worsening lung damage as long as the patient is not significantly acidotic, i.e. pH maintained >7.25. Disadvantages include raised intracranial pressure, impaired myocardial contractility and increased requirement for sedation.

- **End-expiratory volume/pressure**. Positive End Expiratory Pressure (PEEP) can prevent cyclic collapse and opening of alveoli hence preventing the generation of high shear forces across the alveolar epithelium (open lung theory). Unfortunately PEEP can also cause circulatory depression and may increase risks of barotrauma. The optimal level of PEEP to limit VILI is still under debate. Most clinicians have apparently adopted the approach to use the least PEEP necessary to achieve satisfactory arterial oxygenation with a limited inspired oxygen concentration. Lower tidal ventilation in general requires a higher PEEP[2]: 9.4 versus 8.6). Continuous maintenance of PEEP is vital, because a single breath without PEEP may result in alveolar collapse.

- **Mode of ventilation**. In Pressure Control Ventilation (PCV) a target inspiratory airway pressure can be selected preventing stretch induced lung injury. The resulting tidal volume depends on the compliance and resistance of the respiratory system. A deterioration in airways resistance or compliance, or a short inspiration, reduce tidal volume during PCV. A resulting drop in alveolar ventilation may cause severe hypercapnia and acidosis. In Inverse Ratio Ventilation (IRV) the duration of inspiration is extended and the duration of expiration is shortened. Arterial oxygenation improves by recruiting lung and reducing shunt but also through increasing mean airway pressure. The reduction in the expiratory time can cause hyperinflation (auto-PEEP). A reduction in respiratory rate allows prolongation of expiratory time but at the expense of alveolar ventilation.

- **Prone position**. Prone position is known to rapidly improve oxygenation in patients with ARDS due to improved regional ventilation and so reducing shunt. It has not yet been shown to improve survival.

Some very innovative technologies for supporting gas exchange in ALI and ARDS, such as partial liquid ventilation and high-frequency ventilation have yielded disappointing results in carefully conducted clinical trials, will require substantial investments by hospitals and can therefore not be recommended until improved outcome has been demonstrated.

1. Brower RG, Fressler HE. Mechanical ventilation in acute lung injury and acute respiratory distress syndrome. Clinics in Chest Medicine 2000;**21**:491-510

2. The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. New England Journal of Medicine 2000; **342**:1301-1308.

3. Finey JF, Evans TW. Mechanical ventilation in acute respiratory distress syndrome. Current Opinion in Anaesthesiology **14**:165-171

Haemodynamic Management of a Patient with Septic Shock[1]

Haemodynamic support in patients with septic shock aims to maintain perfusion of vital organs with oxygenated blood. In almost all patients with septic shock, adequate volume loading is likely to be the first and probably the most important step in supportive therapy. There are still no definitive prospective studies with respect to the best choice between crystalloid and colloid for restoring the intravascular volume. The adequacy of volume loading seems to be more important than the type of fluid used. [2] The decrease in haemoglobin concentration to 7-9g/dl is well tolerated by many patients, because the induced reduction in blood viscosity may reduce cardiac afterload, and the increased venous return may enhance cardiac output.

Treatment with vasopressors (norepinephrine = noradrenaline) is often indispensable in the therapeutic management of patients with septic shock. As long as the global oxygen supply is in the supranormal range, treatment with norepinephrine alone seems to be without negative effects on tissue oxygenation. Deterioration of tissue oxygenation may occur following the administration of epinephrine (adrenaline), pointing out the limited role for epinephrine in the treatment of septic patients.

When dobutamine is used to increase oxygen delivery, there is some evidence of improvement in tissue oxygenation. However, Hayes et al.[3] found no difference in global oxygen consumption between patients treated with dobutamine and those who were treated only with adequate volume therapy. Surprisingly, survival in the patients with the dobutamine treatment was lower than in the control group. There is some evidence that low dose dobutamine may maintain better splanchnic oxygenation in patients on high doses of norepinephrine.

It remains questionable whether the management of septic shock with high doses of dopamine alone is superior to the combination of dobutamine and norepinephrine. It is doubtful that low dose dopamine can prevent renal failure, and there is evidence that low-dose dopamine has deleterious effects on splanchnic oxygenation.

Summary of the most important steps in supportive treatment of sepsis [1]

- Find and treat the septic source
- Surgical therapy, removal of iv lines, antibiotics
- Maintain adequate volume status
- Fluid resuscitation guided by parameters of global haemodynamics (peripheral temperature, CVP) and organ function (like urine output.)
- Maintain adequate oxygen delivery
- Early intubation and ventilation, low dose dobutamine
- Maintain an adequate perfusion
- Norepinephrine

see also Update in Anaesthesia Number 13 - The Management of Septic Shock

1. Reinhard K, Sakka SG, Meier-Hellmann A.: Haemodynamic management of a patient with septic shock. European Journal of Anaesthesiology 2000;**17**: 6-17

2. Schierhout G, Roberts I. Fluid resuscitation with colloid or crystalloid solutions in critically ill patients: a systemic review of randomised trials. British Medical Journal 1998;**316**:961-964

3. Hayes MA et al. Elevation of systemic oxygen delivery in the treatment of critically ill patients. New England Journal of Medicine 1994;**330**:1717-1722