Beatrice Ratzenhofer-Komenda MD,*
Anton Offner MD,*
Fritz Kaltenböck MD,*
Alfred Maier MD,†
Hans Pinter MD,†
Gerhard Prause MD,*
Freyja M. Smolle-Jüttner MD†

Differential lung ventilation and emergency hyperbaric oxygenation for repair of a tracheal tear

Purpose: To report the anaesthetic management of a case of tracheal rupture, using different types of ventilation and additional hyperbaric oxygenation (HBO).

Clinical features: An 8 cm postintubation tracheal tear was repaired in a 66-yr-old woman with acute myocardial reinfarction, mediastinal and subcutaneous emphysema, cardiac failure and unrecognized lymphoma. Intraoperative monitoring included dual oximetry: arterial (SaO_2) and mixed venous saturations (SvO_2). Maintenance of free surgical access and a series of life-threatening events like dislocation of the jet catheter required many ventilation modes. An episode of supraventricular tachycardia was interrupted by cardioversion. Differential lung ventilation with a combination of conventional and high-frequency jet ventilation (HFJV) modes preserved oxygenation (PO_2 139.2 mmHg, PCO_2 42.4 mmHg, FiO_2 1.0) until acute tube obstruction and decrease of saturation values (SaO_2 58%, SvO_2 45%) required emergency HBO: immediate cardiac and respiratory stabilization was provided by double-lung HFJV and apneic oxygenation under hyperbaric conditions at 2.5 atmospheres absolute for 35 min (SaO_2 100%).

The patient recovered from surgery but died of non-Hodgkin lymphoma.

Conclusion: The combination of different ventilation modes including HFJV and the additional use of HBO resulted in sufficient oxygenation during tracheal repair.

Objectif : Présenter un cas de rupture trachéale où on a utilisé, pendant l'anesthésie, différents types de ventilation et de l'oxygénation hyperbare (OHB) additionnelle.

Éléments cliniques : Une lacération trachéale postintubation de 8 cm a été réparée chez une femme de 66 ans qui présentait une récurrence d'infarctus myocardique aigu, un emphysème médiastinal et sous-cutané, une insuffisance cardiaque et un lymphome méconnu. La surveillance peropératoire comprenait une double oxymétrie : du sang artériel (SaO_2) et du sang veineux mêlé (SvO_2). Le maintien d'un libre accès chirurgical et une série d'événements graves comme le déplacement du cathéter de ventilation en jet ont nécessité l'utilisation de nombreux modes de ventilation. Un épisode de tachycardie supraventriculaire a été interrompu par une cardioversion. Une ventilation pulmonaire différentielle combinée à une ventilation classique et en jet à haute fréquence (VJHF) ont préservé l'oxygénation (PO_2 139,2 mmHg, PCO_2 42,4 mmHg, FiO_2 1,0) jusqu'à ce qu'une obstruction du tube et une baisse de la saturation soudaines (SaO_2 58 %, SvO_2 45 %) exigent une OHB d'urgence : une stabilisation cardiaque et respiratoire immédiate ont suivi grâce à la VJHF bilatérale et à l'oxygénation apnéique en conditions hyperbares à 2,5 atmosphères absolues pendant 35 min (SaO_2 100 %). La patiente s'est bien remise de l'opération mais est décédée d'un lymphome non hodgkinien.

Conclusion : La combinaison de différents modes ventilatoires incluant la VJHF et l'emploi complémentaire d'OHB ont fourni une oxygénation suffisante pendant la réparation trachéale.

From the Departments of Anesthesiology and Critical Care Medicine,* and Thoracic and Hyperbaric Surgery,† University Medical School of Graz, Graz, Austria.

Address correspondence to: Beatrice Ratzenhofer-Komenda MD, Department of Anesthesiology and Critical Care Medicine, University Hospital, LKH - Universitätskliniken Graz, Auenbrugger Platz 29, A-8036 Graz, Austria. Phone: +43-316-385-3359; Fax: +43-316-385-3847; E-mail: beatrice.ratzenhofer@kfunigraz.ac.at

Accepted for publication November 14, 1999

ANESTHESIA in patients with injury of the major conducting airways requires special ventilation techniques to avoid interference with the surgical field.¹ We describe the intraoperative ventilation management in an unstable patient with tracheal rupture.

Clinical features

A 66-yr-old woman (160 cm, 59 kg) developed nausea and retrosternal pain at home and subsequently lost consciousness. According to her son she continued to breathe and maintained a pulse. The emergency team obtained a blood pressure of 90/60 mmHg; the ECG in the ambulance showed tachycardia (170 bpm) and elevated ST segments in leads II, III and aVF. Initially, 2.5 mg midazolam and 20 mmol potassium were administered *iv* and oxygen was given via a face mask. The patient regained consciousness. She had a history of non-insulin dependent diabetes mellitus, arterial hypertension, and posterior wall myocardial infarction three months previously.

On admission to the cardiac care unit, the ECG showed a sinus rhythm of 68 bpm, left axis deviation, elevated ST segments and a negative T, in leads II, III, aVF and V6 as well as descending ST segments in V4 and V5. A diagnosis of recurrent myocardial infarction was made. After a short period of hemodynamic stabilization the patient developed ventricular tachycardia of 193 bpm and dyspnea and the trachea was easily intubated under emergency conditions. Three cardioversion attempts failed and subsequent cardiopulmonary resuscitation was successful. To prevent left ventricular failure she was sedated and ventilation was maintained with synchronized intermittent ventilation. The initial ventilator setting that maintained peripheral arterial saturation between 97% and 100% was: FiO_2 0.3, respiration rate 12 bpm, V_T 0.65 L, I:E ratio 1:2, positive end-expiratory pressure (PEEP) 6.90 kPa. She was anticoagulated with a continuous infusion of heparin (1000 IU·hr⁻¹). Heart rate was controlled by continuous infusion of 0.013 mg·kg⁻¹·min⁻¹ lidocaine. Approximately six hours after intubation the patient developed subcutaneous emphysema of the neck and chest. The subcutaneous emphysema was thought to be the result of a minor tracheal injury or from a small alveolar leak due to low-grade emphysema. Over the next few hours the patient's hemodynamic condition deteriorated. The subcutaneous emphysema expanded to the lower abdomen. Computed tomography of the neck and thorax showed a subglottic lesion of the anterior tracheal wall with emphysema of the adjacent tissue and mediastinum. The heart was dilated and a 1 cm peri-

cardial effusion was visible. Both lungs contained multiple emphysematous and atelectatic areas. There was a small pleural effusion on the right side. The air-filled areas of the subcutaneous emphysema spread into the retroperitoneal space. Ventilation was continued with assisted spontaneous breathing at an inspiratory pressure of 14.71 kPa and a PEEP of 4.91 kPa at FiO_2 of 0.3. Arterial blood gas analysis showed pH 7.40, PaCO_2 42.1 mmHg, PaO_2 66.2 mmHg, base excess 1.0 mEq·L⁻¹, serum bicarbonate 25.8 mEq·L⁻¹, SaO_2 95.1%. Over the next few hours the trachea was extubated and the patient was given oxygen (4 L·min⁻¹) via a face mask providing arterial saturation values between 96 and 98%.

Over the next 48 hr the patient became increasingly hemodynamically unstable due to mediastinal compression: three further episodes of ventricular tachycardia occurred. One episode stopped without intervention. With the next episode, the trachea was reintubated without difficulty to give anesthesia and ventilation for the cardioversion procedure. Cardioversion was successfully performed twice and a continuous infusion of 0.01 mg·kg⁻¹·hr⁻¹ amiodarone was applied in addition to the current medication.

Physical examination showed an extensive subcutaneous emphysema. Bronchoscopy showed an 8-cm tear in the membranous part of the trachea extending from the first tracheal cartilage to the beginning of the right mainstem bronchus. Blood pressure was 115/52 mmHg with a heart rate of 78 bpm. Hemoglobin and electrolyte concentrations were normal. Serum tests were consistent with myocardial infarction (creatinine kinase (CK) 90 U·L⁻¹; CK-MB 22 U·L⁻¹; Lactate dehydrogenase 342 U·L⁻¹, ASAT 36 U·L⁻¹; Troponin I 3 ng·mL⁻¹ (upper limit: 2 ng·mL⁻¹). Transesophageal echocardiography showed left-ventricular dilatation, aneurysm of the anterior wall and the apex, akinesia of the posterior wall and hypokinesia of the rest of the myocardium. The ejection fraction was 25%. The right atrium and the right ventricle were normal in diameter. Arterial blood gas analysis showed pH 7.30, PaCO_2 60.1 mmHg, PaO_2 56.9 mmHg, base excess 1.7 mEq·L⁻¹, serum bicarbonate 28.7 mEq·L⁻¹, SaO_2 85.9%. The blood sample was drawn at the ventilator setting as follows: synchronized intermittent ventilation mode (SIMV): FiO_2 0.7, respiration rate 12 bpm, tidal volume (V_T) 0.65 L; minute volume 7.6 L, I:E ratio 1:1.5, positive end-expiratory pressure (PEEP) 6.90 kPa.

Emergency surgery was planned for the hyperbaric chamber of the Department of Thoracic and Hyperbaric Surgery, which is equipped and used routinely as an operating room for thoracic surgery.²

Anesthesia and ventilation

Induction and maintenance of anesthesia

Heart rhythm and hemodynamics were supported by continuous infusion of $0.016 \text{ mg}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ lidocaine *iv*, $6 \text{ }\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ dobutamine and $3 \text{ }\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ dopamine. Anesthesia was induced with $0.04 \text{ mg}\cdot\text{kg}^{-1}$ midazolam, $0.2 \text{ mg}\cdot\text{kg}^{-1}$ etomidate and $1.27 \text{ }\mu\text{g}\cdot\text{kg}^{-1}$ fentanyl. Total intravenous anesthesia was maintained with continuous infusion of $0.03 \text{ mg}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ midazolam and $4 \text{ }\mu\text{g}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ fentanyl. Muscle relaxation was obtained with $0.08 \text{ mg}\cdot\text{kg}^{-1}\cdot\text{hr}^{-1}$ vecuronium bromide. After induction, a pulmonary artery catheter was inserted for continuous monitoring of mixed venous saturation (SvO_2). Dual oximetry values (SaO_2 and SvO_2) were recorded (Explorer, Edwards Critical Care, Baxter Immuno Corp., Vienna, Austria).

Ventilation modes

STEP 1: DEPENDENT LUNG CONTROLLED MECHANICAL VENTILATION (CMV)

Because airway leakage impaired adequate ventilation the original 7.5-mm tube (inner diameter) was replaced with a reinforced 7.0-mm tracheal tube to pass the leak. The unaffected left main stem bronchus was intubated bronchoscopically and one-lung ventilation was begun

(Figure 1a) with controlled mechanical ventilation (CMV), tidal volume V_T 0.6 L, respiration rate 14 bpm, PEEP 2.90 kPa, FiO_2 0.7-1.0. Oxygenation was adequate with the patient supine but dual oximetry values decreased when she was turned to the left lateral decubitus position to perform a right lateral thoracotomy (Figure 2).

STEP 2: DEPENDENT AND NON-DEPENDENT LUNG CMV

To reestablish ventilatory support the surgeon positioned an armoured 6.0-mm endobronchial tube in the right main stem bronchus distal to the airway leak (Figure 1b). Additional non-dependent lung ventilation was performed through a sterile ventilation hose that crossed the surgical field and was connected to a second ventilator. Laceration of the tracheal tissue was visible at that time. Identification and dissection of the anatomical structures was difficult. The lymph nodes were enlarged and biopsies were taken for histology. Differential lung ventilation resulted in an improvement of arterial and mixed venous oxygen saturations. Differential lung ventilation settings were as follows: dependent lung: CMV: V_T 0.6 L, respiration rate 14 bpm, PEEP 3.90 kPa, FiO_2 1.0; non-dependent lung:

Tube positions during tracheal reconstruction

FIGURE 1 Tube positions during tracheal reconstruction. The arrows indicate the direction of the gas stream. CMV: controlled mechanical ventilation. HFJV high-frequency jet ventilation. **1a:** Step 1: dependent lung CMV. **1b:** Step 2: dependent and non-dependent lung CMV. **1c:** Step 3: dependent lung CMV and non-dependent lung HFJV over the surgical field. **1d:** Step 4: emergency dependent lung CMV over the surgical field and precarinal HFJV through the endotracheal tube. **1e:** Step 5: double-lung hyperbaric HFJV and apneic hyperbaric oxygenation.

FIGURE 2 Intraoperative dual oximetry. Saturation profiles according to the steps of ventilation. The most important ventilation modes used during the several steps are indicated in the graph. See text for detailed description. The arrows indicate the changes of saturation related to events during surgery. SaO_2 : arterial saturation. SvO_2 : mixed venous saturation. *Lat. decub. position*: lateral decubitus position. *CMV*: controlled mechanical ventilation. *HFJV*: high-frequency jet ventilation. *HBO*: hyperbaric oxygenation. *AD*: apneic oxygenation. *PCV*: pressure-controlled ventilation.

CMV: V_T 0.5 L, respiration rate 12 bpm, PEEP 3.90 kPa, FiO_2 1.0 to 0.7. The non-dependent lung tube interfered with tracheal reconstruction and had to be removed.

STEP 3: DEPENDENT-LUNG CMV AND NON-DEPENDENT LUNG HIGH-FREQUENCY JET VENTILATION (HFJV) THROUGH A JET CATHETER VIA THE SURGICAL FIELD

The non-dependent lung, single-lumen endobronchial tube was replaced with a 14-French suction catheter via the surgical field and HFJV was begun through this catheter (Figure 1c) at the following settings: dependent lung: CMV: V_T 0.6 L, respiration rate 14 bpm, PEEP 3.90 kPa, FiO_2 1.0; non-dependent lung: HFJV: 150 bpm, outlet pressure 600.05 mmHg, FiO_2 0.7. The pressure within the bronchial system distal to the tip of the catheter did not exceed 110.26 mmHg; alarm limits interrupting the jet stream delivery were preset at 147.80 mmHg (VDR – Bronchotron-1, Percussionaire Corp., Idaho, U.S.A.).

Oxygenation remained adequate until the jet catheter escaped from the forceps. Repositioning the catheter was unsuccessful and oxygen saturation decreased rapidly. Overall, the dependent lung endobronchial tube laid over the surgical field made surgical access difficult and impeded the reconstruction.

STEP 4: EMERGENCY DEPENDENT-LUNG CMV OVER THE SURGICAL FIELD AND PRECARINAL HFJV THROUGH THE ENDOTRACHEAL TUBE

Because of hypoxia the left main stem bronchus was intubated urgently with a reinforced 6.0-Charriere endotracheal tube. A double-lumen nasogastric tube was inserted into the tracheal tube to provide HFJV through the larger port and optionally a continuous flow of oxygen through the smaller port (Figure 1d). The differential lung ventilation settings were as follows: Emergency dependent lung CMV: V_T 0.6 L, respiration rate 14 bpm, PEEP 3.90 kPa, FiO_2 1.0; non-dependent lung: precarinal HFJV 150 bpm, outlet pressure 1.0 bar, FiO_2 1.0. This provided sufficient

oxygenation but precluded surgical reconstruction. Blood gas analysis showed pH 7.38, PCO_2 41.4 mmHg, PO_2 139.2 mmHg, serum bicarbonate 24.2 mEq·L⁻¹.

Before the positions of the tubes could be changed to obtain better surgical access, the patient developed a sudden episode of supraventricular tachycardia that required cardioversion. Shortly after the hemodynamic status and heart rhythm were restored, the left endobronchial tube became acutely obstructed due to herniation of the cuff. Attempts to deflate the cuff were unsuccessful. With low mixed venous and arterial saturation values, it was decided to continue surgery under hyperbaric conditions (Figure 2).

STEP 5: DOUBLE-LUNG HYPERBARIC HFJV AND APNEIC HYPERBARIC OXYGENATION (HBO)

Compression was begun at a rate of 0.4 atmospheres per minute. In the interest of time, paracentesis for pressure equilibration was not performed.

Only high-frequency tidal volumes were applied via the jet catheter inserted through the endotracheal tube (Figure 1e). The high-frequency ventilation mode remained unchanged except for the reduction of outlet pressure in the beginning of the HBO session.

Ambient pressure was kept at 2.5 ATA (atmospheres absolute). Arterial saturation returned to normal so that additional CMV support via the endotracheal tube was unnecessary. Because dissection and adaptation of the fragile tissue required a calm surgical field, HFJV was replaced by hyperbaric apneic oxygenation for five minutes without a decrease in SaO_2 . Elevated ambient pressure was maintained at 2.5 ATA for 35 min (isopression phase) until reconstruction of the trachea was completed. Hemodynamic variables and peripheral arterial saturation stabilized under hyperbaric conditions and the heart rate remained rhythmic. Mixed venous saturation values were not displayed in the hyperbaric environment.

When the trachea was closed, HFJV was stopped and CMV installed with monitoring of the airway pressures (Hyperlog, Dräger Corp., Lübeck, Germany). Decompression was started at a rate of 0.3 atmospheres per minute with a three-minute safety stop at 0.3 ATA. The hyperbaric protocol followed the guidelines of the U.S. Navy diving manual.³

After decompression, the respiratory regimen was guided with pressure-controlled ventilation (PCV) to avoid high airway pressures. The tube was positioned fibreoptically next to the carina to bypass most of the sutures. The chest was closed under normobaric conditions. Hemodynamic and ventilation parameters remained stable until the end of surgery (Figure 2).

Postoperative course

The trachea was extubated on the fourth postoperative day in a stable respiratory and hemodynamic state. However, over the next eight hours the patient developed dyspnea and the trachea was reintubated. Bronchoscopy showed external compression of the trachea, most likely by the enlarged lymph nodes. Histology of tissue samples obtained at surgery revealed a non-Hodgkin-lymphoma. Insertion of an endotracheal stent was impeded by the fragile tissue prone to local bleeding. The patient recovered from surgery and from myocardial reinfarction with her ejection fraction increasing from 20 to 50 percent but the trachea remained intubated with CPAP. She died of her underlying malignancy three months later after three cycles of chemotherapy.

Discussion

Rupture of the tracheobronchial tree can be due to overdistension of a tube cuff,⁴ elevated intrathoracic pressure (chest trauma, closed chest compression during cardiopulmonary resuscitation, coughing), traumatic intubation,^{5–8} or inherent weakness of the membranous trachea.⁹ The interval between rupture and the occurrence of symptoms ranges between 20 min and five days.¹⁰ In our patient, rupture was caused by intubation of a trachea weakened by extrinsic lymphadenopathy and possibly by external chest compression at cardiopulmonary resuscitation. Symptoms developed after approximately six hours.

Our patient required emergency surgery to close the trachea despite a recent myocardial infarction. During the operation, differential lung ventilation by conventional and high-frequency ventilation (HFV) modes^{11,12} were successful initially. When an episode of supraventricular tachycardia suggesting cardiac compromise was followed by obstruction of the endobronchial tube, respiratory and cardiac function had to be restored immediately. The life-threatening situation did not permit transporting the patient to the OR where the device for extracorporeal membrane oxygenation was installed, nor was a portable device available. Emergency hyperbaric oxygenation was begun as a last-ditch effort to increase oxygen delivery. A higher amount of oxygen dissolved in the plasma can provide sufficient tissue oxygenation during periods of low oxygen supply and prolong tolerance to hypoxia.² There are reports that HBO may be useful as an adjunct in the management of cardiogenic shock.¹³ Outcome evaluations of patients undergoing HBO therapy after acute myocardial infarction suggest a beneficial effect of hyperbaric oxygen.¹⁴ HBO may have benefitted our patient.

High-frequency ventilation is often used with major conducting airway surgery but has not been applied under hyperbaric conditions.¹⁵ High-frequency ventilation during open airway surgery is advantageous over conventional ventilation modes as it provides an unobstructed and immobile surgical field and adequate gas exchange. Magnusson and coworkers¹⁶ report on modest hypercarbia without hypoxaemia in a series of 10 pediatric and seven adult patients undergoing tracheal resection. The major drawback is the risk of barotrauma which is minimized by monitoring airway pressure and qualified surgical assistance to avoid airway obstruction by debris or dislocation of the jet catheter. The pressure valves in the HFV device used in our patient are vented to the atmosphere providing an equilibrium with the ambient pressure which may explain why the device did not fail when it was accidentally exposed to the hyperbaric environment.

Care of the critically ill and surgery in the hyperbaric environment require special considerations.¹⁷ The cuff of the endotracheal tube is filled with water in order to save its seal during compression and to avoid distension during decompression. Glass bottles are replaced by plastic infusion bags to avoid *in situ* infusion of bubbles generated during decompression within the liquid and because of the danger of fracture. Myringotomy is performed in the intubated patient unable to achieve pressure equilibration actively. Myringotomy was impeded in our patient by severe impending hypoxemia. Elevation of pressure did not cause damage to the eardrum, possibly because compression was performed at a moderate rate. Prevention of fire hazard in the hyperbaric environment is crucial. A fire extinguishing system operational from inside and outside of the chamber must be provided.

All equipment and circuits within the hyperbaric facility must be pressure tested, spark proof and explosion proof. Flammable materials must be removed from the patient and no flammable or volatile liquid is allowed inside the chamber. Fire is less of a hazard in the multiplace chamber when oxygen concentration is kept < 23%. At a volume of 75,500 L, the hyperbaric chamber where our patient was treated was flooded at a rate of 100000 L·min⁻¹. Breathing air during the isopression phase, the medical personnel is potentially at risk for developing decompression sickness. Therefore, pure oxygen is inhaled to wash out nitrogen and reduce bubble formation during the decompression period.

The staff working in hyperbaric facilities has to be well trained and familiar with the pathophysiology of hyperbarism for patient and personal safety.

References

- 1 Ratzenhofer-Komenda B, Prause G, Offner A, Smolle-Jüttner FM. Intraoperative application of high-frequency ventilation in thoracic surgery. *Acta Anaesthesiol Scand* 1996; 40(Suppl 109): 149–53.
- 2 Friehs G, Klepp G, Gollmann K, Rader W, Stolze A. Hyperbare oxygenation in der klinik, erste erfahrungen. *Zbl Chir* 1975; 100: 321–31.
- 3 US Navy Diving Manual (1995), Navsea 0094-LP-001-9010. Navy Department Washington, D.C. 20362. Best Publishing Co., 23005/2 South Avalon Boulevard, Carson, California 90745, U.S.A.
- 4 Cooper JD, Grillo HC Experimental production and prevention of injury due to cuffed tracheal tubes. *Surg Gynecol Obstet* 1969; 129: 1235–41.
- 5 Smith BAC, Hopkinson RB. Tracheal rupture during anaesthesia. *Anaesthesia* 1984; 39: 894–8.
- 6 Schwartz DE, Lieberman JA, Cohen NH Women are at greater risk than men for malpositioning of the endotracheal tube after emergent intubation. *Crit Care Med* 1994; 22: 1127–31.
- 7 Beim T, Lenhard F-P, Berger H, et al. Tracheal rupture during difficult intubation. (German) *Anaesthesist* 1991; 40: 456–7.
- 8 Marty-Ané C-H, Picard E, Jonquet O, Mary H. Membranous tracheal rupture after endotracheal intubation. *Ann Thorac Surg* 1995; 60: 1367–71.
- 9 Ratzenhofer-Komenda B, Prause G, Offner A, Kaloud H, Pinter H, List WF. Tracheal disruption and pneumothorax as intraoperative complications. *Acta Anaesthesiol Scand* 1997; 41(Suppl 111): 314–7.
- 10 Kaloud H, Smolle-Jüttner F-M, Prause G, List WF. Iatrogenic ruptures of the tracheobronchial tree. *Chest* 1997; 112: 774–8.
- 11 Benumof JL. High-frequency and high-flow apneic ventilation during thoracic surgery. In: Benumof JL (Ed.). *Anesthesia for Thoracic Surgery*. WB Saunders Company, Harcourt Brace Jovanovich Inc., 1987: 288–96.
- 12 Rouby J-J, Viars P. Clinical use of high frequency ventilation. *Acta Anaesthesiol Scand* 1989; 33(Suppl 90): 134–9.
- 13 Jain KK. Hyperbaric oxygen therapy in cardiovascular diseases. In: Jain KK (Ed.). *Textbook of Hyperbaric Medicine*, 3rd ed. Seattle: Hogrefe & Hogrefe Publishers, 1999: 393–419.
- 14 Shandling AH, Ellestad MH, Hart GB, et al. Hyperbaric oxygen and thrombolysis in myocardial infarction: the "HOT MI" pilot study. *Am Heart J* 1997; 134: 544–50.
- 15 Perera ER, Vidic DM, Zivot J. Carinal resection with two high-frequency jet ventilation delivery systems. *Can J Anaesth* 1993; 40: 59–63.

- 16 *Magnusson L, Lang FJW, Monnier P, Ravussin P.*
Anaesthesia for tracheal resection: report of 17 cases.
Can J Anaesth 1997; 44: 1282–85.
- 17 *Kindvall EP.* The multiplace chamber. *In:* Kindvall
EP (Ed.). Hyperbaric Medicine Practice, 2nd ed.
Flagstaff, Arizona: Best Publishing Company, 1995:
83–94.