CASE REPORTS

Non-invasive positive pressure ventilation for severe thoracic trauma

M. J. Garfield¹* and R. M. Howard-Griffin

Intensive Care Unit, Ipswich Hospital NHS Trust, Heath Road, Ipswich IP4 5PD, UK
¹Present address: Anaesthetic Department, Norfolk and Norwich Hospital, Brunswick Road, Norwich NR1 3SR, UK
*Corresponding author

A 35-year-old man was admitted to the intensive care unit (ICU) following a road traffic accident. He had sustained severe trauma to the left side of his chest, as well as other musculoskeletal injuries. After a short initial period of ventilation of the lungs via a tracheal tube, he was managed using a combination of continuous positive airway pressure and non-invasive positive pressure ventilation. He avoided ventilator-associated pneumonia, and spent a large part of his time on the ICU without any invasive monitoring lines, another potential focus of infection. He was discharged from the ICU after 25 days without having suffered any septic complications. The role of non-invasive positive pressure ventilation in severe thoracic trauma is discussed.

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Victims of thoracic trauma are often referred to critical care services for assistance with analgesia and ventilation. Some of these patients have historically required long periods of ventilatory support, and have therefore been at high risk of acquiring nosocomial pneumonia. We report a case in which a patient with severe thoracic trauma (Abbreviated Injury Scale score 5/6 for thoracic injury (where 0 represents no injury, and 6 represents an unsurvivable injury¹)) was managed mainly using non-invasive positive pressure ventilation (NIPPV), an approach which, we postulated, would be as effective as conventional ventilation, but expose him to a lower risk of nosocomial pneumonia.

Case report

A 35-year-old pedal cyclist was hit by, and trapped under, a heavy goods vehicle. He was extricated by the emergency services, and brought to the accident and emergency department on a spinal board. His Glasgow coma score was 15 throughout. He was complaining of severe left-sided chest pain and marked shortness of breath. He had a respiratory rate of 40 b.p.m., and the clinical features of a left flail chest. His oxygen saturation by pulse oximeter was 85% on a fractional inspired oxygen concentration (FIO₂) of 0.6. This was thought to be accurate as the probe was on an uninjured limb, there was a good trace on the monitor, and the patient was centrally cyanosed. He was tachycardic, with a heart rate of 140 beats min⁻¹, and hypotensive, with an initial systolic blood pressure of 55 mm Hg. He was resuscitated initially with crystalloid solution, followed by blood when it was available. He received a total of 5.5 litres of crystalloid, followed by 9 units of packed red blood cells. His blood pressure responded well, if transiently, to intravascular filling.

A chest x-ray (Fig. 1) showed fractures of the second–ninth ribs on the left, with disruption of the costovertebral joints, resulting in a large flail segment. There was severe contusion of most of the left lung and contusion of the right lower lobe. He had a disruption of his left shoulder joint, a large laceration over his left iliac crest, and a fractured left ankle. His Revised Trauma Score was 6.0, which represents an expected mortality of approximately 10%, and his Trauma Score – Injury Severity Score (TRISS) expected mortality was 18%.¹

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In view of his injuries and cardiorespiratory instability, his trachea was intubated and his lungs ventilated. A left chest drain drained initially 500 ml of blood, but the loss quickly decreased. A right chest drain was inserted prophylactically, in accordance with current Advanced Trauma Life Support (ATLS) teaching for a patient with severe thoracic injury requiring positive pressure ventilation. This approach has arisen in view of the high risk of an occult pneumothorax in such patients.

Computed tomography (CT) of the chest, cervical and thoracic spine, performed after stabilization confirmed the initial chest x-ray findings, as well as revealing bilateral small pneumothoraces and fractures of many of the spinous processes of the thoracic vertebrae. The neural arches were, however, intact and there was no evidence of spinal cord injury. The cervical spine appeared normal.

The patient’s lungs were ventilated using volume controlled ventilation, with a positive end-expiratory pressure (PEEP) of 6 cm H2O, and an \( FIO_2 \) of 0.35 (Servo 900C ventilator, Siemens-Elema, Sweden). This resulted in an arterial oxygen partial pressure \( (Pa_O_2) \) of 13.7 kPa. He was thought not to require an open lung strategy as his peak airway pressure was acceptable (<25 cm H2O), and oxygenation was not problematic. His initial cardiovascular instability was such that high levels of PEEP were thought inadvisable.

After a 48-h period of stabilization, and once his coagulopathy (a result of the massive transfusion) had been treated by infusion of fresh frozen plasma and cryoprecipitate, a thoracic epidural was inserted at the T7/8 level, and infusion of bupivacaine commenced. The epidural provided good analgesia for the thoracic injuries, but the shoulder fracture continued to produce a lot of pain. A patient-controlled analgesia infusion of morphine was therefore also commenced. These infusions resulted in good analgesia, ventilatory support was weaned off, and his trachea was extubated on the fourth day after admission.

On day 5, increasing oxygen requirement was treated with periods of intermittent positive pressure breathing (IPPB) via a Bird ventilator and continuous positive airway pressure (CPAP) by facemask. IPPB is a form of treatment, usually administered by the physiotherapists, which is designed to re-inflate collapsed pulmonary segments and so reduce shunting of deoxygenated blood. The patient uses a mouthpiece; the lungs are ventilated with a constant pressure. Despite this, by day 7 his \( FIO_2 \) had risen to 0.6, giving a \( PaO_2 \) of only 9 kPa. Facial CPAP at a level of 7.5 cm H2O was continued and negative fluid balances were obtained by fluid restriction and forced diuresis by loop diuretics. Fluid balance was monitored in the usual way by detailed input/output charts and urethral catheterization. These treatments resulted in reduction of his \( FIO_2 \) to 0.45.

On day 9, after internal fixation of his ankle, the patient’s lungs were ventilated overnight. Following tracheal extubation the next morning, he was given CPAP at a level of 7.5 cm H2O by facemask. At this stage he still had a large flail segment, and radiological and clinical evidence of continuing contusion in his left lung. The right basal contusion had now resolved.

Over the next 72 h, the patient developed increasing respiratory distress, with an increasing respiratory rate (up to 50 b.p.m.), shallow breathing and an uneven respiratory pattern, and the \( FIO_2 \) had increased to 0.6, in order to maintain a \( PaO_2 \) of 10 kPa. On day 12, NIPPV was commenced via a nasal mask (NIPPV2 ventilator, Aeromed, UK), with an immediate reduction in his oxygen requirements. He remained on continuous nasal NIPPV for a further 9 days, with a reducing level of pressure support and CPAP; the NIPPV was tolerated extremely well throughout. His invasive lines were all removed on day 18.

By day 20, he was receiving only intermittent NIPPV, and otherwise had an oxygen saturation (\( SaO_2 \)) of 98% on 3 litres min\(^{-1}\) of oxygen via nasal cannulae. On day 22, he was given NIPPV overnight only, and the NIPPV was discontinued on day 24. Nocturnal CPAP by facemask continued for a further night, and he was discharged to the ward on day 26.

Apart from a 5-day course of flucloxacillin to treat a Staphylococcus aureus infection in a laceration, the patient required no antibiotics during his stay on the intensive care unit (ICU). At no stage was there any evidence of systemic or pulmonary infection.

**Discussion**

Ventilator-associated pneumonia represents a major subgroup of nosocomial pneumonia, a condition that is associated with a mortality of 30%. In those patients who survive, ventilator-associated pneumonia is associated with increased morbidity, longer ICU stays, and hence increased costs. The pathogenesis of this condition involves bacterial colonization of the upper aerodigestive tract, and hence oropharyngeal secretions. These contaminated secretions
pool above the cuff of the tracheal tube, and are aspirated into the trachea during patient movement, coughing, tracheal suctioning and many other manoeuvres.\textsuperscript{2,9} Tracheal intubation has indeed been shown to be an important risk factor for the development of ventilator-associated pneumonia,\textsuperscript{10} and so it would seem sensible to avoid this if at all possible.\textsuperscript{11}

NIPPV has been shown to be as effective as conventional ventilation in a variety of settings, including exacerbations of chronic obstructive pulmonary disease\textsuperscript{12} and acute respiratory failure.\textsuperscript{13} The causes of the acute respiratory failure in the latter paper ranged from infection and pulmonary oedema to acute respiratory distress syndrome. To date, there have been no reports of the use of NIPPV in severe chest trauma.\textsuperscript{14}

Nava \textit{et al.}\textsuperscript{12} allocated random patients with exacerbations of chronic obstructive pulmonary disease to either NIPPV or conventional ventilation, after an initial period of 48 h conventional ventilation. Seven out of 25 patients in the conventionally ventilated group developed ventilator-associated pneumonia, compared to none of the NIPPV group (\textit{P}=0.009). Antonelli \textit{et al.}\textsuperscript{13} allocated random patients to either NIPPV or conventional ventilation from the outset. Again, the patients ventilated non-invasively had a significantly lower incidence of ventilator-associated pneumonia and sinusitis. While this study had some minor methodological flaws, it demonstrates a clear difference in infective complications between the two groups, and adds further evidence to the hypothesis discussed above, that avoidance of tracheal intubation reduces the incidence of ventilator-associated pneumonia, and thus decreases length of ICU stay and other variables.\textsuperscript{6}

Thoracic injury consists of a spectrum of disease, ranging from rib fractures with mild pulmonary contusion to disruption of the thoracic cage and acute respiratory distress syndrome. Although many of the less severely injured patients are now managed without ventilatory support, with a thoracic epidural and aggressive chest physiotherapy,\textsuperscript{14} there will always be a number for whom this is not sufficient. These patients, usually with significant hypoxaemia, often require an extended period of invasive ventilation,\textsuperscript{15} and the risk of ventilator-associated pneumonia is thus high. We believe that in this group, avoidance of tracheal intubation and hence avoidance of ventilator-associated pneumonia may be a major step in reducing ICU stay, morbidity and mortality. NIPPV is safe, effective, and, we believe, may be the mode of choice for managing patients with thoracic trauma who have no contraindications to its use. We have since used NIPPV in a further two patients with bilateral flail chest and lesser degrees of pulmonary contusion, and had very similar results with regard to effectiveness of the ventilatory support, and avoidance of ventilator-associated pneumonia.

\textbf{References}

Lobectomy for cavitating lung abscess with haemoptysis: strategy for protecting the contralateral lung and also the non-involved lobe of the ipsilateral lung

J. Pfitzner1*, M. J. Peacock2, E. Tsirgiotis3 and I. H. Walkley1

1Department of Anaesthesia, 2Thoracic Surgical Unit and 3Respiratory Medicine Unit, North Western Adelaide Health Service, 28 Woodville Road, Woodville, South Australia 5011, Australia
*Corresponding author

We describe the anaesthetic management of a patient undergoing lobectomy for cavitating lung abscess complicated by haemoptysis. Surgery for lung abscess is one of the absolute indications for the use of a double-lumen tube (DLT). Because pus or blood could impede fibreoptic-assisted DLT placement, a traditional, blind placement of the DLT was performed. To protect the uninvolved parts of the operated lung, ventilation of the lung with the abscess was not performed until the resection of the involved lobe had been completed.

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Current management of lung abscess1–3 in the main involves appropriate antibiotics, physiotherapy and, where indicated, chest tube drainage: thoracotomy tends to be restricted to those cases not responding to intensive and prolonged medical management or those complicated by haemoptysis,4 empyema, malignancy or suspected malignancy. Thus, unlike the era before antibiotics, today the thoracic anaesthetist has limited experience in anaesthetizing patients with cavitating lung abscesses. One of the main aims of anaesthesia is to protect the so-called good lung from contamination, and the introduction of the endobronchial tube and the double-lumen tube (DLT) in the 1950s overcame the need to employ awkward and inconvenient patient positioning, such as the Parry Brown or the Overholt positions. The former relied upon intra-operative drainage by gravity of infective material via the trachea, while the latter relied upon retention by gravity within the abscess cavity. Now, the use of a DLT or a tube-and-blocker is mandatory during thoracotomy for lung abscess or for frank haemoptysis.5

Even so, when planning anaesthesia for a patient with a high risk of purulent material and/or blood draining by gravity into the major airways, the anaesthetist and surgeon still face several dilemmas. First, should the patient stay in the sitting or semi-sitting position until lung separation is achieved? Secondly, what is the operative team’s preferred DLT placement plan for such a case? Thirdly, should an effort be made to minimize the dissemination of infective material throughout the operated lung as well as the good lung?

Case report

Pneumonia that had failed to resolve over a 12-month period, in a 32-year-old previously well woman, led to a consolidated left lower lobe with a large cavitating lung abscess. Early in this disease, she had developed haemoptysis and on many occasions had coughed varying amounts of frank blood, sometimes in excess of an estimated half-teacup. Later, she was coughing purulent sputum streaked with blood. She said that the episodes of haemoptysis were caused by her lying flat, and as a consequence she slept in a sitting position and would not allow herself to be placed otherwise. Such was her fear of further haemoptysis that she refused physiotherapy and totally rejected any suggestion of postural drainage. Her weight decreased from 75 to 46 kg, and she developed marked clubbing of the fingers and toes. The preoperative chest x-ray and CT scan are shown in Figures 1 and 2 respectively.

Extensive investigation failed to identify any underlying pathology. There was no evidence of immunosuppression or aspiration. Bronchoscopy revealed no endobronchial lesion and washings were positive for Pseudomonas aeruginosa. She was admitted on several occasions with fever, increas-
ing dyspnoea and/or haemoptysis, and although on each occasion there was a response to broad-spectrum intravenous antibiotics, lobectomy was advised when the haemoptysis became more voluminous and she became significantly anaemic. The favoured preoperative diagnosis was either localized bronchiectasis or pulmonary sequestration. Histopathology of the resected lobe revealed an actinomycosis abscess but no other underlying abnormality.

The patient was anaesthetized in a semi-sitting (50° head-up) position, with meticulous preoxygenation, midazolam 1 mg, fentanyl 200 μg in divided doses, an initial dose of propofol 80 mg and rocuronium 40 mg. While the patient was maintained in the 50° head-up position, gentle hand ventilation with oxygen was performed for 3 min. The patient was then placed horizontally and, without further ventilation, elective rigid bronchoscopy was performed by the surgeon to assess bronchial anatomy/pathology and to perform tracheobronchial suction. An estimated 3 ml of purulent material was removed by direct suction from the trachea and airways, and the surgeon advised that the major airways were small.

In the light of this comment, and with fibreoptic bronchoscope and left and right Robertshaw DLTs immediately at hand, a 35 FG Left Sheridan DLT was inserted blind, to the perceived desired depth. Inflation of the bronchial cuff to a tension greater than usual in the 5 ml inflating syringe and the pilot cuff was achieved with a relatively small volume of air. Cautious hand ventilation via a single catheter-mount connected to the DLT bronchial lumen was observed to produce left chest movement. Transfer of ventilation to the tracheal lumen produced right chest movement, and the tracheal cuff was inflated to produce a seal. Next, the single catheter mount was transferred back to the bronchial lumen and auscultation over the anterior apical region of the left chest during gentle hand ventilation confirmed the presence of vesicular breath sounds. From that point onward, single-lung intermittent positive pressure ventilation was restricted to the right lung until the left lower lobe resection had been completed. During the period when it was not ventilated, the left lung was connected to a 3-litre oxygen reservoir at ambient pressure. For reasons that will be discussed, the fibreoptic bronchoscope was not used either before or after positioning the patient for surgery.

During the chest incision and until the thoracic cavity was opened, the reservoir bag was seen to distend and collapse as expected with each cycle of single-lung ventilation to the dependent lung. This tidal movement of the reservoir bag was in this case less pronounced than usual and, although this was most likely a consequence of the left lower lobe pathology, the possibility of distal migration of the DLT to occlude or partially occlude the left upper lobe bronchial orifice was also considered. However, because the peak and plateau inflation pressures in the ventilated dependent right lung were as expected and because there was no reduction in pulse oximetry while performing single-lung ventilation with a fresh gas flow of 50% N₂O/O₂, no intervention was thought necessary or justifiable. This decision was vindicated by prompt left upper lobe collapse when the chest was opened and easy re-expansion when the surgery was completed.

Once the chest was opened and the operated lung was able to collapse down, the oxygen reservoir was temporarily

![Fig 1 Posteroanterior chest x-ray revealing a left lower lobe opacity and an abscess with fluid level.](image1)

![Fig 2 CT scan at the level of the apical segment of the left lower lobe, revealing consolidation and cavity formation.](image2)
detached from the bronchial lumen of the DLT while further purulent material was removed by suction. Only then was some air cautiously removed from the DLT bronchial cuff to return it to a more usual tension. This was performed whilst listening with an ear pressed to the reservoir bag in order to immediately identify possible gas leakage past the cuff during the inspiratory phase of ventilation. At surgery, the left lower lobe was distended and also patchily consolidated. It was densely adherent over a large part of its diaphragmatic surface, but, apart from the need to divide these adhesions, the lobectomy was uneventful and the chest was closed with an apical and a basal drain. Before the re-expansion of the left upper lobe, the bronchial tree on that side was again cleared by suction via the DLT bronchial lumen. At this time it was noticed that a considerable volume of purulent material had drained from the operated lung, filling the DLT bronchial lumen and extending into the tubing leading to the reservoir bag.

Throughout the period of single-lung ventilation and an $P_{1O_2}$ reading of approximately 0.4, the $S_{PO_2}$ remained between 98 and 100%.

Postoperative recovery was uneventful, and the patient was able to lie happily in whatever position she liked.

**Discussion**

With a cavitating lower lobe abscess, the first dilemma facing the anaesthetist and surgeon is whether or not to aim to achieve DLT lung separation while the patient is maintained in a steep sitting or semi-sitting position. With the case described, this decision was influenced by the fact that a preoperative rigid bronchoscopy was planned in order to make a surgical assessment of the pathology, to clear secretions and to identify any atypical bronchial anatomy. Conacher has said for the same reasons, with regard to the need for rigid bronchoscopy in the assessment or therapy of bronchopleural fistula, that ‘most experienced personnel prefer to base the anaesthetic technique around this requirement’.

For the rigid bronchoscopy, the patient was anaesthetized in the semi-sitting position (about 50° from horizontal) but was put in the horizontal position with the onset of good muscle relaxation. The bronchoscopy and bronchial suction under direct vision were performed without delay, and no further ventilation was performed until after the DLT was positioned and the bronchial cuff inflated. Even so, some management teams might strive to retain the patient in the semi-sitting position for the bronchoscopy and until such time as lung separation is secured.

The course of action chosen depends on the preferred practice of the individual surgical/anaesthesia team and by the clinical circumstances of the individual patient. Rigid bronchoscopy with the patient 50° head-up used to be the accepted management for sputum retention after pulmonary resection, but today it is a disappearing skill. Any partial reduction of the degree of head-up tilt of our patient might well have resulted in spillage of infective material into the lower lobe bronchus of the ‘good’ lung.

Alternatively, performing or attempting to perform fiberoptic assessment and fiberoptic-assisted intubation with a patient maintained in the sitting position can be difficult or prolonged if secretions obscure vision.

Should suxamethonium have been used? Probably not in this patient, but it would certainly be advisable in any patient in whom difficulties might be expected with gentle face-mask hand-ventilation.

Should anaesthesia have been induced with the patient sitting on the horizontal lower half of an operating table that is broken at the mid-point, with the patient’s thorax reclining, abscess-side dependent, against the steeply angulated upper half? Probably, yes! Once asleep and paralysed, the patient could easily have been maintained abscess-side dependent, and the head-up tilt could have been reduced if necessary to facilitate bronchoscopy and intubation.

The next dilemma relates to the choice of DLT and the plan for its placement. The necessary decisions will depend on previous experience and preferences. In this case, a range of DLTs and a fiberoptic bronchoscope were immediately at hand. The first management option was the blind placement of a 35 FG Left Sheridan DLT, because it was felt that pus or blood might obscure the view at fiberoptic bronchoscopy. A plastic tube was chosen in preference to a Robertshaw DLT because the external diameter of a medium Robertshaw could have been too tight for the reputedly small airways. Otherwise, the more firmly anatomically shaped Robertshaw tube would have been chosen. This DLT frequently appears to drop into place and, in the authors’ view, is less likely to become dislodged by surgical traction in the course of the operation.

If the left-sided Sheridan DLT had entered the right rather than the left main bronchus, the plan was to replace it immediately with a medium or small left Robertshaw. If the left Robertshaw had also entered the right main bronchus, it was to have been replaced immediately with a right Robertshaw. Use of the fiberoptic bronchoscope to guide one of the left-sided clear plastic tubes down the left main bronchus was considered as a last resort in our team’s order of priorities for this particular patient. However, since a plastic and not a Robertshaw DLT was being used, a good case could have been made for positioning a fiberoptic bronchoscope down the tracheal lumen and observing, if secretions had permitted, the passage of the bronchial component into the left main bronchus.

A conscious decision was made not to check DLT placement with the fiberoptic bronchoscope that was immediately at hand. The small volume of air injected into the bronchial cuff of the left-sided DLT to produce an airtight seal, the free gas movement in and out of the right lung and the confirmation of vesicular breath sounds in the left upper lobe by the anaesthetist who was also performing the gentle hand ventilation were considered assurance.

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enough. With these findings and in this particular case, the anaesthetist would not have deflated the cuffs and pulled back the tube, even if bronchoscopy via the DLT tracheal lumen had failed to reveal any sign of the bronchial cuff.

The third dilemma relates to the protection of the healthy parts of the lung that contains the abscess. The frequent removal of secretions during surgery by suction via the DLT lumen on the diseased side is the standard recommendation.5 A further and not previously reported measure is to refrain from ventilating the lung with the abscess until the risk of contamination is removed. If adopted, this will involve a period of single-lung ventilation while the chest is being opened and hence before the non-ventilated lung is able to collapse away from the chest wall. We recommend that the airway of the non-ventilated lung is connected to an ambient-pressure oxygen reservoir to prevent ambient air and therefore nitrogen from entering the non-ventilated lung.10 This practice has several benefits, both theoretical and practical.6

One of the possible theoretical benefits while the patient is being positioned ready for surgery and while the chest is being opened is the oxygenation of the blood that continues to flow through the non-ventilated but not yet collapsed lung. This results in an atypical ventilation/perfusion mismatch, with good oxygenation of the shunted pulmonary blood flow but with no elimination of carbon dioxide once the \( P_{CO_2} \) of gas in the reservoir bag has equilibrated with that of the mixed venous blood. This atypical mismatch will in theory be expected to result in a small further increase in the relatively large arterial to end-tidal carbon dioxide gradient seen during thoracic anaesthesia.11 Thus, if there is a delay in opening the chest and hence a delay in lung collapse, arterial blood gases may be required to guide the level of minute volume ventilation.

In the case presented, an additional practical benefit of the ambient-pressure oxygen reservoir was demonstrated. Once the chest was opened and surgery was progressing, the anaesthetist was able, by listening with the ear pressed against the reservoir bag,6 to deflate the bronchial cuff carefully to a more usual tension without risk of creating an unidentified loss of the airtight seal.

The small tidal movement in the reservoir bag before the chest was opened was probably caused by the pathological process in the left lower lobe, and by infective material draining by gravity and obstructing or partially obstructing major airways on that side. It may also have been partly a consequence of dependent lung ventilation causing mainly diaphragmatic rather than mediastinal displacement in this very thin patient.10

In conclusion, anaesthesia for thoracotomy for cavitating lung abscess, with or without a history and hence risk of haemoptysis, should involve preoperative physiotherapy to reduce secretions, appropriate patient positioning with the lobe containing the abscess dependent relative to the rest of the bronchial tree, careful preoxygenation, and an anaesthesia plan that achieves prompt lung separation. Elective rigid bronchoscopy before DLT placement allows both surgical assessment of bronchial pathology and anatomy, and the efficient removal of secretions or pus. Once lung separation is achieved, not ventilating the lung with the abscess may reduce the risk of contamination of healthy parts of this lung. While the lung containing the abscess is not being ventilated, there are both theoretical and practical benefits in connecting this lung to an oxygen reservoir at ambient pressure. After the diseased lobe has been excised, suction via the DLT lumen on that side before re-expansion of the residual lobe will remove any purulent material that has drained from the abscess in the course of surgery.

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Adult epiglottitis: an under-recognized, life-threatening condition

W. A. Ames1*, V. M. M. Ward2, R. M. D. Tranter2 and M. Street2

1Department of Anaesthesia, 1G323 University Hospital, 1500 East Medical Center Drive, Ann Arbor, MI 48109-0048, USA. 2Royal Sussex County Hospital, Eastern Road, Brighton, UK

*Corresponding author

Epiglottitis in the adult can be fatal and should be treated with the same degree of concern and suspicion in respect of airway patency as in children. We present three cases of adult epiglottitis in which the airway was lost prior to or during the intervention of an anaesthetist. We suggest that an emphasis on conservative management is distracting and belies the serious nature of this disease.

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Epiglottitis is an inflammation of the supraglottic structures that can occur at any age. There is a reported increase in the incidence of epiglottitis in the adult population.1 In contrast to the aggressive management of epiglottitis in children, a more conservative approach has been advocated in adults.2 We present three cases of adult epiglottitis where the airway was lost either before or during intervention of an anaesthetist.

Case reports

Case 1

A 41-yr-old male presented with a sudden onset of sore throat, stridor and dyspnoea. He was previously fit but regularly smoked heroin. On arrival to the accident and emergency department, he rapidly developed complete airway obstruction resulting in a respiratory arrest. Manual ventilation by facemask proved ineffective and no i.v. access could be secured. Laryngoscopy was impossible because of trismus. Intravenous succinylcholine 100 mg was injected and permitted laryngoscopy. A large mass arising from the epiglottis was observed, completely obscuring the larynx. Following oxygenation via an emergency cricothyroidotomy, the trachea was intubated nasally using a fibreoptic bronchoscope. Intravenous cefotaxime was commenced. A surgical tracheostomy was performed under general anaesthesia and the patient admitted to a high dependency area on the otolaryngology ward. A lateral soft tissue neck radiograph (Fig. 1), MRI scan (Figs 2 and 3) and fibreoptic nasendoscopy (Fig. 4) were performed once the airway had been secured. The patient made a full recovery. Candida albicans was eventually cultured from the mass.

Case 2

A 73-yr-old male was admitted with less than a 24-h history of sore throat, dysphagia and dyspnoea associated with mild inspiratory stridor. Fibreoptic nasendoscopy in the accident and emergency department revealed a swollen, hyperaemic epiglottis. Blood cultures were taken and he was commenced on i.v. cefotaxime and dexamethasone 4 mg. Four hours later, on the otolaryngology ward, his respiratory status deteriorated rapidly. A surgical tracheostomy was attempted under local anaesthesia but the procedure abandoned when the patient became hypoxic and began convulsing. The anaesthetist was able to intubate the trachea orally in the unconscious patient, prior to transfer to the operating room for conventional tracheostomy. The patient was admitted to the intensive care unit and made an uneventful recovery. The trachea was decannulated 4 days later. Two blood cultures taken on the day of admission both grew Neisseria meningitides, later typed as Group Y. There was no other positive microbiology and virology screen was normal.

Case 3

A 56-yr-old man was admitted to the accident and emergency department with acute onset of a sore throat and dysphagia. He was sitting up, dyspnoeic, and drooling saliva. A flexible nasendoscopy showed an oedematous, enlarged epiglottis. An i.v. line was inserted, blood cultures taken and Augmentin 1.2 g and hydrocortisone 200 mg were given intravenously. The patient was transferred to the operating theatre. During an inhalational induction of anaesthesia with sevoflurane, he developed complete airway
obstruction. Intravenous succinylcholine 100 mg was administered and orotracheal intubation was attempted but failed. An emergency cricothyroidotomy was then performed using a 13-gauge transtracheal catheter. Jet ventilation was commenced and continued whilst a surgical tracheostomy was performed. The trachea was decannulated 3 days later. Blood cultures were negative.

**Discussion**

Adult epiglottitis has an incidence of between 1–4 per 100 000 per annum.² ⁴ ⁵ It is described as having a low morbidity and mortality.² ⁵ Nonetheless, mortality in adults is around 7%, and some reports suggest it is as high as 20%.³ Hingorani and colleagues⁶ observed that a similarly high mortality in children prompted a change to an interventional practice. Even though adult epiglottitis is unpredictable and occasionally catastrophic in outcome, conservative management is still preferred by many authors.² ⁵
**Haemophilus influenzae** type B is found in as few as 17% of adult patients with suspected epiglottitis. There is also a high rate of negative blood cultures possibly suggestive of a viral cause (although only herpes simplex has been reported). Meningococcal epiglottitis is extremely rare and we present only the seventh reported case; the first was reported in 1995. Alternatively, epiglottitis may occur following mechanical injury such as the ingestion of caustic material or the inhalation of hot objects, smoke or vapours. Epiglottitis following illicit drug use has been described as a result of the accidental inhalation of a heated objects. Although there has been an association with cigarette smoking, there are no reports linking epiglottitis and the smoking of heroin. The causal organism or factor is, therefore, often less identifiable in adults than in children.

An adult with epiglottitis usually presents with symptoms of sore throat and painful dysphagia. Drooling and stridor are infrequent presenting signs. In fact the presence of stridor, dyspnoea and a short duration of symptoms prior to presentation, are all described as predictors of airway loss in the adult with epiglottitis. This is, however, controversial. For example, Wolf presented 30 patients in whom stridor was present and who were subsequently successfully treated conservatively. On the other hand, Mayo-Smith described a patient who had no history of stridor, yet suddenly developed airway obstruction and died. Indeed the three cases we present support the fact that the disease presentation and progression is variable and that there are no reliable markers that predict the need for invasive airway support.

The diagnosis of epiglottitis is essentially clinical but can be supported by indirect laryngoscopy. Typically, there is diffuse swelling of the aryepiglottic structures unlike the classic cherry red epiglottis in children. Otherwise, once the airway is deemed safe, a lateral, soft tissue radiograph may show a thickening of the epiglottis (‘thumb print sign’; see Fig. 1). Ducic and colleagues have proposed the ‘vallecula sign’ to improve the diagnostic accuracy of soft tissue radiographs. This stepwise approach attempts to identify the vallecula as it nears the level of the hyoid bone. In the absence of a ‘deep and well-defined vallecula’, the radiological findings support the diagnosis of epiglottitis.

If the clinical diagnosis of epiglottitis is made and the airway judged to be at risk, intervention should not be delayed by attempts to obtain cultures or radiographs. Treatment should begin promptly with intravenous antibiotics. Steroids have no accepted place and the benefit of epinephrine, either nebulized or intramuscular, has yet to be confirmed.

Either orotracheal intubation or tracheostomy may be performed under local anaesthesia but both are potentially stimulating procedures which may precipitate sudden loss of the airway. General anaesthesia may be performed with an inhalational induction but can be complicated by a relatively prolonged excitation phase in adults (as occurred in case 3). Friedman therefore recommended a rapid sequence induction with the facility to perform a cricothyroid puncture if intubation proves difficult. Bag and mask ventilation can simply worsen or complete the airway obstruction and should be avoided.

Neuromuscular blocking drugs are traditionally avoided in epiglottitis (although succinylcholine was listed on a recent protocol for the management of paediatric epiglottitis). We used succinylcholine in two of the above case reports, including one intralingual injection. Although this route is described in children, we could find no reference to its use in adults.

In summary, a belief that epiglottitis is rare in adults has contributed to misdiagnosis and high mortality rates. We present three cases that are typical of adult epiglottitis, in that there is no one identifiable causal agent or factor that would allow rationalisation of a particular therapy. To advocate conservative management belies the aggressive nature of this disease. We believe there should be a greater emphasis on early interventional support of the airway.

References

Falsely elevated bispectral index during deep hypothermic circulatory arrest

G. Mychaskiw*, B. J. Heath and J. H. Eichhorn

University of Mississippi School of Medicine, Cardiac Anesthesiology, University of Mississippi Medical Center, 2500 North State Street, Jackson, MI 39216-4505, USA

*Corresponding author

A 2-month-old infant underwent repair of a ventricular septal defect under deep hypothermic circulatory arrest. Bispectral index and EEG suppression ratio were evaluated using an Aspect BIS monitor. Erroneous readings from the monitor could have led to a potentially dangerous alteration in surgical and anaesthetic management.

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The bispectral index (BIS) is rapidly gaining acceptance as a measure of hypnosis under anaesthesia. Additionally, the BIS monitor unit can be used as a readily available and easily interpreted tool to ensure EEG burst suppression during deep hypothermic circulatory arrest. This is displayed as a suppression ratio, which is the percentage of time during the preceding 60 s in which EEG burst suppression occurred. We report a case where a BIS monitor (Aspect Medical Systems, Spotsylvania, VA, USA) demonstrated erroneous readings during deep hypothermic circulatory arrest that could have altered the anesthetic and surgical management of the patient.

Case report

A 2-month-old, 3 kg male patient presented to the cardiac surgery service for repair of a ventricular septal defect (VSD). Cardiac catheterization revealed a large muscular VSD with a pulmonary to systemic blood flow ratio of greater than 4 to 1. The patient was medically managed with digoxin and furosemide and had no clinical evidence of congestive heart failure at the time of surgery.

After placement of standard, non-invasive monitors, the patient was anaesthetized with fentanyl, 10 μg kg⁻¹, vecuronium, 0.1 mg kg⁻¹, 30% oxygen in nitrous oxide, and isoflurane, 0.5–2.0 vol%. Anaesthesia was maintained with the volatile agent and occasionally supplemented with fentanyl and vecuronium. Additional monitors placed after induction included a femoral arterial catheter, internal jugular triple lumen catheter, Aspect model A-2000 BIS monitor (system revision 0.41) and cerebral venous oximeter (Somanetics Corporation, Troy, MI, USA). In preparation for deep hypothermic circulatory arrest, methylprednisolone, 10 mg kg⁻¹ was administered and the head was packed in ice. Following anticoagulation with heparin, aortic and right atrial cannulae were placed and cardiopulmonary bypass (CPB) was started. At the start of CPB the BIS was in the 40s. The patient was cooled to a core temperature of 18°C and deep hypothermic circulatory arrest was instituted after 15 min of cooling time on CPB. At the time of deep hypothermic circulatory arrest the BIS was zero with a suppression ratio of 100. The cerebral mixed venous oxygen saturation (SvO₂) had increased from baseline by more than 100% during cooling.

Within the first 5 min of deep hypothermic circulatory arrest, the Svo₂ decreased to baseline. By 15 min of deep hypothermic circulatory arrest the Svo₂ had decreased over 50% from baseline and was below the limits of measurement. The patient’s core temperature had started to slowly drift upward and by this time had reached 22°C. At 16 min of deep hypothermic circulatory arrest the BIS abruptly increased to 98–100, with a suppression ratio of 0–10 (Fig. 1). The raw EEG tracing on the BIS monitor appeared flat and unchanged. The EMG readout on the monitor throughout CPB was negligible. The surgeon was advised of the monitor readings and was asked to resume CPB as soon as possible. The repair was expedited and the patient returned to CPB within 5 min. Immediately on return to CPB, the Svo₂ again increased to over 100% of baseline. After approximately 10 min of rewarming the BIS returned to its pre-deep hypothermic circulatory arrest level in the 40s. The patient was weaned from CPB and the procedure concluded without further incident. He was
extubated the next morning having suffered no apparent neurological sequelae.

**Discussion**

The bispectral index is an artificial variable arrived at by computer processing of, most commonly, a one-channel EEG pattern via a proprietary algorithm of the Aspect Corporation. This value, scaled from 0 to 100 reflects a patient’s level of hypnosis, and potentially, probability of recall.\(^1\) This has become an important, if somewhat emotionally charged, anaesthetic safety issue in the last few years. Some have even speculated that BIS monitoring will become the next ‘standard of care’.\(^2\) This has led to a rapid proliferation in the number of BIS monitors available in operating rooms across the USA. The Aspect monitors, the only presently available, are easy to use and interpret, requiring very little time to fully orient and train most anaesthesia practitioners. An added advantage of the Aspect monitors is their ability to display real-time EEG tracings, accompanied by the suppression ratio. With the caveat that this offers a very limited picture of the total brain EEG activity, the BIS monitors are thus a readily available and easily used EEG processing system for the detection of gross global changes.

EEG monitoring during deep hypothermic circulatory arrest is of controversial benefit. It has been suggested that outcome is improved when EEG burst suppression is achieved by cooling.\(^3\) Burst suppression has been used as an indicator of adequate brain protection during deep hypothermic circulatory arrest.\(^4\) There are other studies, however, which do not demonstrate improved outcome from use of EEG monitoring.\(^5\) As a result of these issues, coupled with the logistical and economic pressures of equipment and personnel availability, EEG monitoring during deep hypothermic circulatory arrest is not applied universally and varies greatly among institutions. The wide availability and ease of use of the Aspect BIS monitors obviates some of these concerns. Although its benefit has not been demonstrated in peer-reviewed studies, the suppression ratio detected by BIS monitors may be of value in helping to assess optimal brain protection prior to deep hypothermic circulatory arrest.

In this case the brain was cooled for 15 min prior to deep hypothermic circulatory arrest. The period of cooling was somewhat shorter than the usual practice at this institution, and was dictated by the surgeon as he felt the arrest time would be very brief for anatomic reasons. This appeared to have the desired result of metabolic suppression, as evidenced by the significant increase in $S_{\text{R}O_2}$. Appropriately, the BIS decreased while the suppression ratio increased, correlating with EEG suppression induced by cooling. The rapid decrease in $S_{\text{R}O_2}$ following deep hypothermic circulatory arrest, however, was disturbing and suggested a possible depletion of oxygen stores in the brain, which may be seen following heterogeneous cooling, especially following an abbreviated cooling time. As the temperature drifted upward, the BIS increased abruptly to high levels at 23°C after 16 min of deep hypothermic circulatory arrest. The most worrisome thought was that this could possibly represent the monitor’s interpretation of seizure EEG secondary to anoxic insult. The surgeon was advised of these concerns, but this was tempered by the real-time EEG waveform, which appeared to be highly suppressed. It was our impression that this was most likely an artefactual product of the algorithm’s misinterpretation of EMG activity or other radiofrequency interference. The ice surrounding the patient’s head was wrapped in plastic and towelling, thus increased electrode impedance was not thought to be a factor. The surgeon quickly completed the repair and the patient was returned to CPB. When the brain had been rewarmed, the BIS returned to pre-cooling levels, further reinforcing our belief that this was an artefact, although amelioration of seizure activity by improved perfusion remained a possibility. In approximately 30 deep hypothermic circulatory arrest cases prior to the one reported, the BIS had not displayed this activity, but rather remained highly suppressed until the brain had been rewarmed. It has also not appeared in the 10 cases subsequent to the case reported. In the postoperative period the patient demonstrated no evidence of neurologic insult and was discharged home after an uneventful recovery.
Both EMG activity and radiofrequency interference from electrical equipment in the operating theatre influence the BIS algorithm. Under normal conditions, the EEG is of sufficient power to override these other electrical sources and is thus weighted appropriately by the algorithm. In circumstances of extreme EEG suppression, as during deep hypothermic circulatory arrest, it is possible that electrical interference from either EMG or radiofrequency noise may be interpreted by the algorithm as EEG activity and assigned a high BIS value. When EEG activity resumes, as during rewarming, the monitor then re-interprets it appropriately, discounting the EMG and radiofrequency activity in the algorithm (personal communication, David Zaraket, Aspect Corporation). This appears to have happened in our case, as there was no demonstrable evidence of neurologic insult, such as seizure activity or choreoathetosis, seen post-operatively.

In conclusion, we present a case where electrical interference was misinterpreted as EEG activity by an Aspect A-2000 BIS monitor during a period of intense EEG suppression. This misinterpretation was displayed as a high BIS value with a low suppression ratio. Observation of the real-time EEG waveform suggested that this was an artefact, but was not absolutely conclusive. During periods of significant EEG suppression, BIS monitors may misinterpret electrical interference as EEG and could possibly lead to unnecessary therapeutic interventions. Observation of the real-time EEG waveform may aid in the diagnosis of this artefact, which, in our experience, occurs infrequently. Further studies are necessary to determine if monitoring suppression ratio and BIS during deep hypothermic circulatory arrest is beneficial and cost effective.

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Perioperative management of a patient requiring surgery for pituitary apoplexy and severe angina pectoris

H. Kitagawa*, K. Takahashi, Y. Hirasaki and T. Ishii

Department of Anesthesia, Nagahama City Hospital, 313 Ohinui-cho, Nagahama, Shiga, 526-8580, Japan

*Corresponding author

We describe the management of a 71-yr-old man with pituitary apoplexy and severe angina pectoris who underwent treatment of an intra-cranial haemorrhage and open-heart surgery requiring anticoagulant therapy within a very short period. Subtotal removal of the pituitary tumour was undertaken under stable cardiovascular conditions. But ventricular fibrillation occurred after the neurosurgery in the intensive care unit. After the patient was defibrillated, intra-aortic balloon pumping was necessary to assist coronary artery blood flow. Twenty hours after neurosurgery, oozing from the surgical wound stopped and coronary artery bypass grafting with full heparinization was performed uneventfully.

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Pituitary apoplexy is a clinical syndrome of sudden haemorrhagic necrosis of the pituitary gland.\textsuperscript{1,2} Subsequently, tumour expansion causes headache, visual field deficit and ophthalmoplegia. Recovery of the patient depends on the duration and severity of symptoms. Surgical treatment of intracranial haemorrhage is recommended immediately after appearance of the neurological symptoms.

We experienced a rare case of a patient with pituitary apoplexy coexisting with severe angina pectoris, which required urgent coronary artery revascularization. However, anticoagulant therapy for cardiopulmonary bypass (CPB) might have induced intra-cranial haemorrhage. Pituitary apoplexy has been reported to occur following coronary artery bypass grafting.\textsuperscript{3-7} Sequential management of the intracranial haemorrhage and open-heart surgery requiring anticoagulation present a challenge to the anaesthetist.

Case report
A 71-yr-old man (weight 54 kg) was referred to our hospital for surgical treatment of severe triple-vessel coronary artery disease. On admission, he was suffering frequent episodes of angina pectoris at rest requiring treatment with nitroglycerine (0.3 \text{ \textmu }g \text{ kg}^{-1} \text{ min}^{-1}), diltiazem (0.06 \text{ mg kg}^{-1} \text{ h}^{-1}) and lidocaine (1 \text{ mg kg}^{-1} \text{ h}^{-1}) i.v. He had a past medical history of hypertension and a myocardial infarction.

A pituitary tumour had been diagnosed by cranial computed tomography (CT) 2 days before admission. He had had a slight headache but no other neurological deficits. In hospital awaiting cardiac surgery, he became comatose and was found to have a Glasgow Coma Score (GCS) of 6. Neurological examination revealed dilated bilateral pupils, a visual field deficit, ophthalmoplegia and proptosis. Review of the CT showed a fairly large, iso-dense and high-density sellar mass with haemorrhage. The tumour extended into the suprasellar cistern with compression of the optic nerves and cavernous sinus on both sides. There was haemorrhage into the ventricle and brain tissue.

He underwent emergency neurosurgery before cardiac surgery. In the operating room, invasive blood pressure, central venous pressure, pulse oximetry, end-tidal carbon dioxide and the electrocardiogram were monitored. Anaesthesia was induced with midazolam 2 mg and fentanyl 200 \text{ \mu g}. Pancuronium 8 mg was administered to facilitate tracheal intubation. Anaesthesia was maintained with 0.5–2% sevoflurane and an i.v. fentanyl (2 \text{ \mu g kg}^{-1} \text{ h}^{-1}) infusion, and muscle relaxation was maintained with intermittent boluses of pancuronium. Mechanical ventilation with a tidal volume of 10 ml kg\textsuperscript{-1} and respiratory rate of 12 min\textsuperscript{-1} was adjusted to maintain normocapnia. Dopamine 3–7 \text{ \mu g kg}^{-1} \text{ min}^{-1}, nitroglycerine 0.15–0.7 \text{ \mu g kg}^{-1} \text{ min}^{-1}, diltiazem 0.06–0.1 mg kg\textsuperscript{-1} \text{ h}^{-1} and lidocaine 1 mg kg\textsuperscript{-1} \text{ h}^{-1} were administered continuously throughout surgery. After bifrontal craniotomy, the neurosurgeon approached the tumour inter-hemispherically and performed subtotal tumour resection. The 10-h surgery was completed without complication and with a blood loss of only 300 ml. Examination of the surgical specimens revealed haemorrhagic and necrotic pituitary tissue. After surgery, the patient was haemodynamically stable and was transferred to an intensive care unit, where he was ventilated mechanically and sedated with midazolam. When suctioning was performed through the tracheal tube, ventricular fibrillation suddenly occurred. The patient was cardioverted, additional lidocaine (1 \text{ mg kg}^{-1}) was administered and sinus rhythm returned. However, premature ventricle complexes occurred frequently and systolic blood pressure was below 90 mm Hg. An intra-aortic balloon pump was placed through the left femoral artery to assist coronary artery blood flow. We did not use any anticoagulant to prevent bleeding from the surgical site. We delayed cardiac surgery for about 20 h after neurosurgery until oozing from the subcutaneous drainage site stopped. The CABG procedure was then undertaken. Upon arrival in the operating room, a pulmonary artery catheter was placed via the internal jugular vein. Anaesthesia was induced with midazolam 2 mg and fentanyl 100 \text{ \mu g}, then maintained with propofol 2 \text{ mg kg}^{-1} \text{ h}^{-1} and fentanyl 2 \text{ \mu g kg}^{-1} \text{ h}^{-1} intravenously. Muscle relaxation was obtained with pancuronium 6 mg and maintained with intermittent boluses. The patient continued to receive dopamine, nitroglycerine, diltiazem and lidocaine at the same doses until starting CPB. The duration of aortic clamping was 60 min and that of CPB was 100 min. The patient was weaned from CPB uneventfully, but balloon pump was continued. Mechanical ventilatory support was continued in the intensive care unit. On the second day postoperatively, CT brain revealed no haemorrhage. The patient regained airway reflexes and spontaneous ventilation. The trachea was extubated. A week after neurosurgery, diabetes insipidus developed and was treated with vasopressin 10 \text{ u. day}^{-1} intravenously. For about a month after the CABG procedure, his conscious state fluctuated between somnolence and delirium. When delirious, the patient was sedated by a midazolam 1–2 mg h\textsuperscript{-1} infusion.
Within a month, he recovered and had a GCS of 14. Visual field deficit and ophthalmoplegia gradually improved.

**Discussion**

Pituitary apoplexy is due to an intra-cranial tumour, into which haemorrhage produces an expanding mass, causing visual and neurological impairment. The patient requires neurosurgical treatment immediately. Severe unstable angina with the necessity for an urgent CABG procedure coexisted with the pituitary expansion in this patient. It is often suggested that non-cardiac surgery and CABG should be performed simultaneously. In this case, however, anticoagulant therapy with CPB would have induced intra-cranial haemorrhage and the neurological prognosis could have been worse. Confronted with such conflicting situations, we had a dilemma regarding the best surgical management.

In patients with severe angiina pectoris and a history of myocardial infarction, the risk of perioperative cardiac complications including death is increased. The American Heart Association reported that the frequency of myocardial infarction and death during simultaneous carotid endarterectomy and a CABG procedure is lower than in a staged operation. Allie and colleagues recommended that a rapid staged procedure (an interval between endarterectomy and CABG procedure of less than 24 h) with intra-aortic balloon pumping was safe and effective in the very high risk patient population with coronary artery disease. But the optimal strategies for the management of patients with carotid and coronary artery disease have not been established. In this patient, the neurological prognosis depended on immediate neurosurgical decompression. We, therefore, performed neurosurgery before the CABG procedure as a staged operation.

The point at which intra-cranial haemostasis is achieved after neurosurgery remains uncertain. Wijdicks reported that restarting anticoagulation therapy in a patient with mechanical heart valves is safe 1–2 weeks after intracranial haemorrhage. However, in this patient, we could not delay the CABG procedure because the patient had ventricular fibrillation and required mechanical support using intra-aortic balloon pumping. When the oozing from the neurosurgical wound stopped, we immediately performed the cardiac surgery using anticoagulant therapy 20 h after neurosurgery.

Endocrine failure is a problem in a staged operation. If acute adrenal insufficiency, hypothyroidism and diabetes insipidus occur during the perioperative period of cardiac surgery, it might worsen the angiina pectoris. For example, administration of vasopressin induces coronary vasoconstriction. It is, therefore, important to perform cardiac surgery before any uncontrollable endocrine failure occurs. This is one of the reasons why we performed the neurosurgery before the cardiac surgery.

In conclusion, we managed a patient with severe angiina pectoris and pituitary apoplexy. We have described a two staged operation with sequential management of intra-cranial haemorrhage and open-heart surgery requiring anticoagulant therapy. Our case illustrates the importance of coronary care during the time between the two operators.

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