A 54-yr-old man was scheduled for laryngopharyngectomy with jejunal free flap, as surgical treatment for recurrence of a laryngeal tumour involving the posterior cricoid ring. Seventeen years earlier he had received a cadaveric heart–lung transplant, as treatment for histiocytosis X. He continued to have good cardiorespiratory function, and had no evidence of graft rejection, although mild renal impairment was present. The patient’s airway was unobstructed, and recent anaesthesia for microlaryngoscopy had been uneventful. 

Venous access, including central venous cannulation of the femoral vein, and radial arterial cannulation were achieved under local anaesthesia. A crystalloid preload was given. An isoproterenol (isoprenaline) infusion was commenced at a rate of 1 µg min⁻¹ in anticipation of bradycardia occurring on induction. Anaesthesia was induced with propofol 140 mg, and fentanyl 200 µg, anticipated of bradycardia occurring on induction. Anaesthesia was induced with propofol 140 mg, and fentanyl 200 µg, followed by atracurium 40 mg. A 7.0 mm oral tracheal tube was placed easily at direct laryngoscopy. The patient’s lungs were ventilated, and anaesthesia maintained with an oxygen/air mixture and desflurane to an end-tidal concentration of 4.3%. A remifentanil infusion was then started at an initial rate of 0.05 µg kg⁻¹ min⁻¹, and increased during surgery to an infusion range of 0.1–0.2 µg kg⁻¹ min⁻¹, titrated according to arterial blood pressure and surgical stimulation. The isoproterenol infusion was continued at the same background rate. Mild, transient bradycardia was encountered on induction (65 beats min⁻¹), but not during the administration of remifentanil. During the procedure, the patient exhibited no significant haemodynamic instability. The patient’s pulse rate varied between 80–105 beats min⁻¹, and his blood pressure was easily maintained within the range 90–120 mm Hg (systolic).

The total duration of surgery was 10 h, during which the larynx and lower pharynx were excised, and a portion of jejunum was anastomosed to reform the pharynx. At the completion of surgery, remifentanil and the volatile agent were discontinued, and the patient awoke promptly. Local anaesthetic had been infiltrated into the small incision through which the jejunum flap had been harvested, and further postoperative analgesia was provided by incremental dosages of morphine totalling 10 mg. The patient was transferred, breathing spontaneously, to the high dependency unit for overnight care, during which he required a further total of morphine 4 mg, self-administered from a PCA device. He was discharged to a general ward on the following morning, and made a full recovery.

The anaesthetic management of heart transplant recipients has been reviewed elsewhere.\(^1\)\(^2\) Metabolism of anaesthetic drugs may be altered if renal impairment is present, while the immunosuppressant drug cyclosporine has been shown to increase the MAC of isoflurane.\(^4\) However, the behaviour of the donor heart, which has been separated from its native autonomic supply, dominates anaesthetic considerations. Most anaesthetic techniques can potentially provoke bradycardia, or decrease venous return, neither of which are well tolerated by the transplanted heart. Any technique needs to anticipate these occurrences, and have the means to counteract them to hand.

To our knowledge, the use of remifentanil in a heart transplant recipient has not yet been described, nor is it mentioned in the two most recent reviews on this subject.\(^5\)\(^6\) However, it is successfully used in cardiac surgery,\(^7\) and from our experience, during the heart transplant procedure itself. The propensity of remifentanil, when administered as a bolus, to cause bradycardia and cardiovascular instability has been reported,\(^8\) and is probably related to a centrally mediated increase in vagal activity. Theoretically, the denervated heart might be less susceptible to this effect, but in practice it would remain a concern. We intended to minimize these risks by administering remifentanil as an infusion, starting at the lowest possible dose. Also, by avoiding the prolonged use of neuromuscular blocking agents, antagonism with neostigmine, which has been associated with asystole in a heart transplant recipient,\(^9\) was not required.

In this patient, remifentanil was successfully used without adverse effects. It is likely that there will be increasing numbers of transplant recipients presenting for surgery of this nature. The immunosuppressant regimens that prevent rejection so successfully, may also render patients more prone to malignancies of this type.\(^10\) Further cases involving the use of remifentanil need to be reported however, before potential problems can be identified.

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Hyperventilation-induced transient spastic quadraparesis

Editor—We would like to report a case of transient spastic quadraparesis in a parturient presenting with extreme hyperventilation during active labour. During such conditions of extreme pain, patients hyperventilate, overriding their involuntary brainstem respiratory centre control.\(^1\) An otherwise healthy 21-yr-old, G3P1 full-term parturient, presented to our labour and delivery unit. She reported a successful labour epidural followed by 6 months of lower back pain, after her previous delivery. Following rapid progress of her current labour, the patient requested neuraxial analgesia after initial refusal, reporting unbearable contraction pain. Before neuraxial analgesia had been established, however, and after a period of hyperventilation, the patient was found lying supine with both arms and legs extended, and both wrists in extreme flexion (carpopedal spasm), looking upwards and breathing vigorously (60–80 bpm). The patient had experienced sudden spastic paresis involving both upper and lower extremities 5 min earlier, with loss of passive
ability to straighten her upper extremities, and a high muscle tone. She also complained of dizziness, blurred vision, and numbness in her lower extremities during the event, and a 5 min prodromal episode of paraesthesia in her nose and toes leading up to it. Management of the patient included re-breathing of carbon dioxide by placing a non-rebreathing reservoir face mask, with the oxygen supply turned off over her face, and i.v. sedation (fentanyl 50 μg), leading to resolution of the symptoms over 10 min. The patient delivered a vigorous healthy boy, 25 min later. Neurological and laboratory investigations revealed only a low total blood calcium (8.6 mg dl^{-1}) and a high alkaline phosphatase (207 U litre^{-1}), which is normal during pregnancy because of increased placental production. Both patient and baby were discharged home after a normal neurological examination.

The most likely cause of this event, we feel, is transient hypocalcaemia induced by hyperventilation-induced respiratory alkalosis. Raised maternal progesterone increases minute ventilation during pregnancy, but does not change arterial pH. Acute hyperventilation in excess of this will, however, increase arterial pH, the effect of which may not be favourable to the fetus. In vitro work has shown that changing serum pH by 0.5 pH units can alter ionized calcium from between 0.2–0.4 mM. This occurs because of increased calcium binding to serum albumin, reducing ionized serum calcium. While bovine parturient paresis is common when taking a low calcium diet, other hypocalcaemic disorders do not appear to cause significant neuromuscular problems in pregnancy.

It is possible that this patient was experiencing a seizure or pseudo-seizure. However, we feel this is unlikely as the patient had no previous history of seizures. There was no history of pre-eclampsia or hypertension, and the transient nature and quality of this single episode with no loss of consciousness, would not suggest such an aetiology. Conditions such as periodic paralysis occur because of changes in serum potassium and lead to hypotonic or flaccid paralysis. 2 No change in serum potassium was found in this patient. There is a report of tetany induced by hyperventilation in a dental patient, showing that it can be caused by acute hyperventilation in normal patients, and cases of tetany have been reported in normocalcaemic patients. Our patient had a dramatic 15 min sustained spastic quadraparetic episode, after a prolonged period of hyperventilation during labor. The most likely cause of this event is transient hypocalcaemia from hyperventilation-induced respiratory alkalosis. Both awareness of this differential diagnosis and prevention of extreme hyperventilation is important in the management of obstetric patients, especially those without prenatal education (e.g. instruction in the Lamaze breathing pattern or similar preparatory training). Finally, this is important before establishing neuraxial and/or other means of analgesia.

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Left double lumen tube malposition

Editor—We read with interest the paper by Inoue and colleagues describing the importance of left double lumen tube (L-DLT) malpositioning during one-lung ventilation (OLV) and the prevalence of hypoxaemia.

The authors defined their correct DLT position according to Slinger 2 as ‘an unobstructed view into the left upper and lower lobe bronchus through the endobronchial lumen with the bronchial cuff immediately below the carina and just visible in the main left bronchus through the tracheal lumen’. They defined malposition ‘if the tube had to be moved (in or out) by more than 1.0 cm to correct its position’.

We disagree with their definition of DLT malpositioning. Clinically, a malpositioned L-DLT occurs when the tube obstructs the left upper lobe bronchus or when the top of the endobronchial cuff is above the carina. This type of malpositioning may explain desaturation or insufficient lung separation. A position between these two extremes has no clinical significance and should be considered inside the margin of safety. The authors did not report the type of malpositioning they encountered (proximal or distal), and it seems to us that a 1 cm displacement is difficult to evaluate by fibreoptic bronchoscopy.

Finally, the authors discussed ‘how can we stop DLTs becoming misplaced? We have no solution so far…’. We would like to highlight the fact that the L-DLT was redesigned in 1994 to increase its margin of safety. These modifications cause problems when the tube is positioned using the classic approach, and the patient is therefore placed in lateral decubitus before checking the L-DLT position. We conducted a study to address the problem of L-DLT malpositioning and described a new method of positioning by visualizing the carina through the transparent wall of the L-DLT. This new method necessitated less re-positioning of the L-DLT than the classic technique (23% compared with 53%), and should be use routinely in thoracic surgery.

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Editor—Thank you for giving us the opportunity to reply to this interesting letter. We agree with the comment that we used the classic definition by Slinger 2 for DLT positioning and we would have had different results if we had used the new definition described by Fortier and colleagues. However, the main point that we would emphasize is that patients who have DLT malposition after being placed patient in the lateral position...