Hypoxic. The original intrapulmonary shunt secondary to one-lung ventilation with 100% oxygen ensures adequate ipsilateral lung without the need for carbon dioxide insufflation. Ventilation of the contralateral lung while allowing collapse of the lung depends on double-lumen intubation which provides selective ventilation during thoracoscopy to endobronchial anaesthesia because of its simplicity, decreased cost, and improved oxygenation. To minimize the risk of inadvertent tension pneumothorax secondary to the continuous insufflation of carbon dioxide during thoracoscopic anesthesia, a pressure-limited, variable flow of carbon dioxide is recommended.1,2

Experimental work in swine has shown that positive pressure insufflation during thoracoscopic surgery results in significant haemodynamic compromise despite the use of selective lung ventilation: cardiac index, mean arterial pressure, and left ventricular stroke work index decreased, whereas pulmonary artery and central venous pressure increased at an insufflation pressure of 5 mm Hg or greater.3 Thus, carbon dioxide insufflation into the closed chest cavity to a pressure as low as 5 mm Hg may create a physiological response very similar to that of a unilateral tension pneumothorax,4 with a consequent haemodynamic instability secondary to decreased venous return and/or mediastinal shift.

Our anaesthetic technique during video-assisted thoracoscopic surgery depends on double-lumen intubation which provides selective ventilation of the contralateral lung while allowing collapse of the ipsilateral lung without the need for carbon dioxide insufflation. Selective lung ventilation with 100% oxygen ensures adequate oxygenation throughout the procedure, as checked by continuous pulse oximetry. HPV is most active when 30–70% of the lung is hypoxic.2 The original intrapulmonary shunt secondary to one-lung collapse is about 40–50%, and is decreased to 20–30% by active HPV,5 as well as by gravity, and collapse of the non-ventilated lung. Partial collapse of the lung on the thoracoscoped side occurs when air enters the pleural cavity. To augment collapse, the lumen of the double-lumen tube on the thoracoscoped side is normally left open to room air, while suction is intermittently applied. Since we have used selective contralateral lung ventilation, and avoided carbon dioxide insufflation into the ipsilateral pleural space, we have encountered no serious haemodynamic complications secondary to inadvertent tension pneumothorax.

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Midazolam given as an intranasal spray in children

Editor.—We read with interest the article about the pharmacokinetics of midazolam given as an intranasal spray by Björkman, Rigemar and Istdal.1 The authors described the intranasal application of midazolam spray as a practicable method with high bioavailability in adult surgical patients. As the authors noted, intranasal spray application of midazolam could also be useful in children because of the described pharmacokinetic and pharmacodynamic advantages.

In particular, ambulatory anaesthesia in children requires rapid and effective premedication. Midazolam is well known for its anxiolytic, euphoric, amnesic and sedative qualities. Although various routes of administration in children have been approved for this drug, none is without problems.

After rectal application of midazolam 0.5 mg kg−1, an effect typically appears within 15 min. However, 20% of children refuse this approach.2 Sublingual administration of midazolam produces a faster effect, but requires a flavour corrigent because of its bitter taste.3 Even then, acceptance is poor; the liquid is often expectorated,4 and many children reject a second application. Additionally, the incidence of postoperative nausea and vomiting is highest after oral midazolam.

In the search for an alternative, it has been shown that nasal bolus administration of midazolam 0.2 mg kg−1 is an effective way to achieve rapid onset of sedation immediately before induction of anaesthesia in children.5,6 A higher dosage of midazolam 0.3 mg kg−1 did not increase the effects.7 Intranasal midazolam achieves good conditions for the induction of anaesthesia with a maximum effect attained 5–10 min sooner than with rectal or oral application. The euphoric effect is more potent, and the sedative component is less than with rectal midazolam.8 The main disadvantage of intranasal midazolam administration is a burning discomfort in the nasal mucosa and—similar to oral application—a bitter taste when midazolam runs into the pharynx.9 In all these articles, nasal midazolam was administered as a bolus dose.

Because of the reported side effects, we introduced undiluted midazolam (15 mg in 3 ml, Dormicum, Hoffmann-La Roche, Grenzach-Wyhlen, Germany) via a nasal spray. Our pump spray applicator (Calmar-Albert dispensing systems, Hemer, Germany), normally used for rhinorrhoea, produces a fine, equally distributed aerosol of midazolam. Each application delivers a standard 0.12 ml, which corresponds to midazolam 0.6 mg. A new application set is used for each patient. The glass bottles and aerosol applicators can be sterilized after use.

For 1 year, children in our outpatient department aged 1–10 years have received midazolam 0.2 mg kg−1 via pump spray for premedication. We noticed a rapid onset of mild sedation, and euphoria 5–10 min after administration, which allowed a calm separation of the child from the parent. For safety reasons, pulse oximetry was always used after nasal midazolam administration. However, we did not observe any impairment of vital functions, as has been reported by other authors.10 In our preliminary observations, children tolerated the spray application much better than the bolus administration. We observed fewer complaints of bitter taste and mucosal irritation. This leads to better acceptance and enhances the child’s and the parent’s trust in the anaesthetic procedure. On the basis of our experience, we recommend this new form of nasal midazolam administration via nasal spray.

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Editor,—We thank Drs Thum, Heine, Hollenhorst and Leuwer for supporting our suggestion with some clinical data. It should be noted, that judging by our results, midazolam 0.2 mg kg⁻¹ given by spray is a higher effective dose than 0.2 mg kg⁻¹ given as drops or bolus. But a general recommendation to use midazolam by intranasal spray in children should be based on a full report of the effect profile. We look forward to seeing these data published.

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The CardioPump: CEPOD guidelines and validation of new techniques

Editor,—Modern methods of cardiopulmonary resuscitation were introduced in 1960 after publication of the classic article by Kouwenhoven, Jude and Knickerbocker on closed-chest cardiac massage. The CardioPump has been developed for resuscitation during cardiac arrest with the perceived advantage of increasing the forward flow of blood. We report a case in which the CardioPump was used, and a complication that may be associated with it and which was discovered only at postmortem examination. A 60-year-old brickyocker with a basivisceral fracture of the right hip underwent general anaesthesia for a right-sided AO dynamic hip screw. He was admitted to hospital 7 days before operation. Anaesthesia was induced with thiopentone 400 mg and fentanyl 75 μg. A 3 in 1 block was performed with 30 ml bupivacaine 0.375% using a nerve stimulator. The patient was subsequently given atracurium 50 mg and intubated with a size 9.0 oral tracheal tube. Anaesthesia was maintained with isoflurane in oxygen and nitrous oxide, using intermittent positive pressure ventilation. Following manipulation of the injured leg, desaturation from an SpO₂ of 96% to 90% was noted, together with a fall in end-tidal PCO₂. The patient developed a tachycardia that proceeded to a electromechanical dissociation (EMD) cardiac arrest. Cardiopulmonary resuscitation (CPR) was started using the European Resuscitation Council Guidelines for an EMD arrest. The Ambu CardioPump was used to aid resuscitation attempts, which were continued for 45 min with no effect. Resuscitation was then abandoned. At postmortem, a pulmonary embolism was found to be the cause of the cardiac arrest. Other findings included a haemoperitoneum associated with a 4 × 4 cm tear in the mesentery of the small bowel adjacent to the third part of the duodenum. Associated with this was a small traumatic laceration on the superior surface of the liver, adjacent to the insertion of the falciform ligament. Three fractured ribs (four, five and six) on the left side of the chest consistent with attempted resuscitation were also noted. The CardioPump device consists of a horizontal handle, a piston and a suction cup. The radius of the suction cup is 3.8–5.1 cm in standard CPR. The decompression phase produces a negative pressure of approximately 10 kg. The compression and decompression phases are performed in a ratio of 1:1.1

Laceration of the liver and tearing of the mesentery of the small bowel adjacent to the third part of the duodenum can arise from standard CPR, especially if it is carried out incorrectly by the mal-positioning of sternal compression. However, a new device was used in this case and its contribution to this complication is not known. The intra-abdominal trauma may be related to the higher pressures consistently produced in the compression phase with this device compared with standard CPR. We postulate an alternative theory. The active decompression phase may have contributed to the trauma as a considerable negative pressure is generated, and compared with standard CPR there is a greater change in intra-thoracic and hence intra-abdominal pressure. We would also suggest that if this device were to be incorrectly positioned, that is below the sternal midpoint, the potential for intra-abdominal trauma would be increased.

The recent postgraduate resuscitation issue of the British Journal of Anaesthesia raises the question of the effectiveness of new resuscitation devices, asking if they are more beneficial than traditional CPR and if they carry added risks. Only when these questions are answered can definitive recommendations be made about the use of such devices.

There is little information available about complications associated with active compression–decompression CPR. One of the many reasons for carrying out postmortem examinations is to monitor the effectiveness and side effects of new medical and surgical therapies. A joint recommendation from the Royal Colleges of Surgeons, Physicians and Pathologists published in 1991 stated that there should be an increase in hospital postmortem examinations, in part to gather information on new treatments. This point has been reiterated in the National Confidential Enquiry into Perioperative Deaths.1 We feel that nonsurvivors of CPR using these new methods of resuscitation should undergo postmortem examination to look for complications before such techniques are accepted as standard practice.

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6. The report of the National Confidential Enquiry into Perioperative Deaths. 1993/94.

Submandibular duct stone and difficult tracheal intubation

Editor,—A 54-year-old male presented for extracorporeal shock wave lithotripsy. Apart from renal calculi he was in good health (weight 64 kg, height 166 cm, BMI 23.23). He had no symptoms relating to the mouth, tongue or saliva. He had received two uneventful general anaesthetics in the preceding 3 weeks, neither of which needed tracheal intubation. There was no abnormality detected on physical examination: oral examination revealed a Mallampati class II intubation score.

Anaesthesia was induced with propofol 150 mg, fentanyl 100 μg and atracurium 50 mg. His lungs were ventilated easily with oxygen 33%, nitrous oxide 66% and sevoflurane 2%. On laryngoscopy, 3 min later, the tongue was found to be slightly deviated to the left and only the tip of the epiglottis was visualized. Manual ventilation of the lungs was resumed. After two unsuccessful...
attempts at intubation had been made, a size 4 laryngeal mask airway (LMA) was inserted. Surgery proceeded uneventfully and the LMA was removed after reversal of the neuromuscular blocking drug. Detailed examination of the mouth revealed a swelling at the base of the tongue on the right side. Postoperatively, the patient was referred to an otorhinolaryngologist, who suspected submandibular gland swelling because of stone formation in the submandibular duct. Subsequently, sialolithiasis of the submandibular duct was confirmed by radiography. Two, weeks later, the stone was removed under general anaesthesia; tracheal intubation was achieved using the McCoy laryngoscope and a gum elastic bougie.

Difficult laryngoscopy and intubation because of masses under the tongue such as a lingual thyroid, salivary distension or sublingual haematoma have been reported. Swelling of the tongue following general anaesthesia can also occur in association with angioedema. It is unlikely that the swelling under the tongue in this patient was related to the recent episodes of general anaesthesia; both were atraumatic and neither required intubation. There was no evidence related to the recent episodes of general anaesthesia; both were atraumatic and neither required intubation. There was no evidence of sialadenitis or sublingual haematoma and angioedema usually causes generalized swelling of the tongue that becomes apparent within a few hours.

This case serves as a reminder that the anaesthetist should always make a thorough preoperative intra-oral inspection, especially where preoperative Mallampati is not class I. This case serves as a reminder that the anaesthetist should always make a thorough preoperative intra-oral inspection, especially where preoperative Mallampati is not class I.

Local analgesia in early and late stages of labour

Editor,—As an obstetrician, I read with interest the article by Capogna and colleagues. The authors measured the minimum local analgesic concentration (MLAC) of bupivacaine in both the early and late stages of labour. They conclude that advancing labour requires an increased concentration of extradural bupivacaine for pain relief. Regional anaesthesia is a direct influence over obstetric practice in terms of operative delivery and progress of labour, and research into optimal intrapartum analgesia is always of interest. Research like this is lacking.

From an obstetric point of view, there are several factors of which anaesthetists should be aware when considering this study. Although the two groups were well matched with respect to maternal characteristics, it would have been helpful to have had more intrapartum details. Labours induced with prostaglandins are longer than spontaneous labours, and an induction of labour with prostaglandins or oxytocin can cause uterine activity in the absence of progesterone. Did the authors take into account the lengths of labour included in this sample? We are told that 20 out of 30 women had oxytocin. Was the oxytocin given before the visual analogue profile scores (VAPS)? It would be wise to consider these women as a separate group, as the use of oxytocin suggests that labour was prolonged because of incoordinate uterine activity, cephalopelvic disproportion or induction. Either way, labours with oxytocin are abnormal and should be considered separately from spontaneous labours, which on average will be shorter in duration and hence have different, possibly lower, VAPS.

Finally, I disagree with combining data from nulliparous and multiparous women, as these two groups have very different intrapartum profiles. Nulliparous women are prone to incoordinate uterine activity at any stage of labour and require oxytocin to correct this. As a result labour can be longer. This is rarely the case with multiparous women.

Cervical dilatation is only one of many important intrapartum findings used to assess the stage of labour. Not only the degree of cervical dilatation, but also the rate of dilatation and foetal head descent should be considered, together with the women’s parity, the quality and frequency of the uterine contractions and the size of the foetus.

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Native text:

**Local analgesia in early and late stages of labour**

**Editor**—As an obstetrician, I read with interest the article by Capogna and colleagues. The authors measured the minimum local analgesic concentration (MLAC) of bupivacaine in both the early and late stages of labour. They conclude that advancing labour requires an increased concentration of extradural bupivacaine for pain relief. Regional anaesthesia is a direct influence over obstetric practice in terms of operative delivery and progress of labour, and research into optimal intrapartum analgesia is always of interest. Research like this is lacking.

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Cervical dilatation is only one of many important intrapartum findings used to assess the stage of labour. Not only the degree of cervical dilatation, but also the rate of dilatation and foetal head descent should be considered, together with the women’s parity, the quality and frequency of the uterine contractions and the size of the foetus.

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A guide for tube exchange using a fibrescope and the plastic sheath of a guidewire in small children

Editor,—When managing a difficult airway, occasionally there is a need to replace a tracheal tube. For example, one may have to change a tracheal tube for a larger one after intubation through a laryngeal mask airway (LMA). But, in small children, tube exchange techniques are not readily available.

As a guide for tracheal tube exchange, we used the plastic sheath of a guidewire within a central venous line set (B. Brown Medical, Bethlehem, PA). The sheath is a hollow plastic tube and has an internal diameter of about 3 mm and is about 65 cm long. It is supplied in a circular form (fig. 1A) and can be uncoiled in a half-circle shape (fig. 1B) with its plastic character. One may use any kind of plastic sheath that is a little malleable, has sufficient length, an inner diameter large enough to accommodate a fibrescope, and an outer diameter small enough to pass through a tracheal tube. The length of the sheath should be adjusted to only a few cm shorter than the working length of a fibrescope (the length of an Olympus LF-P is 60 cm). The square-cut leading edge of the sheath may be blocked half way on the curved inner surface of the tracheal tube, so we suggest making a bevel of about 45° on the convex side of its tip. The length of the tracheal tube to be changed should be pre-marked on the shaft of the sheath.

We have exchanged a tracheal tube for a larger one after intubation through an LMA in two small children. One was a 4-year-old, 16-kg boy with Treacher–Collins syndrome, in whom a tracheal tube of 4.5 mm internal diameter was exchanged for a 5.5 mm tube. The other was a 5-year-old, 7.7-kg boy with Goldenhar’s syndrome, in whom a 4 mm tracheal tube was exchanged for a 4.5 mm tube. To prevent potential mucosal damage from the sharp tip of the sheath, we inserted an ultrathin fibrescope (Olympus LF-P, outer diameter 2.2 mm) through the sheath beforehand (fig. 1B). We advanced the fibrescope into the trachea ahead of the sheath, with its proximal end abutting the fibrescope handle. We maintained the fibrescope in situ to confirm its intratracheal location while removing the tracheal tube and LMA from the mouth. While holding the sheath, we removed the fibrescope, off-loaded the tracheal tube and LMA, reloaded a larger tracheal tube over the sheath, reinserted the fibrescope into the sheath, and finally railroaded the larger one over the sheath to the desired depth. This method may prevent inadvertent extubation by continuously confirming the intratracheal location during the critical period of removing or inserting a tracheal tube over a tube exchanger, and can simultaneously adjust its depth. Similar techniques have been reported, but these were used in adults. An alternative technique is also available: one can cut away the former tracheal tube as described by Gatell and colleagues. Then it is not necessary to pull out the fibrescope during removal of the fibrescope from damage by a pair of scissors or scalpel.

The presence of a fibrescope within the sheath can stabilize and strengthen it and may reduce potential mucosal injury by minimizing contact of the tip with tracheal mucosa. Additionally, use of a fibrescope can prevent tracheobronchial rupture, which has been reported with the blind use of a tube exchanger.

The sheath can be used only for 4.0 mm tracheal tubes or larger. It also can be connected to an oxygen source or a capnograph through an appropriate adaptor. For example, a 15 mm connector for a 2.0 mm tracheal tube can fit snugly into the sheath, or it may be wedged into the distal end of a 3.5 mm tracheal tube cut at the shaft (fig. 1C).

It is preferable if a tube exchanger, which can accommodate a fibrescope and has a blunt tip, is made of clear material to allow surrounding tissues to be seen. Additionally, if it has an inflatable balloon at the tip, just like a cuffed tracheal tube, it may reduce mucosal injury and anchor the tube exchanger at the subglottic area while the tube is being exchanged in young children.

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A guide for tube exchange using a fibrescope

Figure 1  (A) a guidewire and sheath in an original form, supplied as a central venous line set (B. Brown Medical, Bethlehem, PA). (B) The modified guidewire sheath, shortened about 6 cm, through which a fibrescope (Olympus LF-P) is inserted. (C) The guidewire sheath is wedged into the distal end (arrow) of the shaft of a tracheal tube of 3.5 mm internal diameter cut at the middle.


Sevoflurane for difficult tracheal intubation

Editor,—We read with interest the report and subsequent correspondence on the use of sevoflurane in patients with difficult
airways. These reports describe the use of a stepwise inhalation induction technique. While the use of such a cautious approach would seem prudent in patients with anticipated airway problems, the evidence suggests that this technique is associated with a high incidence of cough. A stepwise increase of inspired sevoflurane concentration is not only slower but also prolongs the second stage, resulting in an unacceptably high level of excitement and coughing. We would suggest that the technique of choice in patients with a difficult airway is to breathe sevoflurane 8% from the start, as this has a lower incidence of cough and excitement (2% and 10% respectively) compared with a stepwise technique (12% and 31% respectively). Apnoea is relatively uncommon with this technique (16%) and typically short-lived, the average time from loss of consciousness until resumption of spontaneous breathing being 10 s. As the addition of nitrous oxide has a small effect on the speed or quality of inhalation induction with sevoflurane, we would recommend that in patients with difficult airways, sevoflurane is administered in oxygen 100%.

Sevoflurane alone can be used to facilitate tracheal intubation. We have found intubation conditions to be satisfactory at about 4 min in unpremedicated adults and at about 2.5 min in children. An alternative approach to facilitate tracheal intubation after a sevoflurane induction is to give a neuromuscular blocking agent once the vocal cords have been clearly visualized.

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Editor—The authors refer to two papers1,2 which, they suggest, recommend sevoflurane 8% in oxygen as the technique of choice in patients with a difficult airway. Yurino and Kimura report a 31.25% incidence of movements during inhalational induction with stepwise increments of sevoflurane 0.5% every third to fourth breath, compared with an incidence of 6.25% in a vital capacity induction of sevoflurane 4.5% in nitrous oxide–oxygen. The incidence of coughing was 6.25% in the vital capacity induction compared with 12.5% in the stepwise technique. However, they suggest that their stepwise technique is in fact safer, with none in a propofol control group. Patient comfort is surely the key. A slow, gradual induction using sevoflurane 8% in nitrous oxide–oxygen to i.v. propofol (n = 102), and reported purposeful movement in three patients in the propofol group compared with five in the sevoflurane group.

Although the incidence of coughing was only 2% during inhalational induction, the same authors also report a 16% incidence of coughing during the transition from induction to maintenance of anaesthesia.

I have not found coughing and excitement to be a major problem in an ongoing audit of a predominantly Asian group of patients with head and neck tumours. A 16% incidence of coughing in the use of a sevoflurane 8% induction cannot be ignored. Indeed, in one patient with tonsillar carcinoma who underwent sevoflurane 8% vital capacity induction in our hospital, the duration of apnoea would have led to hypoxia if assisted ventilation had been impossible. Although the use of sevoflurane 8% results in faster onset of anaesthesia, the advantage of gas induction is not speed but safety. A stepwise induction enables the experienced anaesthetist to assess airway patency and the ability to assist ventilation. The low blood-gas solubility of the agent allows the option to abort before an unnecessarily deep plane of anaesthesia is reached.

In conclusion, before inhalational induction with sevoflurane 8% can be favoured over a stepwise technique, the outcome from the management of actually difficult cases needs to be reported. P.C. IP-YAM
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Editor—The figures quoted for cough and excitement with a high-dose technique of inhalation induction with sevoflurane are 2% and 10% respectively. However, other research has reported complications rates of 25% for coughing1 and 38% for excitement4 when using sevoflurane 8% from the outset. These are higher than the quoted rates for a step wise approach (12% and 31% respectively).2 Complication rates seem to be lower if a vital capacity technique is used, but such a technique would not be easy to use in someone with a difficult airway.

Coughing may also be linked to how quickly a concentration of sevoflurane 8% is reached. It is the theory that if the anaesthetic circuit is pre-primed with sevoflurane 8%, this leads to an abrupt increase in inspired vapour concentration when the facemask is applied. This is irritating and leads to coughing. The alternative — pre-oxygenating the patient and then adding the sevoflurane 8% to the flow — allows dilution of the agent and a more gradual increase in inspired vapour concentration. This is better tolerated by the patient (and is more akin to the stepwise approach).

More serious complications, such as laryngospasm, have been reported in patients in whom anaesthesia was induced with sevoflurane 8%.5 Severe hypotension has also been noted to occur in children.6 There are several reports of profound bradycardia in children during induction of anaesthesia with sevoflurane using the high-concentration approach. These are reasons for adopting a more cautious approach, especially with a difficult airway.

Finally, 29% of patients in whom anaesthesia was induced with sevoflurane recalled an unpleasant smell afterwards.7 Half of these described the whole induction experience as unpleasant, compared with none in a propofol control group. Patient comfort is surely the most compelling reason for an incremental approach; the stepwise approach allows patients to become familiar with the smell in a gradual, controlled manner rather than being suddenly exposed to the full pungency of the vapour. They remain calm and confident in their anaesthetist. There is still rapid loss of consciousness, because of the low blood gas solubility coefficient of sevoflurane, following which rapid progression to maximum concentration minimizes the excitement phase.

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Early vs late tracheal extubation after CABG surgery

Editor,—The recent paper by Berry and colleagues highlights the important issue of the safety of early tracheal extubation after coronary artery bypass graft (CABG) surgery. A crucial question is whether the potentially adverse effects of tracheal extubation on myocardial oxygen balance are significant when the heart is recovering from the ischaemic insult associated with proximal aortic crossclamping. Early extubation after CABG surgery is safe only if it does not exacerbate perioperative cardiac muscle injury.

The regulatory cardiac contractile protein, cardiac troponin I (cTnI), is present exclusively in cardiac muscle tissue and is an accurate biochemical marker of perioperative cardiac muscle injury. In a recent study comparing Holler continuous EGG ST segment analysis with serum cTnI concentrations during the postoperative period after CABG surgery, we found that early postoperative electrocardiographic myocardial ischaemia was associated with elevated postoperative serum cTnI concentrations. Data from three out of 30 patients in this study indicated that electrocardiographic myocardial ischaemia within the first 8 h after the end of cardiopulmonary bypass was precipitated or exacerbated by withdrawing sedation in preparation for tracheal extubation. Although for these three patients the electrocardiographic myocardial ischaemia was immediately resolved by reseating and mechanical ventilation, postoperative serum cTnI concentrations were elevated. Data from the study by Berry and colleagues show that a small but significant proportion of patients (10%) who meet standard early extubation criteria have a high myocardial ischaemic burden (> 15 min h−1) during the early postoperative period. As the incidence of myocardial ischaemia falls exponentially with time during the early postoperative period, the earlier that extubation is performed, the greater is the chance that it will occur simultaneously with electrocardiographic myocardial ischaemia.

Therefore for patients who fulfil the criteria for early extubation after CABG surgery, during weaning from postoperative sedation, the continuous EGG ST segment monitor (now incorporated into most ICU or operating theatre monitors) should be closely observed. If myocardial ischaemia is present, extubation should be postponed until these EGG ST segment abnormalities have been resolved. Although in the study by Berry and colleagues 19 out of 359 episodes of electrocardiographic myocardial ischaemia occurred during the extubation period, it is not clear first, in how many patients this occurred or second, whether extubation precipitated or exacerbated myocardial ischaemia.

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Editor,—Thank you for the opportunity to reply to the comments of Dr Tupper-Carey. As he suggests, it is important to assess the impact of any adverse haemodynamic changes associated with early extubation on the total ischaemic load experienced. If the “extubation period” is defined as 30 min either side of tracheal tube removal, then in our study, six patients in the early extubation group and two in the late extubation group experienced ischaemic episodes in this period. It is clear from previous studies that the incidence of electrocardiographic ischaemia is greatest in the 8 h after surgery and declines with time. Our early extubation group were allowed to wake as soon as they returned from the operating theatre and the average time of extubation was almost 11 h earlier than in the late group. Nevertheless, we found no difference in the total incidence or severity of postoperative ischaemia. Review of our haemodynamic data demonstrated that the major haemodynamic changes were seen within the first 8 h during the operation, therefore the period of mechanical ventilation prior to the time of extubation is unlikely to influence the frequency or severity.

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Venous air embolism and PEEP: a clinical research blind-spot

Editor,—The recent clinical study by Giebler and colleagues on 89 patients undergoing neurosurgery in the sitting position reported that the use of 10 cm H2O positive end-expiratory pressure (PEEP) did not reduce their overall 26% incidence of venous air embolism (VAE). While not necessarily disagreeing with their conclusion that the use of PEEP “cannot be recommended”, I believe they misinterpreted one of the findings of Meyer and colleagues, who reported a zero incidence of VAE in 30 children being managed with PEEP and a MAST suit, compared with 26% in a control group. Though the “filling pressures” in both the Meyer and the Giebler studies “did not differ”, it should be noted that, in the Meyer study, the reported right atrial pressures would have been sufficient to distend the venous system of many of the sitting children to a level approximating the level of the jugular bulb. The protective mechanism of a MAST suit and PEEP could not be translated to adult patients, as it is not clinically feasible to maintain the venous pressure at equivalent high levels.

Although maintaining an elevated venous pressure will not prevent VAE in adult patients, it will in theory serve, to promote the temporary collection of embolized air in the superior vena cava...
and even the internal jugular veins,1,2 and to reduce the risk of a massive life-threatening embolism.3,4 I believe that greater emphasis should be placed on the need to maintain a raised central venous pressure, whether it be by MAST suit application, transfusion or a combination of these.

In our hospital, neurosurgery with the patient in the sitting position has not been performed for many years. For clarification of several issues, therefore, I seek advice from centres where such surgery is performed. Does maintaining the central venous pressure at, for example, 10 mm Hg using a transducer zeroed at the level of the upper part of the right atrium, result in embolized air collecting or ‘pooling’ upwards in the superior vena cava, either temporarily or in part, as it does in a model1 and in sheep?5 Second, does the entry of even small volumes of air in create in humans, as it does in sheep, a readily identifiable pressure differential between two different levels in the vertically positioned superior vena cava, so allowing a diagnosis of VAE earlier than, or at the same time as, it is identified by Doppler?2

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Editor,—We appreciate Dr Pfitzner’s letter in response to our article1 and the interest in our clinical research. Dr Pfitzner feels that we may have misinterpreted the findings of Meyer and colleagues who reported a zero incidence of venous air embolism (VAE) in children managed with both PEEP and a MAST suit, compared with a 26% incidence in a control group. The distance between the right heart and jugular bulb or brain surface is much smaller in young children than in adults. As Meyer and colleagues reported an average central venous pressure of 11 mm Hg in children in the sitting position, this pressure may have been sufficient to minimize airway VAE in children, and is unlikely to be sufficiently high in adults, as indicated by the results of our study.1 Accordingly, children are like neither adults nor sheep.

We rarely use anti-gravity suits, because the increase in filling pressures has been reported as only transient. We care for many adults with heart disease and try to minimize cardiac volume overload during surgery. Whether PEEP at levels markedly in excess of 10 cm H₂O may be effective in decreasing the incidence of VAE and yet not impair cardiovascular performance is beyond the scope of our investigation. This is unlikely, however, as although PEEP increases venous pressure relative to atmosphere, it also decreases cardiac transmural filling pressures and thus cardiac filling and output.6

Whether an increased venous pressure will, in theory, serve to promote the temporary collection of embolized air in the superior vena cava and even in the internal jugular veins, as speculated by Pfitzner and colleagues,1 and reduce the risk of massive life-threatening embolism, is certainly interesting. To our knowledge, clinical data on this topic are not available. However, Pfitzner himself could not demonstrate clear evidence that blood volume or PEEP influenced the yield of air at aspiration.

Furthermore, from a clinical viewpoint, this hypothesis can hardly be tested in a quantitative fashion that a Doppler probe is placed over the heart but the superior vena cava cannot reliably be visualized by transoesophageal echocardiography. Accordingly, data collection from sitting sheep may be the only way to address Dr Pfitzner’s hypothesis, but may be of questionable clinical relevance!

Therefore, we cannot provide an answer to Dr Pfitzner’s question, as to whether a certain central venous pressure results in “trapping” of embolized air in the superior vena cava or, of even greater interest, in a step pressure gradient along the superior vena cava in the vertical position, allowing a potential diagnosis of VAE. The answer to this question may also depend on the presence and competence of venous valves in the jugular venous system, which in certain individuals may or may not be functional. Indeed, differences in the venous valve system may benefit sheep but not human adults during VAE.

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Walking extradurals

Editor—Thank you for the opportunity to reply to the letter published in the May issue of the British Journal of Anaesthesia. We are sorry that our title and editorial confused the authors. The inverted commas around “walking extradural” were meant to convey the fact that although such a regional blockade is commonly called an epidural it often involves a spinal component.

The use of combined spinal epidural analgesia in labour is controversial. Several authors from various centres including one from Queen Charlotte’s Hospital itself have questioned its routine use in labour.

Our editorial attempted to explore the possibility of ambulation in labour, its safety, the techniques available and possible effects on the mode of delivery. Few studies involving low dose regional blockade in labour have described accurately either the ability to walk or actual rates of ambulation. Low rates of ambulation and the use of higher concentrations of local anaesthetic solution in late labour may explain the inability of some studies to demonstrate any difference in obstetric outcome. We accept that the lower the total dose of local anaesthetic administered into the epidural space, the lower will be the resultant motor blockade. A small dose of local anaesthetic administered intraoperatively with combined spinal epidural analgesia in labour has a very rapid onset for the anatomical reasons given. However, there is little evidence that this influences dose requirement and subsequent motor block several hours after the initial dose.

We accept that the combined spinal-extradural technique is popular and useful but we hoped that our editorial gave a balanced, cautious assessment of its potential in obstetrics.

We discussed the first use of the epidural catheter after a combined spinal-extradural. In this situation, the first dose is the test dose and should be given by someone able to identify and treat the consequences of intravenous and intrathecal placement of the catheter. Our use of the term “appropriately trained person” was intentional and not a substitution of the word “anaesthetist”. It is entirely possible that a midwife could be trained to give this first dose provided that he or she is also trained to recognise the consequences of inadvertent intrathecal administration and that an anaesthetist is immediately available. The first dose should not be
given by a patient-controlled epidural analgesia device in the presence of an appropriately qualified individual.

We did not advocate the use of a test dose of high concentration lignocaine or bupivacaine. Indeed, in our discussion on proprioception, we suggested that this was undesirable. We do not recommend flushing the epidural catheter immediately after placing it in the epidural space.

We did not say that there was an excess incidence of meningitis after combined spinal-extradural but stated that an excess would limit the use of this technique. Penetrating the dura represents an even greater compromise of host defences than merely penetrating the ligamentum flavum. We do believe it is important to pay scrupulous attention to aseptic technique and the avoidance of chemical contamination when giving intrathecal drugs. We accept that the use of the phrase “provides evidence for the inherent safety of the technique” was open to misinterpretation.

We wholeheartedly agree that the provision of safe, efficacious analgesia in labour should be a health care priority. We believe in a woman-centred approach based on informed choices of analgesia. Further work is necessary to establish the benefits of ambulation in labour and to justify the routine use of additional interventions such as dural puncture; recently, disadvantages of subarachnoid opioid analgesia such as delayed gastric emptying and an association with transient uterine hypertension and resultant fetal distress, have been reported.

The procedure took 15 min and can be recommended as a safe and simple method of placing a nasogastric tube in a difficult pose, although it can give useful insights into the way our physiology is without explanation, and possibly without purpose. Just because something is interesting — as cardioventilatory coupling is — does not mean that it has a purpose, although it can give useful insights into the way our physiology works. There is nothing wrong with continuing to make observations on coupling, but I should prefer to refer to introductions, “It is not known if coupling has any physiological role...” rather than “Although the physiological role is unknown...”

Change of nasogastric tube via Seldinger technique

Editor,—A 14-year-old patient was admitted to our intensive care unit with multiple facial fractures and a head injury. During surgical repair of the fractures, a tracheostomy was performed and delayed gastric emptying and resultant fetal distress, have been reported. The CT scan was examined and the faciomaxillary tube was gently withdrawn in an attempt to straighten it. This was impossible to open the patient’s mouth. The kinked nasogastric cal repair of the fractures, a tracheostomy was performed and dural puncture headache. British Journal of Anaesthesia 1998; 80: 123–124.


Observation and purpose: are ventilatory periodicities fine tuning or accidents of physiology?

Editor,—The recent paper from Galletly and Larsen reports an expansion of their previous work, in which they provided an explanation for one type of ventilatory periodicity,7 cardioventilatory coupling. This causes the breathing rate to be entrained by the cardiac cycle: each breath tends to last a whole number of cardiac cycles and, in effect, the start of inspiration waits for the next cardiac cycle to begin. Their explanation was particularly welcome for being so simple, unlike some of the previous suggestions.1 It is also welcome for acknowledging a 70-year-old observation by Coleman, of which I, probably like many others with a respiratory interest, was completely unaware. I am less at ease with the subtext running through their papers. In their most recent introduction1 they write: “Although the physiological role of [cardioventilatory coupling] is not known...”, implying that it does serve some physiological function, and they go on to speculate what this might be. At the end of their first paper,2 they wrote that the relevance of coupling was unknown, but that it had implications for the interpretation of studies of heart rate variability and for understanding the genesis of breathing pattern. That is undeniable: it happens, and therefore it has consequences, whether or not these consequences are important. It is a different matter to talk about coupling having a role. Even if coupling has consequences, it does not mean that those consequences are the purpose of coupling.

In their latest study,3 on the relation between respiratory sinus arrhythmia and cardioventilatory coupling, Galletly and Larsen suggest that these mechanisms are important (that is, by implication, they have a purpose) in reducing the effect of sleep or posture on pulmonary gas exchange. They also suggest that their finding of a link between the two phenomena strengthens the argument for a physiological purpose.

I suggest that a link between respiratory sinus arrhythmia and cardioventilatory coupling is entirely to be expected, if not inevitable, given that both depend on the interaction between the same two rhythmic processes. While it is possible that either or both phenomena are physiological adaptive mechanisms, they might just as easily be an accident of the closeness with which respiratory and cardiovascular control are interdependent. If coupling confers some advantage, what sort of evolutionary pressure is supposed to have brought it about? Cardioventilatory coupling is seen in humans only when at rest. Disorders of gas exchange in sleep are irrelevant in healthy subjects, and unlikely to have exerted evolutionary pressure.

I admit that evolutionary supposition is only supposition, and I have no evidence that respiratory sinus arrhythmia and cardioventilatory coupling are just an accident. It is difficult to think how supportive evidence could be gathered, which makes my suggestion of an accident a speculation rather than a hypothesis. Nonetheless, there is a lesson that carries over to the whole of research: beware of ascribing meaning to observation. In anaesthesia, we do not have to go far to find a similar mystery: why is there butyrylcholinesterase in our plasma? More generally, why do men go bald? Much of our physiology is without explanation, and possibly without purpose. Just because something is interesting — as cardioventilatory coupling is — does not mean that it has a purpose, although it can give useful insights into the way our physiology works. There is nothing wrong with continuing to make observations on coupling, but I should prefer to refer to introductions, “It is not known if coupling has any physiological role...” rather than “Although the physiological role is unknown...”


Editor,—During anaesthesia, the reflex coupling of inspiratory onset to the cardiac cycle is an important determinant of ventilatory timing. In a recent paper, we examined the relationship that exists between respiratory sinus arrhythmia (RSA) and cardioventilatory coupling (CVC). We observed that, during CVC, heart beats are generally placed at positions of the ventilatory cycle where they are maximally affected by RSA. This observation was unexpected and not, as Dr Goodman suggests, "inevitable". As experimental evidence has demonstrated that the presence of RSA may be important in optimizing pulmonary gas exchange, we put forward a hypothesis that CVC, by maximising RSA, might also help pulmonary oxygenation. Because CVC occurs primarily during low arousal states, we suggested that coupling may aid gas exchange during sleep. In our paper we clearly stated "Whether the relationship between RSA and phase coupling is simply fortuitous... or the result of evolutionary design is open to question." We did not assume that coupling had a physiological role, only that a benefit may exist and warrants investigation.

Our conjecture of coupling optimizing cardiopulmonary function is a testable or falsifiable hypothesis. This is in contrast to the classification of coupling simply as an accident of evolution as Goodman proposes. To categorize any physiological process as an accident, simply because one cannot see what evolutionary pressure produced it, does little to advance our understanding of physiology. The case of butyrylcholinesterase is interesting. We cannot understand why the drug-metabolizing enzyme should be present, but that does not mean that it is without function. The "accident" may simply represent the anaesthetist's blinkered view of a world dominated by pharmacology. Other roles far removed from that of drug metabolism have been suggested. Unless one takes a creationist or Lamarckian view, all mutations are Darwinian accidents, selected or not selected according to their benefit to the organism. What is initially an "accident" might over time become important, because it confers some advantage to the organism or because other critical processes may become adapted to, and dependent upon, it.

Goodman believes that little evolutionary "pressure" would have been brought to bear on the gas exchange of the otherwise fit adult. This is possibly correct, but our present level of health and longevity is evolutionarily recent; any advantage of coupling may be more apparent during early childhood development and in certain animals (perhaps those that hibernate). Coupling occurs in infancy as it does in adults; it is present in rats, guinea pigs and rabbits and the work of Coleman suggests its presence in almost every mammalian species that he examined at London Zoo. Although the lung structure did not evolve from that of gills, it is also interesting that Schoenlein in 1895 demonstrated the coupling of gill movements and heart beats in fish. Plainly coupling is evolutionarily old and has persisted throughout the mammals.

It is natural for us to attempt to ascertain the importance of coupling in the dynamics of human cardioventilatory control and to examine whether the process is of physiological benefit. By defining physiological advantage we move from basic science in the direction of clinical relevance. Correct or otherwise, it is surely desirable for a scientist to conjecture as to the meaning of an observation. Rather than "Beware of ascribing meaning to observation", we would prefer to read "We have tested Galletly and Larsen's hypothesis and found...".

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