Chronic obstructive pulmonary disease
Chronic obstructive pulmonary disease (COPD) is a common condition with high morbidity and mortality rates.1 The condition, which is primarily a complication of smoking, is a chronic, slowly progressive disorder characterised by airway obstruction.2 The definition includes chronic bronchitis and emphysema with permanent destructive enlargement of distal pulmonary airspaces. Consequently, there is loss of normal lung architecture resulting in loss of elastic recoil of lung tissue leading to collapse of small airways, expiratory airflow limitation, air trapping, hyperinflation of the lungs and progressive enlargement of the thoracic cage. Expansion of the thorax leads to flattening of the diaphragm, in-drawing of the lower ribs and compromised chest wall mechanics. The ribs are lifted and flattened leading to increased total lung capacity and residual volume, with reduced FEV1, and increased work of breathing. As the disease progresses, patients must breathe at a higher lung volume to achieve the flows necessary to meet ventilatory requirements. At end-stage disease, the patient is dyspnoeic and has a severely restricted exercise capacity.3 Once the patient has reached a stage where the FEV1 < 0.75 L, the 1-year mortality is in the region of 30%4 and the patient will require frequent hospital admission for treatment of exacerbations of the condition.

Medical management options
The goals of therapy in emphysema are to halt the progressive decline in lung function, prevent exacerbations of the disease, improve exercise capacity and quality of life, and prolong survival. The only treatment shown to alter the rate of progression of COPD is cessation of smoking.5 Exacerbations of disease are treated with antibiotics, steroids, β-adrenergic agonists, theophylline, and anticholinergics. Although these interventions shorten the duration of individual episodes and minimize symptoms, there is little evidence that they alter the natural history of the disease or reduce mortality. Bronchodilators improve lung function, exercise capacity, and quality of life in patients with COPD, but are of limited benefit to patients without reversible airway disease. As the medical management of these patients appears to offer only limited benefits, various attempts have been made to improve the quality of life and possibly to reduce mortality through a variety of surgical techniques over the past 90 years including pneumoperitoneum formation, phrenic nerve paralysis, thoracoplasty, denervation of the lung, and stabilization and fixation of the trachea. None of these techniques resulted in any substantial benefit to the patients.

Anaesthesia for Lung Volume Reduction Surgery

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Surgical management
Lung volume reduction surgery as an approach to the management of severe emphysema was first described by Brantigan in 1959.6 In this technique, multiple resections of hyperinflated lung areas were performed through a standard thoracotomy. Although 75% of patients reported clinical improvement, the lack of objective documentation of benefit from the procedure, and an operative mortality of 18% prevented widespread acceptance of the procedure.7 Subsequently, Cooper et al7 reported a modified Brantigan procedure in 1995, in which 20-30% of lung tissue was resected after median sternotomy. In the initial series of 20 patients undergoing lung volume reduction surgery with this method, there was no operative mortality, and the operation produced an 82% increase in FEV1 associated with marked relief of dyspnoea and improvement in exercise tolerance and quality of life.7 Since then there have been a number of reports describing acceptable levels of perioperative mortality (<5%) and varying degrees of improvement in respiratory function.8-10

Respiratory function appears to improve following lung volume reduction through improvement in diaphragm and chest wall function as well as better gas exchange. Resection of the most grossly over-distended lung segments allows for some re-expansion of previously compressed but less compromised lung tissue. The reduction in total lung volume improves the shape of the chest wall and the position of the diaphragm, resulting in a reduction in the work of breathing that is evident within 24 hours of the procedure. However, improved dia-
Phrenic function may take several months to develop. Re-expansion of areas of lung with better elastic properties results in improved lung recoil, which in turn reduces ventilation-perfusion mismatch, expiratory flow limitation and dynamic pulmonary hyperinflation. These physiological improvements may result in an increased FEV1, together with reduced steroid dependence and reduced reliance on supplemental oxygen. To date, it seems accepted that selected patients with end-stage emphysema benefit from lung volume reduction surgery by means of early improvement of ventilatory mechanics and by somewhat later improvement in dia-phragmatic function. The precise mechanisms and the duration of improvement, however, remain unclear.

Cardiac function is also improved following successful lung volume reduction surgery. The reduction in dynamic hyperinflation decreases the tamponade of the right atrium and particularly the right ventricle, resulting in improved right ventricular filling and performance. Similar improvements in left ventricular filling and consequentially left ventricular performance have also been described recently.

These benefits have resulted in improved exercise tolerance and a better quality of life in appropriately selected patients which appear to be sustained for up to 1 year in patients on a pulmonary rehabilitation programme. However, the basic pathological process is unchanged and continues to lead to a decline in lung function following surgery, and most patients returned to baseline pulmonary function between 18 months and 2 years after the procedure. Nevertheless, several controlled trials support the contention that these patients have better palliation of their respiratory condition than comparable patients on medical therapy, The National Emphysema Treatment Trial (NETT), a federally sponsored, multicentre, randomised clinical trial, found no significant difference in overall mortality between selected patients undergoing medical or surgical treatment. However, at two years from entry into the trial, only 3% of medically treated patients had achieved the targeted increases in exercise capacity as compared to 15% of the surgically treated patients (p<0.001).

From this review, it is clear that lung volume reduction surgery is a technique that has the potential to improve pulmonary function in carefully selected patients, but it is also a technique that carries significant morbidity and mortality, particularly in patients whose physiological status and anatomical pattern of disease are less suitable to the procedure. Patient selection is therefore critically important.

**Patient selection**

Whilst there is no overall consensus regarding patient selection for lung volume reduction surgery, typical criteria from the United States and from Europe are included in Table I (adapted from reference 5):

<table>
<thead>
<tr>
<th>United States</th>
<th>Europe</th>
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<tbody>
<tr>
<td>Diagnosis of chronic obstructive pulmonary disease (patient history, physical examination, lung function test, chest radiograph, etc.)</td>
<td>Diagnosis of chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td>Smoking cessation for more than 1 mo</td>
<td>Smoking cessation for more than 6 mo</td>
</tr>
<tr>
<td>Age &lt; 75 y</td>
<td>Age &lt; 75 y</td>
</tr>
<tr>
<td>FEV1 between 15% and 35% of predicted</td>
<td>FEV1 between 10% and 40% of predicted</td>
</tr>
<tr>
<td>PaCO2 &lt; 55 mm Hg</td>
<td>PaCO2 &lt; 65 mm Hg</td>
</tr>
<tr>
<td>Prednisone dosage &lt; 20 mg/d</td>
<td>Prednisone dosage &lt; 20 mg/d</td>
</tr>
<tr>
<td>PAP &lt; 50 mm Hg</td>
<td>PAPsys &lt; 0.5 X SAPsys</td>
</tr>
<tr>
<td>No previous thoracotomy or pleurodesis</td>
<td>No previous bilateral thoracotomy or pleurodesis</td>
</tr>
<tr>
<td>Absence of symptomatic coronary artery disease</td>
<td>Absence of symptomatic coronary artery disease; absence of significant coronary stenosis, verified by left heart catheterization</td>
</tr>
<tr>
<td>Absence of chronic asthma or bronchitis</td>
<td>Absence of significant chronic asthma and bronchitis</td>
</tr>
<tr>
<td>Commitment to preoperative and postoperative supervised pulmonary rehabilitation for 6 wk</td>
<td>Commitment to preoperative and postoperative supervised pulmonary rehabilitation</td>
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The NETT study also demonstrated that the distribution of the pulmonary disease was an important contributing factor to patient outcome. Patients with predominantly upper lobe disease and low baseline exercise capacity in the surgical group had a lower risk of death than equivalent medically-treated patients and also had a far greater likelihood of achieving targeted increases in exercise capacity. They were also more likely to have improved quality of life at 2 years. By contrast, patients whose disease was not confined to the upper lobe, and who had good exercise capacity had a higher mortality with surgical treatment and similar quality of life to medically-treated patients at 2 years. It is important to note that, even in patients who demonstrated improved quality of life after 24 months relative to their comparator group had, in fact, returned to pre-surgery baseline, whereas their medically treated equivalent group had declined below the baseline. Although these data cannot be regarded as definitive at present time, it does appear that lung volume reduction surgery is probably beneficial in patients meeting the above criteria who also have anatomically favourable disease and a low exercise capacity.

**Preoperative preparation**

Most patients presenting for this procedure will be on maximal medical therapy for their chronic obstructive lung disease, including bronchodilator therapy, steroids and intermittent antibiotics. Most centres recommend optimizing and continuing maximal bronchodilator and mucolytic therapy before lung volume reduction surgery, including the day of surgery. The patient must be free of respiratory infections for at least 3 weeks before lung volume reduction surgery, and must require no antibiotic therapy preoperatively. Most patients present with productive cough. If the sputum is purulent, surgery should
be delayed for aggressive treatment of the respiratory infection. Steroid therapy should be reduced to the lowest tolerated dose before lung volume reduction surgery.

Other input regarding the patient’s fitness for the procedure should come from support programs including physiotherapy, occupational therapy and social services, with the aim of maximising exercise capacity prior to surgery. Cessation of smoking is mandatory, although the period for which this is required varies from centre to centre. Good psychological preparation of the patient is also important, and the patients must be willing and able to undertake rigorous rehabilitation in the postoperative period.

Anaesthetic management
Successful conduct of lung volume reduction surgery requires careful teamwork between physicians, surgeons and anaesthetists as well as critical care and physiotherapy practitioners. It is important for the anaesthetist to establish a good rapport with the patient as part of the perioperative preparation.

Premedication
Most centres do not recommend sedative premedication other than mild anxiolysis with a benzodiazepine, as these patients cannot tolerate heavy sedation. Intravenous midazolam may be useful in the anaesthetic room for invasive preanaesthetic procedures.

Anaesthetic technique
Most centres favour the use of thoracic epidural anaesthesia with local anaesthetic agents for intraoperative management and a combination of local anaesthetic and opioid by infusion for postoperative pain relief. Some have argued that the use of lumbar epidural analgesia with diamorphine reduced the requirement for vasopressor agents during the procedure and avoided the risk of impaired intercostal muscle function postoperatively. However, it has been shown that 0.25% bupivacaine does not significantly impair respiratory function, and our preference is to use this option intraoperatively, and a continuous infusion of 0.1% bupivacaine and fentanyl 1-2µg/mL postoperatively. Accordingly, it is our standard practice to place a thoracic epidural catheter, usually in the T₄₋₆ interspace, and to establish a working block prior to the induction of anaesthesia. The current surgical practice at our institution is to perform a median sternotomy and bilateral lung resection.

Arterial and central venous pressure monitoring should be established prior to induction of anaesthesia, and great care given to the avoidance of possible lung injury during the placement of central venous lines. Whether or not pulmonary artery catheters should be used remains controversial, but most authors feel that they do not contribute substantially to patient management. Other authors have advocated the use of transoesophageal echocardiography, but there have been no studies demonstrating the value of such monitoring in terms of improved patient management decisions.

Intravenous induction of anaesthesia is routine, together with neuromuscular blockade, but consideration in the choice of neuromuscular blocking agent must include the fact that early extubation of these patients is mandatory, and that no residual neuromuscular blockade should be present at the end of the procedure. This probably precludes the use of pancuronium. The insertion of a left-sided double-lumen tube is mandatory and, although this is not universal practice, the need for optimal airway management is such that confirmation of correct placement of the endobronchial tube with a fibre-optic bronchoscope would appear to be appropriate. Excellent lung separation and endobronchial tube seal is essential during extended periods when lung resection and sealing of the cut surface is taking place.

Anaesthetic maintenance with propofol may avoid the theoretical risk of impaired hypoxic pulmonary vasoconstriction induced by inhaled anaesthetics, but the requirement for volatile anaesthesia in the presence of a good thoracic epidural block should be minimal and volatile anaesthesia, particularly with a short-acting agent, is widely utilised. There have been no reports of its use, but the addition of remifentanil, with its very short duration of action makes good physiological and pharmacological sense. Nitrous oxide should be avoided. Should hypotension occur, a differential diagnosis should be considered including pneumothorax and pulmonary tamponade with the impaired right ventricular filling (before the chest is opened), volume depletion and myocardial ischaemia. Unless there is obvious fluid loss, hypotension should generally be managed with vasopressors, and it is important to maintain the diastolic pressure where there is right ventricular compromise. Haematocrit should be maintained at > 30%.

Intraoperative positive pressure ventilation must be tailored to the patient’s requirements, and pressure-limited ventilation with a long expiratory time is generally advocated to minimise the risks of lung rupture and dynamic hyperinflation. The ventilatory strategy will almost certainly involve acceptance of some degree of hypercapnia, but this is usually well tolerated. Inspiratory pressures should be limited to 20 cmH₂O and the I:E ratio should be in the order of 1:4. Intraoperative hypoxia should be managed with increased inspired oxygen partial pressures, apnoic inflation of oxygen into the collapsed lung or, if necessary, occasional reinfation of the collapsed areas. Our practice is to preoxygenate for 10 minutes with 100% O₂ before lung isolation and resection. This facilitates identification of areas which are receiving little or no perfusion, since when the lung is isolated, these areas will remain hyperinflated (figure 1). Following lung resection, careful reinfation of the lung to allow the surgeon to detect any leaks is important, and good teamwork is mandatory.

Figure 1: Persistent hyperinflation of non-perfused areas of lung, after lung isolation.
Early tracheal extubation, which should always be performed in the operating room before the patient is transferred to the intensive care unit, should be the rule. Prior discussion with the patient concerning the technique of the extubation is helpful. It is important that the patient is warm, well perfused, pain free and fully restored to normal neuromuscular function. Consideration should be given to changing the double-lumen tube for a single lumen tube before attempting to establish spontaneous ventilation, as the airway resistance associated with the DLT is substantially higher. Consideration may also be given to extubating the patient deep and maintaining ventilation with a less irratant airway management device such as a laryngeal mask airway, as attempts should be made to minimise coughing during emergence from anaesthesia. To ensure the best possible respiratory function, the patient should be placed in a semi-sitting position during this phase. Restoration of adequate spontaneous ventilation may take some time, and patience is required.

Good postoperative pain management should be maintained into the postoperative period to allow the aggressive physiotherapy during recovery. The use of opiates in addition to bupivacaine in the epidural infusion will enhance analgesia, but may lead to CO2 retention. Addition of oral or intravenous COX-2 inhibitors to the epidural analgesia may provide improved pain control, particularly where pain due to chest drains is not fully controlled with the epidural. There is evidence that the COX-2 inhibitors do not increase the risk of bronchospasm. Pain from the upper end of median sternotomy incisions is frequently not controlled with the epidural alone and local infiltration of this part of the wound may be considered.

Recent advances
In view of the significant morbidity attached to the procedure, bronchoscopic lung volume reduction, involving the placement of deflating valves in the hyperinflated lung regions has recently been described. Although this technique is still in its infancy, good results have been reported in the small number of patients thus far evaluated. It is too early to say whether or not the results of this technique are comparable to those of surgical lung excision. Anaesthetic considerations for this procedure remains similar to those for the median sternotomy approach, although the postoperative analgesia problems are largely avoided.1,18,19

Conclusions
Although definitive, prospective, randomised controlled trials with large numbers are still awaited, current evidence seems to suggest that, in carefully selected patients, particularly those with non-homogenous, predominantly upper lobe pathology and limited exercise tolerance, lung volume reduction surgery in expert hands appears to have a definite place.

References