

Anaesthetic Considerations for Lung Volume Reduction Surgery—A Case Report

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Abstract

Introduction: This case describes some of the unique problems faced by the thoracic anaesthesiologists during anaesthesia for lung volume reduction surgery. **Clinical Picture:** The usual pulmonary function requirements for lobectomy are normally not met in these patients with severe emphysema. **Treatment:** Maintenance of the functional residual capacity of the lung and normocapnia during anaesthesia are not as important. Instead problems due to barotrauma and dynamic hyperinflation from positive pressure ventilation are. **Outcome:** Modification of ventilation strategy and providing an anaesthetic tailored towards early extubation is the cornerstone of the anaesthetic plan. **Conclusion:** A good understanding of the respiratory physiology in patients with severe emphysema is essential.

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Key words: Dynamic hyperinflation, Early extubation, Emphysema, One-lung ventilation, Permissive hypercapnia

Introduction

Only until a few years ago, the only surgical alternative for patients with severe emphysema was lung transplantation. However, with transplantation, there are problems of rejection, infection, immunosuppression and declining donor pools. Lung volume reduction surgery (LVRS) is an alternative surgery, which involves the removal of localised diseased areas of the enlarged lungs from patients with emphysema. It was proposed that when the remaining lung expands, it would exert radial traction on small airway, hence promoting bronchial patency during expiration.¹ Cooper et al² found that by returning the chest wall and diaphragm to a more normal shape and size, respiratory dynamics would improve and the work of breathing would reduce. There would also be reopening of the compressed alveoli and pulmonary vasculature thus leading to improved ventilation and perfusion matching. To date, hundreds of articles have been written on the subject of LVRS but only a few have reported their anaesthetic approach in a limited numbers of patients. We report a case of LVRS, highlighting the anaesthetic problems in managing such patients.

Case Report

A 65-year-old Chinese male (weight 56 kg) with chronic emphysema was referred for LVRS. He had been a chronic

smoker for more than 40 years (80 sticks a day) but had stopped smoking for the last 10 months. He required home oxygen therapy for 15 to 16 hours a day and managed to walk 50 to 100 m unassisted. His current medication included theophylline SR 125 mg twice a day, inhaled salbutamol and ipratropium bromide.

Clinically, his lungs were clear. The base-line arterial blood gases on enriched oxygen via nasal prongs at 3 L/min were as follows: pH, 7.360; PaCO₂, 65 mm Hg; PaO₂, 108 mm Hg; and SpO₂, 97%. The lung function test after 6 weeks of pulmonary rehabilitation showed some improvement (Table I). (Pulmonary rehabilitation programme refers to a multidisciplinary approach for individuals with end-stage lung diseases, which consists of optimising medical therapy, education, exercise, breathing retraining, nutrition and psychosocial support. The aim is to lessen airflow limitation, decrease respiratory symptoms and improve quality of life.)

High-resolution chest computed tomographic (CT) scan revealed a diffuse bilateral emphysema (heterogeneous pattern), especially in the region of the upper lobes. Cardiovascular assessment revealed blood pressure of 130/80. Electrocardiography showed sinus rhythm with a heart rate of 84 beats per minute. A 2D echocardiogram demonstrated a dilated right ventricle and right atrium. The left ventricular function was normal. The systolic pulmonary

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TABLE I: LUNG FUNCTION TEST RESULT BEFORE AND AFTER PULMONARY REHABILITATION

Parameter	Before rehabilitation	After rehabilitation
FEV ₁	0.4 L (16%)	0.53 L (21.6%)
FVC	1.56 L (38%)	1.64 L (40.5%)
TLC	7.99 L (146%)	7.17 L (131%)
RV	5.86 L (260%)	5.27 L (264 %)
D _L CO	(38%)	

FEV₁: forced expiratory volume in 1 second; FVC: forced vital capacity; TLC: total lung capacity; RV: residual volume; D_LCO: carbon monoxide diffusing capacity

artery pressure was raised and estimated to be 65 mm Hg. There was also evidence of mild aortic sclerosis and mild to moderate aortic regurgitation.

The patient was fasted overnight for the surgery. Premedication with sedatives was avoided. His usual medication of oral theophylline and steroid inhaler were administered in the morning of the operation. Salbutamol with ipratropium nebuliser was given on call to the operating theatre and he was transported with oxygen mask 4 L/min to the operating theatre. Before the induction of anaesthesia, an intravenous line with a 16G cannula was placed on the forearm. A radial artery line, a central venous line and a pulmonary artery catheter via the right internal jugular vein were also set under local anaesthetics. Finally, a thoracic epidural catheter at T4/5 was placed in a sitting position. Bupivacaine 0.5% was titrated into the epidural space until some paraesthesia was achieved around the level of the sternum (total 5 mL).

Anaesthesia was induced with propofol and intubation with a left-sided double-lumen endobronchial tube (Bronchocath size 37F) was facilitated with succinylcholine and remifentanyl infusion (up to 1 µg/kg). Anaesthesia was maintained using air and oxygen mixture with propofol infusion 10 mg/kg/h for 10 minutes followed by 8 mg/kg/h for the next 10 minutes and finally left at 6 mg/kg/h. Epidural analgesia was provided by an infusion bupivacaine 0.25% at 6 to 10 mL/h. A nerve stimulator was used to monitor neuromuscular blockade.

Apical lung resection was performed via a median sternotomy approach and about 20% of the lung from the apices was removed using staples buttressed with bovine pericardium. One-lung ventilation was performed during the resections and the lung was slowly re-expanded at the end of the resection to check for air leaks. We used the Servo 900C (Siemens) ventilator for the case. During one-lung ventilation, our ventilation strategy included the use of pressure controlled ventilation with inspiratory pressure of 30 cmH₂O and no PEEP. Respiratory rate of 10 breaths a minute and I:E ratio of 1:4 was set. The measured expired tidal volumes were between 300 to 400 mL. FiO₂ used was

titrated to keep the SpO₂ >90%. Cardiac output was monitored using the thermodilution method. Dopamine infusion of 2 to 7 µg/kg/min was infused during the case when necessary. The total amount of fluid infused throughout the case was 2.0 L and urine output was 1.3 L. The blood pressures throughout the surgery are shown in Figure 1.

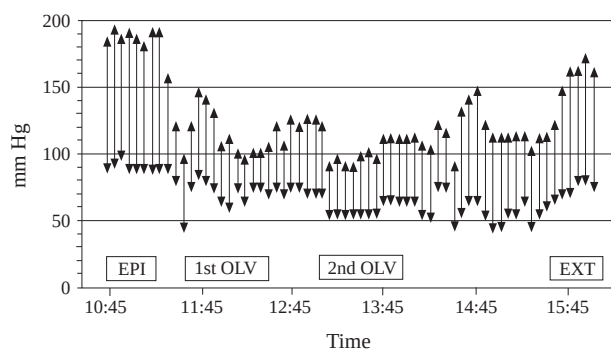


Fig. 1. Blood pressure changes. The mean cardiac output after intubation during the first, second one-lung ventilation and at the end of surgery was 5.2, 5.4, 5.4 and 5.0 L/min, respectively. Pulmonary artery pressures during those events were 55/33, 49/31, 38/21 and 40/24 mm Hg.

EPI: placement of epidural catheter

1st OLV: period of one-lung ventilation during the operation of the first lung

2nd OLV: period of one-lung ventilation during the operation of the second lung

EXT: weaning patient for extubation

At the end of the procedure, a chest tube attached to underwater seal was inserted into each thorax. The propofol infusion was stopped and the neuromuscular blockade reversed. Ventilation was set to a pressure support mode and the patient was weaned slowly off the ventilator and extubated when he was alert and judged to have regained adequate muscle strength. The patient was transferred to the intensive care unit for monitoring. The total duration of the anaesthetic time was about 5 hours.

The blood gasses during one-lung ventilation for resection of the first (OLV1) and second (OLV2) lung and post extubation period of 24 hours are shown in Table II.

Postoperatively, analgesia was provided by an infusion of bupivacaine 0.125% with fentanyl 2 µg/mL via the epidural catheter. The blood gasses improved gradually and the air leaks in both chest subsided on the 4th postoperative day and the right chest tube was removed and he was transferred to a high dependency ward.

Discussion

Some of the adverse effects on respiratory function during general anaesthesia include a reduction of functional residual capacity, decreased compliance, increased propensity of upper and lower airway obstruction with increased airway resistance and increased ventilation-

TABLE II: ARTERIAL BLOOD GAS RESULTS

	OLV1	OLV2	Post extubation (h)			
			0	2	7	24
pH	7.23	7.24	7.24	7.33	7.38	7.43
PaCO ₂ (mm Hg)	95	86	90	68	62	50
PaO ₂ (mm Hg)	82	71	106	94	73	75
SpO ₂ (%)	94	92	97	97	94	95
HCO ₃ (mmol/dL)	39	36	31	31	35	32
BE	12	10	7	7	9	7
FiO ₂	1.0	1.0	0.5	0.5	0.4	0.4

BE: base excess; FiO₂: inhaled oxygen fraction; OLV1: period of one lung ventilation during the operation of the first lung; OLV2: period of one lung ventilation during the operation of the second lung; PaCO₂: partial pressure of arterial carbon dioxide; PaO₂: partial pressure of arterial oxygen; pH: -log[H⁺]; SpO₂: arterial oxygen saturation

perfusion (V/Q) mismatch.³ Patients with chronic obstructive lung diseases have increased risk of bronchospasm, pulmonary complications and mortality.⁴ Indeed, most of the patients presenting for LVRS have poor pulmonary function test results and would not fulfill the criteria for lobectomy. However, assessment of postresection respiratory mechanics, cardiopulmonary reserve and lung parenchyma function, which are important criteria for lung resection,⁵ cannot be applied. This is because these parameters are expected to improve after LVRS.² Various centres have different patient selection criteria for performing LVRS.⁶⁻⁸ However, there is general agreement that the most important criteria would be the presence of severe airway obstruction with marked air trapping as a result of the emphysema. There should also be “target areas” in the lungs with heterogeneous pattern of emphysema on chest CT scan. Ideally, a lung ventilation-perfusion scan could have been performed for our patient to help locate these areas.

Exclusion criteria have similarly been published,^{6,8} but real data to support all these criteria are lacking. Some workers consider the presence of advance age, hypercapnia and pulmonary hypertension (all of which our patient had) as relative⁶ or no contraindication⁷ for LVRS. However, co-morbid conditions, which put patients at high risk for perioperative morbidity and mortality, should be taken into consideration when selecting such patients for LVRS. Emphysematous patients with long history of smoking are also at higher cardiac risk. There is an increased risk of perioperative myocardial infarction after LVRS in some of these patients,^{9,10} but they may be difficult to identify. Exercise tests are difficult to perform as most patients are unable to exercise to heart rate limits because of pulmonary limitations. A preoperative cardiovascular assessment together with an electrocardiogram is essential, but the cost effectiveness or other diagnostic tests are not known. We

chose to perform a 2D echocardiogram for our patient to further assess ventricular function as it was relatively non-invasive and readily available in our institution. As anaesthesiologists, we are involved in selecting and optimising patients who would benefit most from LVRS. However, the more conclusive patient selection criteria may have to depend on the outcome of a currently ongoing multicentric prospective trial.¹¹ Hopefully, when it is completed, we will then know whether the benefits of LVRS outweigh the associated risk and cost and issues about patient selection and safety can be answered.

During anaesthesia, the haemodynamic monitors that we used included a central venous catheter via the right internal jugular vein. However, this was used mainly for drug administrations as the actual value does not reflect ventricular filling due to the increased thoracic pressures during intermittent positive pressure ventilation in this patient. We decided to use a pulmonary artery catheter in this case because we felt that there was increased risk of cardiovascular complications as the patient had evidence of pulmonary hypertension and right ventricular dilatation preoperatively. During the placement of the catheters, we used a small gauge needle to locate the internal jugular vein. We also avoided trying to locate it in the lower position of the neck as this may lead to puncturing the apical lung bullae during placement of the catheter. The radial arterial line was used for frequent arterial blood sampling, especially during the periods of one-lung ventilation as the capnography would not be accurate to assess PaCO₂. Some workers have used a continuous method of blood gas analysis from the arterial line.⁹ Our haemodynamic parameters throughout the anaesthesia were fairly stable although we relied on some inotropic support (dopamine up to 7 µg/kg/min) at some stages. The pulmonary artery pressures and cardiac outputs were also within acceptable limits during the anaesthesia and one-lung ventilation. This was in agreement with the findings by other workers.^{9,12}

A thoracic epidural anaesthesia (TEA) with local anaesthetics is strongly recommended by many authors.^{9,13,14} Other workers have used epidural opioid^{15,16} or intercostal blocks¹² for thoracoscopic surgery. Triantafillou et al¹³ recommended placement of TEA catheter under fluoroscopy at T3/4 level. However, we believe that if catheter placement is done properly (perhaps in sitting position if the lateral position cannot be tolerated for prolonged periods) and paraesthesia established before induction of anaesthesia, fluoroscopy would not be necessary. Given at the mid-thoracic level, it will provide good pain relief both during the surgery and postoperatively.

Traditionally, patients with severe emphysema are intubated with sedation and with the use of local anaesthesia

to avoid intermittent positive pressure ventilation.¹⁵ The use of spontaneous respiration under anaesthesia has also been suggested as an option.¹⁷ However, these patients have increased risk of bronchospasm⁴ and hypoventilation¹⁸ under anaesthesia. A controlled intubation with a short acting neuromuscular blocker may be preferred. We used propofol for the induction and maintenance of anaesthesia. Propofol causes less pulmonary shunting compared with inhalational agents during one-lung ventilation.¹⁹ It would also be easier to control the delivery of anaesthetic in the event of severe air leak. We also avoided the use of nitrous oxide as this would fill the air spaces and may increase pulmonary vascular resistance by increasing intraalveolar pressure on pulmonary capillary blood flow.²⁰ Lung isolation was essential for the surgery. We chose a left-sided double-lumen endobronchial tube as it had a larger margin of safety.²¹ The size of the double-lumen tube used was based on chest CT scan measurements of the diameter of the left main bronchus²² and the double-lumen tube was positioned with the help of a fiberoptic bronchoscope to ensure proper placement.

Patients with chronic emphysema have increased risk of gas trapping during intermittent positive pressure ventilation.²³ This introduces the problems of barotrauma, decreased venous return, increased pulmonary vascular resistance and right ventricular output impedance. The ventilation strategy we used was a low I:E ratio (1:4 or 1:5) to allow more complete expiration. This would reduce breath-stacking and air trapping, which can cause haemodynamic instability and risk a bullae rupture and tension pneumothorax. During one-lung ventilation, the inspiratory pressure was limited to 30 cm H₂O to avoid barotrauma. No extrinsic PEEP was added as intrinsic PEEP was known to occur during one-lung ventilation.²⁴ Ideally, a flow detector in expiratory limb to assess intrinsic PEEP may optimise the ventilation strategy. Various types of positive pressure ventilation have been used in the past for patients with emphysema undergoing surgery.¹⁷ We used a critical care type ventilator (Servo 900C, Siemens) which allowed us to use pressure-controlled ventilation with high inspiratory flow at lower peak pressures to overcome air leaks especially when ventilating the operated lung. Although PaCO₂ increased during anaesthesia, some degree of permissive hypercapnia is known to be tolerated.²⁵ The blood gasses showed no evidence of severe acidemia during one-lung ventilation despite PaCO₂ of 95 mm Hg. There was also no problem with oxygenation as atelectasis was not a serious concern due to the presence of air trapping. Air leak after stapling will need to be assessed. We re-expanded the collapsed lung slowly after surgery to prevent tearing of staple lines. Zollinger et al⁹ advised that if more than 20% to 30% of the tidal volume is lost by air leak, surgery to the other side should be refrained.

Postoperatively, early extubation will reduce risk of air leak and bronchopulmonary fistula and nosocomial infections. However, patients with chronic obstructive lung diseases are generally more sensitive to respiratory depression effects of inhalational agents, barbiturates and benzodiazepams.^{18,26} Extubating such patients in the operating theatre may require quite a long time⁹. In Cooper et al,² Triantafillou et al¹³ and Buettner et al's¹⁴ patients who had median sternotomy, 95% to 99% of patients were successfully extubated at the end of the procedure. Liu et al,¹⁶ on the other hand, chose to extubate his patients who had median sternotomy in the intensive care. They had used lumbar epidural diamorphine infusion for intra and post-operative analgesia. Two of their patients required naloxone to reverse the respiratory depression after extubation. A thoracoscopic LVRS approach does not necessarily result in earlier extubation for the patients. Zollinger et al⁹ extubated almost all their patients after thoracoscopic LVRS and had a reintubation rate of 8%. We decided to "wean" our patient on the operating theatre at the end of the surgery as the air leak was minimal and he did not have problems with inadequate analgesia, bronchospasm, excessive secretions, hypothermia or acidosis. We felt that there was an increased risk of excessive coughing and air leak if there was prolonged ventilation in the intensive care unit. Triantafillou et al¹³ recommended "deep extubation" to avoid coughing. We prefer to use the pressure support mode of ventilation with gradual reduction of the pressure support before extubating the patient. Immediately after extubation, the PaCO₂ can be increased up to 90 mm Hg.^{9,13,14} In our patient, the hypercarbia gradually improved over the following 24 hours. No adverse cardiovascular or neurological event occurred during this period.

One of the most common postoperative complications reported by Cooper et al² was air leak. Barker et al,²⁷ who performed thoracoscopic laser ablation for bullous emphysema, required postoperative ventilation for an average of 5 days with up to 50% of the patients having air leak. A chest X-ray should be done routinely to assess the problem. If necessary, chest tubes may be inserted but should not be placed on suction if the leak is small. However, some major air leaks may require re-exploration. Respiratory failure can also be due to inadequate pain control, bronchospasm, excessive secretions, residual neuromuscular paralysis, hypothermia, hypercarbia and acidosis. In Buettner et al's series,¹⁴ 15% of the patients had to be reintubated due to respiratory failure from secretions, air leak, inadequate analgesia or respiratory depression from opioid. If patients need to be reintubated, a single-lumen endotracheal tube should be used. The same ventilation strategy should be used. The use of bronchodilators, mucolytics and respiratory physiotherapy should be encouraged. It may also be important not to overload the

patient with fluid which can lead to pulmonary congestion. Postoperative analgesia is important to optimise respiratory function. During the postoperative period, we continued the epidural infusion with a combination of bupivacaine 0.125% and fentanyl 2 µg/mL. We believe that low-dose local anaesthetic may have less effect on the respiratory muscles strength and low-dose opioids have little central nervous system effects on the respiratory function and this will allow early mobilisation and physiotherapy. Other workers have used patient-controlled analgesia with intravenous morphine.¹³ However, one should avoid excessive use of opioids, which can easily result in respiratory depression.

In summary, the anaesthetic care patients undergoing LVRS requires a good understanding of the respiratory physiology. Unlike pulmonary resection for bronchogenic tumours where there is a lag period of decrease in lung, chest wall and diaphragm compliance due to the trauma of surgery, LVRS results in improved pulmonary function. The ventilation strategy may allow permissive hypercapnia during one-lung ventilation. One should also minimise auto-PEEP and recognise the problems associated with it. Good analgesia is essential and anaesthesia should be tailored towards early extubation. Finally, close post-operative monitoring is essential for all patients after LVRS.

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