

ONE-LUNG ANAESTHESIA VENTILATION DURING THORACOTOMY

Brig. M. Salim,

FCPS, FRCA, PhD

Islamic International Medical College, Rawalpindi

Ventilation during thoracotomies becomes a challenge for the anaesthesiologist for a number of reasons. Such patients have usually both lungs effected by the disease to a variable extent. Lateral position itself has physiological consequences. Loss of large amounts of blood, difficult approach to the airway during the surgery, and ultimately loss of lung mass pose other problems to be dealt with. Thus a working knowledge of the practical approach towards ventilation is required for a successful outcome.

VENTILATION DURING THORACOTOMY

In the awake subject in the lateral position, blood flow to the dependent lung increases to approximately 60% of the total because of the effect of gravity on the low pressure pulmonary vasculature. This increase in blood flow is matched by an increase in ventilation as the lower lung is on the steep part of the pressure-volume curve and the lower diaphragm, which is pushed into the chest by abdominal contents, contracts more effectively from a position of mechanical advantage.

During anaesthesia in spontaneously breathing patients the situation changes. There is a reduction in functional residual capacity (FRC) and both lungs decrease in volume. The non-dependant upper lung now moves to the steeper part of the pressure-volume curve and receives more ventilation. Additionally, there is loss of diaphragmatic tone. The result of these changes is that the upper, non-dependant, lung is preferentially ventilated whilst the increased pulmonary blood flow to the lower lung continues.

Paralysis and intermittent positive pressure ventilation (IPPV) are used during thoracotomy to overcome the problems of the open pneumothorax created by surgery. The compliance of the non-dependent lung remains higher than that of the lower lung during IPPV so that preferential ventilation continues to the upper lung and may be further accentuated when the chest is opened.

One-lung ventilation is employed at various times during lung resection primarily to improve surgical

access to the upper non-ventilated lung. This eliminates preferential ventilation but creates a far more serious problem of ventilation/perfusion mismatch.

PHYSIOLOGY OF ONE-LUNG ANAESTHESIA

VENOUS ADMIXTURE

Pulmonary blood flow continues to the upper lung during one-lung anaesthesia, creating a true shunt in a lung where there is blood flow to the alveoli but no ventilation. This shunt is the major cause of hypoxaemia during one-lung ventilation, although the alveoli with low ventilation/perfusion ratios in the dependent lung contribute to some extent. The blood to the upper lung can not take up oxygen and therefore retains its poorly oxygenated mixed venous composition. This mixes with oxygenated blood in the left atrium causing venous admixture and lowering arterial oxygen tension (PaO_2). Total venous admixture can be calculated from the shunt equation (Nunn. 1993) which estimates what proportion of the pulmonary blood flow would have bypassed ventilated alveoli to produce the arterial blood gas values for a particular patient. In published work on one-lung anaesthesia the terms venous admixture and shunt (Q_s/Q_t) are used synonymously.

Venous admixture increases from a baseline value of approximately 10-15% during two-lung ventilation to level of 30-40% during one-lung ventilation. The PaO_2 can be maintained in the safe range of 9-16 kPa with an inspired oxygen concentration between 50% and 100% in the majority of patients. In individual patients, however, the PaO_2 may fall considerably lower than this, despite a high inspired oxygen concentration. This variation is hardly surprising considering the number of interrelated physiological factors that come into play.

THE ROLE OF HYPOXIC PULMONARY VASOCONSTRICTION

Hypoxic pulmonary vasoconstriction (HPV) is a homeostatic mechanism whereby pulmonary blood flow is diverted away from hypoxic areas of lung, there by

optimizing oxygenation of the arterial blood (Eisenkraft, 1994). The major stimulus for HPV is hypoxia both in the alveoli (low PaO₂) and blood perfusing the lungs (low mixed venous oxygen). The precise mechanism of HPV remains unknown but it appears to be produced by each smooth muscle cell in the wall of the pulmonary artery responding to oxygen tension in its vicinity. The smooth muscle in the wall of the arteries depolarizes and develops spontaneous electrical activity in response to hypoxia. Nitric oxide production may be inhibited by hypoxia so this also could be implicated in the mechanism of HPV.

HPV is not related to innervation of the lung as it occurs after lung transplantation. It is inhibited in a variety of pulmonary pathologies including adult respiratory distress syndrome (ARDS) and pneumonia. Many other factors inhibit HPV. These include pulmonary vasodilator drugs, high pulmonary vascular pressure, alkalosis, acidosis, hypothermia, positive end expiratory pressure (PEEP), volatile anaesthetic agents and handling of the lung. In-vitro experiments have clearly shown that volatile anaesthetic agents inhibit HPV but in-vivo studies it has been difficult to demonstrate inhibition. This is because, although volatile agents do depress HPV directly, they also enhance HPV indirectly by reducing cardiac output. (as cardiac output drops there is an enhancement of the HPV response). There is, therefore, an apparent unchanged HPV response in the presence of volatile anaesthetic agents during one-lung anaesthesia.

Intravenous anaesthetic agents do not inhibit HPV but the majority of studies have failed to demonstrate a significant benefit, in terms of arterial PaO₂, when they are used to provide anaesthesia during one-lung ventilation.

HPV seems, on current evidence, to play little role in reducing hypoxaemia during the time it takes to complete the average lung resection. It must also be remembered when reading the literature, that handling of the lung reduces HPV and this may have a very significant effect in clinical practice. Potent inhaled anaesthetic agents such as isoflurane are not contraindicated during one-lung ventilation and may even be desirable because of their bronchodilator properties and ease of use. Significant inhibition of HPV is more likely with halothane and therefore this drug, although now rarely used in adult practice, should be avoided altogether during lung resection. Finally, despite the comments above, there may be some cases where, if PaO₂ is very low during one-lung ventilation, it is worth changing from an inhalational anaesthetic to an intravenous technique. In practice this probably means substituting an isoflurane based anaesthetic with a total intravenous anaesthesia (TIVA) technique using propofol, and monitoring its effect

on arterial oxygenation.

CARDIAC OUTPUT

Changes in cardiac output are likely to affect arterial oxygenation during thoracotomy. If oxygenation remains steady, a decrease in cardiac output creates a reduced mixed venous oxygen content. Some of this desaturated blood is shunted during one-lung ventilation and further exacerbates arterial hypoxaemia. Cardiac output can decrease for a number of reasons during thoracotomy. These include blood loss and fluid depletion, the use of high inflation pressures, the application of PEEP to the dependent lung and the use of PEEP to the lower lung combined with continuous positive airways pressure (CPAP) to the upper lung.

Simple measures such as replacement of blood loss, attention to fluid balance and adjustment of ventilation can help maintain cardiac output. Surgical manipulation and retraction around the mediastinum, causing a reduction in venous return, are probably the commonest causes of a sudden drop in cardiac output during lung resection. Hence the requirement for invasive arterial and venous pressure monitoring.

PRE-OPERATIVE LUNG FUNCTION

Diseased lung may have a considerably reduced blood supply as a result of hypoxic pulmonary vasoconstriction or, in some circumstances, for physical reasons such as collapse, consolidation, cavitation or infiltration by tumour.

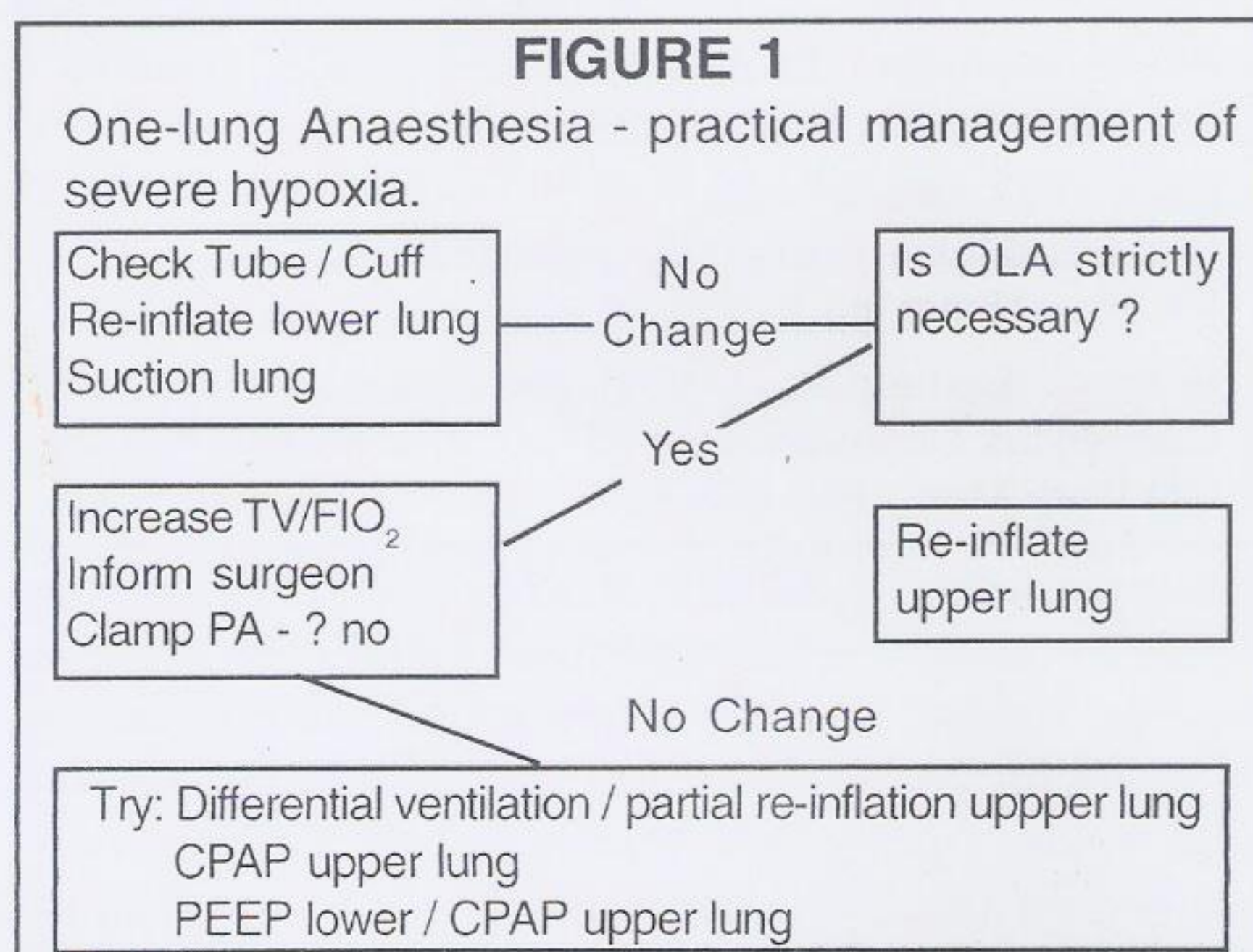
Patients with poor lung function are sometimes accepted for lung resection on the basis that their diseased lung is contributing little to gas exchange. If this type of pulmonary disease is largely confined to the side of surgery one-lung anaesthesia may have little effect on gas exchange. Conversely, patients with near normal lung function are more likely to be hypoxic during one-lung anaesthesia. In a classic study by Kerr (1974) it was reported that patients undergoing lung resection tended to have better arterial oxygenation during one-lung anaesthesia than those undergoing non-resection procedures such as oesophageal surgery. It was presumed that in the latter group an essentially normal lung was being collapsed to provide surgical access. Katz (1982) also found that patients with a pre-operative FEV1 nearest the predicted normal value were more likely to be hypoxic during one-lung anaesthesia.

MANAGEMENT OF ONE-LUNG VENTILATION (OLV)

It is of paramount importance to ventilate the dependent single lung optimally during one-lung anaesthesia. In principle adequate ventilation should be established in such a way as to minimize intra-alveolar

pressure and therefore prevent diversion of pulmonary blood flow to the upper, non-ventilated, lung. In practice this is not always easy to achieve.

We usually use an inspired oxygen concentration (FIO_2) of 50% (0.5) during thoracic surgery, with an inspired gas mixture of oxygen/nitrous oxide or air/oxygen. After OLV is established we increase the oxygen concentration to a concentration as high as 100%, if required (see below). Other authors, particularly



North American, recommend 100% oxygen from the outset.

The majority of operating theatre ventilators are of the volume-controlled type. In order to minimize mean intra-alveolar pressure a tidal volume of approximately 8ml/kg is chosen initially with a conventional inspired / expired ratio (I:E ratio) of 1:2. If the minute volume is kept the same for OLV as it was during two-lung ventilation, elimination of carbon dioxide is rarely a problem. A certain amount of hypercapnia is tolerated if necessary, however, and this can be monitored by end-tidal readings.

If inflation pressure is particularly high at the onset of OLV (i.e. more than 35 cm H_2O) it may be necessary to reduce the tidal volume and/or reassess the position of the double-lumen tube. Hyperinflation of the lungs (volutrauma) is probably more damaging to the lungs than barotrauma and should be avoided if at all possible during OLV because of its part in the aetiology of post pneumonectomy pulmonary oedema. There is, however, limited evidence (Tugrul et al 1997) that pressure-controlled ventilation may be more appropriate for OLV. This method of ventilation can obviously be useful in limiting airway pressures but, as previously mentioned, most operating theatre ventilators do not currently provide this facility.

If inflation pressure remains high it may be helpful

to analyze a flow/volume loop or at least manually inflate the dependent lung to get a "feel" for the compliance. A marked reduction or disappearance of the end-tidal carbon dioxide trace at this stage usually means there is a major problem with tube position or bronchial cuff inflation. Should arterial oxygen saturation drop below 90% there are a number of steps (Fig 1) which can be taken to improve the situation. We initially check the position of the double-lumen tube and re-adjust this as necessary. (When OLV becomes one lobe ventilation hypoxaemia is inevitable.) Suction and manual re-inflation of the dependent lung may also be helpful at this stage.

The above steps may fail to improve oxygenation and therefore the inspired oxygen concentration is increased up to 100% as necessary, taking steps to prevent patient awareness. The increase in inspired oxygen concentration cannot affect the "true shunt" through the upper lung but will improve oxygenation via the alveoli with low ventilation/perfusion ratios in the lower lung. If this step fails to improve oxygenation it may be prudent to re-inflate the upper lung at this stage, providing the surgeon is happy to tolerate this. In theory the shunt can be eliminated during, OLV by clamping the pulmonary artery in the upper lung. This is impractical during surgery for lung cancer, however. By the time the pulmonary artery is fully dissected and exposed for clamping the period of OLV is nearly at an end. Attempts to clamp the artery earlier would be detrimental to the principles of cancer surgery, because dissection of nodes within the hilum and around the pulmonary artery needs to be carried out in a systematic manner.

Insufflation of oxygen to the upper lung via a CPAP circuit or even partial re-inflation of the upper lung with oxygen will improve oxygenation during OLV. This supplemental oxygen is taken up by the blood flow remaining to the upper lung and therefore the shunt fraction is decreased. CPAP can be applied at approximately 5-10 cm H_2O via commercially available CPAP circuits or, more simply, oxygen can be insufflated via a suction catheter to a partially re-inflated lung. The main drawback of this technique is that the lung becomes distended and may hinder surgery.

PEEP applied to the lower lung is not generally helpful in improving oxygenation, presumably because it tends to divert pulmonary blood flow to the upper non-ventilated lung. Some authors recommend the use of PEEP to the lower lung combined with CPAP to the upper lung to overcome this problem. In practice it is rarely necessary to use this combination of techniques during the course of a lung resection of average duration. This technique may improve arterial oxygenation, but the PEEP/CPAP combination is likely to decrease cardiac output and reduce oxygen delivery.

As stated above, PEEP is statistically unlikely to improve oxygenation during OLV in the majority of patients. A recent study, however, (Cohen and Eisenkraft 1996), albeit in a small number of patients, has suggested that PEEP may be beneficial in those patients with a PaO₂ in the lower range (below 80mmHg or 10.6kPa). The authors theorize that if PEEP restores FRC to normal from an initially low level in hypoxic patients the pulmonary vascular resistance should decrease. This results in increased blood flow through the ventilated lung, a decreased shunt and an improvement in arterial oxygenation. Lower lung PEEP may therefore be useful in these circumstances and should not be dismissed as a possible therapeutic option. On the other hand, if PEEP increases the FRC from an initially normal value pulmonary vascular resistance would increase and a larger proportion of the blood flow would be shunted through the non-dependent lung resulting in a decrease in PaO₂.

In the face of persistent arterial hypoxaemia during OLV it may be pertinent to ask "What is a low PaO₂ for this patient?" We take a figure of 90% oxygen saturation below which we start worrying about the effects of hypoxia on cerebral, cardiac, and renal function and general tissue oxygenation. This is an arbitrary figure, however, which is affected by a variety of factors, including acidosis and temperature. In addition, many patients will have a low PaO₂ when measured breathing air pre-operatively, hence the usefulness of this pre-operative measurement.

Arterial hypoxaemia is obviously undesirable but in the anaesthetized patient it may be preferable to accept a PaO₂ slightly lower than the pre-operative value, rather than undertake measures such as upper lung inflation which may hinder and prolong surgery.

ACUTE LUNG INJURY AND. ONE-LUNG ANAESTHESIA

Lung injury has been recognised as a potential complication of lung resection for many years and has been termed post pneumonectomy pulmonary oedema (PPE). In its extreme form, PPE represents one cause of the acute respiratory distress syndrome (ARDS), although many patients not meeting the diagnostic criteria for ARDS fulfill those for the less severe acute lung injury (ALI) (see Table 1). Despite the terminology, PPE is not confined to pneumonectomy patients but also occurs following lobectomy. PPE complicates 4-7% of pneumonectomies and 1-7% of lobectomies, with a very high associated mortality of 50-100%. These statistics do not take into account the incidence of ALI following lung resection however. Figures for ARDS and ALI following lung resection in Royal Brompton Hospital

TABLE 1

Definitions of acute lung injury (ALI) and acute respiratory distress Syndrome (ARDS)

Acute lung injury (ALI) in post-thoracotomy respiratory failure requires the following for a diagnosis:

- * an arterial oxygen tension (PaO₂, kpa) : inspired oxygen concentration (FiO₂) ratio of < 40.
- * diffuse, bilateral pulmonary infiltrates on chest x-ray.
- * a pulmonary capillary occlusion pressure (PAOP) of < 18mm Hg

Acute respiratory distress syndrome (ARDS) requires the following for diagnosis:

- PaO₂ : FiO₂ ratio of < 25
- bilateral diffuse pulmonary infiltrates
- PAOP < 18mm Hg

From: Bernard et al (1994). The American-European Consensus Conference on ARDS. American J. Respir Crit Care Med; 149, 818-824.

are summarised in Table 2. It is becoming increasingly evident that ischaemia-reperfusion injury mediated by reactive oxygen species is implicated in the aetiology of pulmonary injury following lung resection (Williams et al; 1996). During OLV relative ischaemia of the non-dependent lung being operated upon is followed by re-expansion and reperfusion of the remaining lung tissue after lobectomy and by hyperperfusion of the ventilated lung after pneumonectomy. It is therefore likely that all patients undergoing lung resection and OLV are

TABLE 2

Lung injury following lung resection- a retrospective study. Royal Brompton Hospital 1991-1994

All cases of ALI/ARDS identified using criteria outlined in Table 1.

	No. cases	ARDS (n=17)	ALI (n=7)
Pneumonectomy	103	5 (4.8 %)	2 (1.9 %)
Lobectomy	231	12 (5.2 %)	5 (2.2 %)
Wedge resection	135	0	0
Total cases	469	5 (3.6 %)	5 (1.5 %)
Total deaths (overall)	-	15	2
% Deaths of total	-	3.4	0.5

Summary:-lung injury (ALI + ARDS) occurred in 5.1% of patients

mortality for ARDS (once established) was 88%

mortality for ALI was 29%

Data: Courtesy of Prof T. Evans and Dr E. Williams.

Dept of Critical Care Royal Brompton Hospital, London UK.

subjected to conditions under which there is an increased risk of developing lung injury. Factors that determine the degree of endothelial damage in ALI and the release of vasoactive substances such as nitric oxide (NO), and their influence in modulating HPV have yet to be elucidated. Whether further work in this field can lead

to management strategies which will decrease the incidence of ALI following lung resection is also open to question.

NEWER THERAPEUTIC MODALITIES AND ONE-LUNG ANAESTHESIA

Ventilation, as described above, is the main area in which changes are made in order to reduce hypoxia during OLV. Increasing interest is being shown in the pharmacological manipulation of pulmonary blood flow during OLV but this is at an early stage of investigation. The pulmonary vasodilator prostaglandin E1 has been selectively infused into the pulmonary artery of the ventilated lung in adult patients undergoing OLV (Chen, 1996). This has been found to reduce venous admixture/shunt fraction (Q_s/Q_t), improve arterial oxygenation and lower pulmonary vascular resistance. Selective infusion into one single pulmonary artery is relatively difficult to achieve and other workers have studied the effects of inhaled nitric oxide (NO) into the dependent, ventilated, lung during OLV. One of the first studies of this type (Wilson, 1997) failed to show any benefit from this technique in adult patients. There is, however, some animal evidence that NO may be helpful during one-lung anaesthesia and further clinical studies are required in this area. The NO synthetase inhibitor nitro-L-arginine methyl ester (L-NAME) has been used intravenously and in nebulized form in animal experiments to block the production of NO and decrease blood flow to hypoxic areas of lung. This approach could potentially be used to reduce blood flow to the non-ventilated lung during one-lung anaesthesia. Again, this form of treatment is at a very early stage of investigation.

HIGH FREQUENCY JET VENTILATION

High frequency jet ventilation (HFJV) can be used to provide satisfactory gas exchange during thoracotomy either via an endotracheal tube or some form of endobronchial tube. HFJV provides satisfactory ventilation by either route and has the advantage of low peak airway pressures, albeit with the production of obligatory PEEP by the majority of systems. Some clinicians advocate the use of HFJV during thoracic surgery and use it on a routine basis. This method of ventilation has not been adopted widely, however. Reasons for this include difficulty with surgical access as the lung is distended and the inability to administer gaseous anaesthetic agents. HFJV may have a role to play in the management of specific conditions and it has been advocated for a variety of procedures including bilateral bullectomy, management of bronchopleural fistula, sleeve resection of the right upper lobe and airway surgery.

TERMINATION OF SURGERY AND ANAESTHESIA

TESTING OF BRONCHIAL SUTURE LINES

On completion of lung resection bronchial suture lines and lung surfaces are tested for an air leak. Sterile water is instilled into the pleural cavity, following cancer surgery, to cover the bronchial suture lines. After lobectomy the remaining lobe or lobes are then suctioned prior to re-inflation, in the case of pneumonectomy very gentle suction is applied with a soft catheter to the bronchial stump. At this stage it may also be helpful to deflate the bronchial cuff. The bronchial stump is then tested for a leak as a positive pressure of approximately 30cmH₂O is applied manually in a sustained manner via the ventilation circuit to both lumina of the double-lumen tube. (We now keep this testing pressure down for fear of increasing the risk of ALI in the post-operative period).

In the unlikely event of a leak being present, gas bubbles will be seen appearing below the water level in the pleural cavity, indicating the need for further surgery.

We tend to test for lung surface leaks at a lower inflation pressure of approximately 20cm H₂O. A degree of lung leak may be tolerated from a raw surface, if it is thought the remaining lung tissue will expand satisfactorily to fill the thoracic cavity, but a large leak may warrant further suturing.

REFERENCES

- Chen T.-L, Lee Y.-T, Wang M.-J. et al (1996) Endothelin-T concentrations and optimisation of arterial oxygenation and venous admixture by selective pulmonary artery infusion of prostaglandin E₁ during thoracotomy. *Anaesthesia*, 51, 422-426.
- Cohen E. and Eisenkraft J.B. (1996) Positive end-expiratory pressure during one-lung ventilation improves oxygenation in patients with low arterial oxygen tensions. *J. Cardiothorac Vasc Anesth*, 10, 578-582
- Eisenkraft J.B. (1994) Anaesthesia and hypoxic pulmonary vasoconstriction. In: *Recent Advances in Anaesthesia and Analgesia* (R.S. Atkinson and A.P. Adams, eds) Vol 18, Chap 7, 103-122. Churchill Livingstone.
- Katz J.A., Laverne R.G., Fairley H.B. et al (1982) Pulmonary oxygen exchange during endobronchial anaesthesia. *Anesthesiology*, 56, 164-171.
- Kerr J.H., Crampton Smith A., Prys-Roberts C. et al (1974) Observations during endobronchial anaesthesia II: oxygenation. *British Journal of Anaesthesia*, 46, 86-92.
- Nunn J.F. (1993) *Applied Respiratory Physiology* (4th Edition) Butterworth Heinemann, London.
- Tugrul M., Camci E., Karadeniz H. et al (1997) Comparison of volume controlled with pressure controlled ventilation during one-lung anaesthesia. *British Journal of Anaesthesia*, 79, 306-310.
- Williams E. A., Evans T.W. and Goldstraw P. (1996) Acute lung injury following lung resection: is one lung anaesthesia to blame? *Editorial. Thorax*, 51, 114-116.
- Wilson W.C., Kapelanski D.P. et al (1997) Inhaled nitric oxide (40 ppm) during one-lung ventilation, in the lateral decubitus position does not decrease pulmonary vascular resistance or improve oxygenation in normal patients. *J. Cardiothorac Vasc Anesth.*, 11, 172-176.