Airway obstruction and microsleep after surgery

Editor—Rahman and colleagues\(^1\) make the interesting observation that obstructed breathing in post-operative patients was more common when they were awake than when they were asleep. This seems to contradict our study\(^2\) where we found obstructed breathing postoperatively only when patients were asleep. There were important differences between the two studies. We\(^2\) divided 32 patients into two equal groups, one having morphine, the other regional block. The dose of morphine was about 28 mg in 14 h and no opioid was used in the regional block group. In Rahman and colleagues’ study three patients had a median dose of 51 mg morphine in the 14 h study period and five had thoracic epidural blocks which included opioids.

There are differences in the criteria of obstruction. Rahman and colleagues\(^1\) defined obstruction as a phasic abdominal EMG pattern at any time or any change in nasal flow pattern during a 30 s measuring period. My reading of this is that one obstructed breath in a 30 s measuring period registered as a 30 s period of airway obstruction and if every breath was obstructed during the 30 s period it would give the same duration of obstruction. Our criteria and methods were different.\(^2,3\) paradoxical breathing (partial obstruction) was where 50% of breaths in a 5 min period showed phase difference between the movement of ribs and abdomen and obstructive apnoea was recognized only when the sum of rib cage and abdominal movement contributing to tidal volume was zero for more than 10 s. This means that Rahman and colleagues\(^1\) would have detected many, very brief (<10 s), periods of apnoea in ‘awake’ patients all of which we would have ignored. In our study, partial obstruction with snoring\(^1\) lasting 5 or more min was the most common breathing pattern associated with hypoxaemia (\(\text{SaO}_2 < 80\%\)) but occurred only during sleep and only after morphine.

A further crucial difference is the assessment of the ‘sleep/awake’ state. While both studies used the same EEG criteria, Rahman and colleagues\(^1\) defined patients as ‘awake’ up to EEG stage 2 whereas we defined a ‘transition stage’ of sleep at EEG stages between 1 and 2. Thus we labelled as ‘asleep’ some patients that Rahman and colleagues\(^1\) would have labelled ‘awake’ using their criteria. Nevertheless, in this transitional group we found only 94 episodes of obstruction compared to 510 episodes when asleep. In Rahman’s study, there were more episodes of obstruction in their ‘awake’ patients than when they were asleep. I propose that these brief periods of obstruction in patients whom Rahman and colleagues\(^1\) define as ‘awake’, occurred in a non-steady state of altering consciousness or microsleep.

This raises the question about how we define sleep. Rahman and colleagues\(^1\) remind us that the standard definition describes sleep as an EEG sleep pattern lasting more than half of the 30 s recording epoch. Thus, loss of consciousness for a shorter period than 15 s cannot be defined as sleep using this criterion and therefore an obstructive episode during a period of unconsciousness for less than 15 s is registered as occurring during wakefulness. They are rightly critical of these 1968 EEG criteria devised for non-drugged sleep apnoea patients when applied to postoperative patients receiving opioids.

I suggest that these EEG criteria are inappropriate for detecting brief periods of loss of consciousness, which during sedation, are associated with the changes in muscle tone that are the recognized parts of the sleep phenomenon. Thus eye blink duration is a measure of sedation.\(^4\) Normal blink duration is less than 500 ms (median 95 ms) but during sedation blink duration increased to more than 8 s. Inhibition of cortical processing of visual information precedes blink onset by 50 ms and persists for 200 ms after blink onset. In the context of driving a car at 70 mph (103 feet s\(^{-1}\)), a sleepy driver who blinks for 8 s will travel over 800 feet during which time the driver will have no conscious realization that the eye has closed. This brief loss of tone in the eyelid muscles reflects a loss of tone in the neck and elsewhere, probably including the upper airway, and represents microsleep, which is a feature of anaesthetic sedation.\(^5\) I suggest that this may also be a common feature of opioid administration in the postoperative period, and that Rahman and colleagues\(^1\) have identified transient airway obstruction during microsleep. While such brief periods of unconsciousness and hypotonia may be fatal in car drivers, they are probably harmless in postoperative patients in terms of worsening hypoxaemia.

Rahman and colleagues\(^1\) extrapolate from the sleep apnoea syndrome to suggest that intermittent airway obstruction following opioids causes arousal and this disturbs sleep in postoperative patients. This might not be the case for the following reason. I was a subject in a study of the effects of morphine on breathing with and without a considerably increased airway resistance (80 cm H\(_2\)O litre\(^{-1}\) s\(^{-1}\)).\(^5\) I was surprised that morphine abolished the very unpleasant effects of this severe airway obstruction. Morphine predisposes to sleep, hypoxaemia and airway obstruction but part of its danger lies in reducing the arousal effects of the latter.

J. Gareth Jones

Cambridge

UK

Editor—Thank you for the opportunity to respond to Professor Jones’ letter with his comments on our study. We agree that our findings contradict his study,\(^1\) and that there are substantial differences in the methods used that make comparison of the two studies awkward. The most obvious of these is the use of very different time blocks, 5 min in his and 30 s in ours. We are also grateful for the opportunity to comment on some of the difficulties of studies of this type.

However, Professor Jones is wrong when he quotes our definition of airway obstruction. Our exact definition was: ‘Respiration was considered abnormal if there was any change in the clear inspiratory and expiratory pattern, or if the smooth

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domed appearance of the inspiratory wave was reduced in amplitude with a ‘flow limitation’ pattern. We considered each aspect of the records separately, for sleep, phasic activity, and obstruction, in identical 30 s epochs. In each of these 30 s epochs we applied the above criteria to the flow signal. Any period that contained an abnormality of respiration, was classified as abnormal. We do not believe that this result in too many periods being classified as abnormal. We did not often observe the events he suggests, of brief periods of obstruction. Generally (although we have no exact data yet), the patterns we saw were either an abrupt change from small to large breaths or a persistent pattern such as we illustrate in our paper. We feel that this method of detection or obstructed breathing is not perfect but it does have considerable advantages over measurements of chest wall movements. In our account, we cite our previous observations that the relationships between movement, flow, and effort are complex. In particular, observations of oesophageal pressure and gastric pressure at the same time as chest wall movements suggest that in patients after abdominal surgery, chest wall movements are less reliable measures of obstructive apnoea, or even partial obstruction, than they may be in other circumstances. We were particularly keen to avoid bias, in association of events, by considering the signals separately. It is not clear if this was done in Jones’ study.

We did not attempt to re-define sleep staging for our patients, although we were tempted! Jones is correct when he argues that there may be a ‘transience stage’. In our study we stuck carefully to the criteria we cite. Patients were either awake, or asleep, which as we report was almost always stage 2. We did not consider stage 1 as awake, but we rarely saw this stage anyway.

Professor Jones’ other observations are of interest, but we do not think, at present, that we have sufficient information to be certain what is going on when our patients have airway obstruction after surgery. We are presently gathering such data, which confirm that sleep disturbance and arousals are very prevalent, and also support the observations of the study we have already mentioned, using skin prick testing. It was again considered most likely that the patient’s reaction was due to the introduction of chlorhexidine in some way into the circulation. Surgery was rescheduled and on this occasion the patient was admitted to the intensive care unit the day before, where invasive monitoring was established avoiding chlorhexidine, under controlled conditions and without any adverse reactions. At this point, we considered other possible sources of chlorhexidine contamination. We realized that the anaphylactic reactions had occurred not only at the time of insertion of the central venous line, but were also temporally related to the insertion of the urethral catheter. In our institution, this is routinely performed by the surgical team, who use INSTILLAGEL. We were unaware that the urethral lubricant contained chlorhexidine and indeed, of the previously reported cases of anaphylaxis associated with urinary catheter lubricants. We advise caution with the use of chlorhexidine-containing compounds for urethral lubrication in any patient with a history of unexplained anaphylaxis, and emphasize the importance for anaesthetists to remain aware of ‘non-anaesthetic’-related possibilities for the introduction of potential allergens. We have since stopped using INSTILLAGEL for urethral lubrication.

A 71-year-old male was scheduled for elective mitral valve replacement for prolapse of the posterior leaflet of the mitral valve, causing severe mitral regurgitation. His past medical history included allergy to penicillin. Anaesthesia was induced uneventfully, using fentanyl, propofol, midazolam and pancuronium. During suturing of the right internal jugular central lines, the patient developed an acute anaphylactic reaction, characterized by urticaria, erythema and circulatory collapse. Bronchospasm was also not a feature in this case. The anaphylaxis was successfully managed with epinephrine, metaraminol, hydrocortisone and chlorpheniramine. Skin preparation for the insertion of neck lines had been with chlorhexidine. Surgery was abandoned and the patient was transferred to the intensive care unit where he made an uneventful recovery.

Our patient was referred to a dermatologist who performed skin patch testing for midazolam, fentanyl, propofol, pancuronium and chlorhexidine. The only positive reaction was to chlorhexidine. We therefore postulated that the anaphylactic reaction could have been caused by the introduction of chlorhexidine to the circulation from the skin during insertion of the central line. The patient was rescheduled for surgery some weeks later, and preparation for anaesthesia included premedication with hydrocortisone, ramitidine and chlorpheniramine. Anaesthesia on this occasion was induced with etomidate, remifentanil, and pancuronium, again uneventfully. On this occasion, chlorhexidine was avoided in all skin preparations. However, once again following insertion of the central line, the patients developed an anaphylactic reaction in exactly the same manner as on the previous occasion. Resuscitation was successful using epinephrine, hydrocortisone and chlorpheniramine. The patient again made an uneventful recovery. The central venous catheters we used were not coated in any way with chlorhexidine. The serum tryptase was only moderately elevated.

We sought advice from a consultant immunologist and a consultant anaesthetist who run a specialist joint clinic to investigate anaphylactic reactions during anaesthesia. They tested for latex allergy, ethylene oxide allergy, and for the other drugs already mentioned, using skin prick testing. It was again considered most likely that the patient’s reaction was due to the introduction of chlorhexidine in some way into the circulation. Surgery was rescheduled and on this occasion the patient was admitted to the intensive care unit the day before, where invasive monitoring was established avoiding chlorhexidine, under controlled conditions and without any adverse reactions. At this point, we considered other possible sources of chlorhexidine contamination. We realized that the anaphylactic reactions had occurred not only at the time of insertion of the central venous line, but were also temporally related to the insertion of the urethral catheter. In our institution, this is routinely performed by the surgical team, who use INSTILLAGEL® (Clinkmed, High Wycombe, UK) to lubricate the urethra. On examination of the components of INSTILLAGEL®, we discovered that it contains chlorhexidine gluconate 0.25 g per 100 g. We believe that on each occasion the anaphylaxis was unrelated to the insertion of the central venous line and was caused by absorption of chlorhexidine from the urethral mucosa. Our patient was successfully anaesthetized and catheterized without the use of INSTILLAGEL®. Anaphylaxis did not occur, and surgery and recovery were uncomplicated.

Review of the literature supports this theory, and we were able to find a number of case reports of anaphylaxis following absorption of chlorhexidine via the urethra. 2,4

We were unaware that the urethral lubricant contained chlorhexidine and indeed, of the previously reported cases of anaphylaxis associated with urinary catheter lubricants. We advise caution with the use of chlorhexidine-containing compounds for urethral lubrication in any patient with a history of unexplained anaphylaxis, and emphasize the importance for anaesthetists to remain aware of ‘non-anaesthetic’-related possibilities for the introduction of potential allergens. We have since stopped using INSTILLAGEL® for urethral lubrication.

Gordon Drummond
Edinburgh
UK


Anaphylactic reactions due to chlorhexidine allergy

Editor—We read with interest the case report by Stephens and colleagues.1 We have had similar experience, with two life-threatening anaphylactic reactions occurring in the same patient who was also undergoing cardiac surgery and which we believe was due to chlorhexidine allergy.

A 71-year-old male was scheduled for elective mitral valve replacement for prolapse of the posterior leaflet of the mitral valve, causing severe mitral regurgitation. His past medical history included allergy to penicillin. Anaesthesia was induced uneventfully, using fentanyl, propofol, midazolam and pancuronium. During suturing of the right internal jugular central lines, the patient developed an acute anaphylactic reaction, characterized by urticaria, erythema and circulatory collapse. Bronchospasm was also not a feature in this case. The anaphylaxis was successfully managed with epinephrine, metaraminol, hydrocortisone and chlorpheniramine. Skin preparation for the insertion of neck lines had been with chlorhexidine. Surgery was abandoned and the patient was transferred to the intensive care unit where he made an uneventful recovery.

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A. S. Lockhart
C. C. Harle
Blackpool
UK
Autologous transfusion in total knee replacement surgery

Editor—I read the recent study on autologous transfusion in total knee replacement surgery with interest.¹ We have recently introduced into our hospital postoperative drains, which allow re-transfusion of unwashed, filtered, autologous blood after knee replacement surgery (Bellovac A.B.T., Astra Tech, Sweden). At approximately the same time, we introduced a transfusion ‘trigger’ of 9 g dL⁻¹. We audited the effectiveness of these drains and the ‘trigger’ in reducing the requirements for allogeneic transfusion compared to the previous year’s transfusion data obtained from the hospital blood bank database. Twenty-four patients were audited and the results are shown in Table 1.

Both patients who received allogeneic transfusion had an Hb of 8.1 g dL⁻¹ (i.e. below the ‘trigger’ and therefore appropriate). This represents an allogeneic transfusion rate of 8.3% of patients, compared to the historical data showing a transfusion rate of 25% (average number of units transfused 2.5, range 1–4). It is interesting to note that this is a similar reduction in allogeneic rate to that found in the study by Thomas and colleagues.²

Table 1  Effectiveness of postoperative drains and transfusion ‘trigger’ in reducing requirements for allogeneic transfusion

<table>
<thead>
<tr>
<th>Description</th>
<th>Before new drains</th>
<th>After new drains</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume reinfused (ml)</td>
<td>686 (200–1499)</td>
<td>13.9 (10.1–16.9)</td>
</tr>
<tr>
<td>Starting Hb (g dL⁻¹)</td>
<td>11.2 (8.1–13.9)</td>
<td>2.76 (1.3–6.2)</td>
</tr>
<tr>
<td>Postoperative Hb (g dL⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Reduction in Hb (g dL⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Allogeneic transfusion</td>
<td>2 patients (2 and 3 units)</td>
<td></td>
</tr>
</tbody>
</table>

In the re-infused group there were two deep vein thromboses (DVT), one transient ischaemic attack, and one patient had a ‘sweaty’ episode during the autologous transfusion which was therefore stopped. The ‘in-hospital’ DVT rate of 8.3% compares favourably with quoted rates of 20% and 36%, which are at the lower end of the published range. A cost analysis is presented in Table 2. There was approximately £14 excess per patient after the introduction of the new drains, which is considerably better than the £113 excess per patient in Thomas’ study.

One of the main worries about re-transfusion of unwashed blood is the possibility of the transfused blood containing an excessive amount of inflammatory mediators. Studies have shown that while this is the case, transfusion of this blood did not increase the circulating levels of these mediators.³ In addition, it has been shown that infective episodes are significantly reduced by the avoidance of homologous blood.⁴ I believe this audit suggests that the introduction of drain systems that allow re-transfusion of autologous blood, along with a transfusion trigger, can significantly reduce the amount of allogeneic blood used, and at the same time produce a cost saving without an excess of side effects.

J. C. Hughes
Bridgend
UK

Table 2  Cost analysis comparing transfusion before and after new drains

<table>
<thead>
<tr>
<th>Description</th>
<th>Before new drains</th>
<th>After new drains</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cost per patient of allogeneic blood per patients at current cost of £90 unit⁻¹</td>
<td>£54</td>
<td>£18.75</td>
</tr>
<tr>
<td>Cost of drains in excess of standard drains per patient</td>
<td>£0</td>
<td>£50</td>
</tr>
<tr>
<td>Total per patient</td>
<td>£54 (£18.75)</td>
<td>£68.75 (£28.75)</td>
</tr>
</tbody>
</table>


Editor—We are grateful to Drs Lockhart and Harle for reporting their similar experience with chlorhexidine allergy and the opportunity to add some further comments.

We also had considered chlorhexidine in the skin preparation used for insertion of the urethral catheter, and the INSTILLAGEL® lubricant as potential causes of the anaphylaxis. Hence, we had also sought to avoid both of these agents on the second occasion. In fact, the anaphylactic episode occurred exactly as the urethral catheter had been placed on the first occasion, and it was the timing of this event that led us to believe that the urethral catheterisation had been the likely precipitant!

However, on the second occasion, urethral catheterization had not yet occurred, as we were endeavouring to carry out each procedure separately as a precaution against confusing any subsequent adverse event, with a particular incident. On this occasion, the timing of the event coincided only with the i.v. flushing of the newly inserted central venous line. It was not appreciated at this time that this particular central venous line was coated in chlorhexidine. It was only subsequently when the package inserts were retrieved from the waste bins that the written warning to avoid such lines in patients with known sensitivity to chlorhexidine was appreciated.

We would, therefore, advise caution even when using equipment with which you are familiar, but in which there are identifiable package inserts which might not have been read for some time!

Wynne Davies
London
UK

Correspondence

Editor—I agree with the comments made by Dr J. C. Hughes. The use of re-infusion devices to return unwashed, filtered and salvaged blood has proved very successful following knee surgery. It is encouraging that, in his group of patients, he found a similar reduction in the use of allogeneic blood to our study, by employing a transfusion trigger of 9 g dl$^{-1}$ and using a blood salvage technique.

It appears that the re-infusion of relatively low volumes of unwashed blood following primary unilateral knee replacement surgery does not have serious clinical consequences. Larger volumes salvaged during other surgical procedures may cause greater clinical effects. Also, blood salvaged following hip arthroplasty may contain a greater concentration of inflammatory mediators.

The excess cost involved in postoperative salvage using a cell salvage machine as described in our paper was definitely overestimated. The operator time was calculated for the whole collection period and processing. In practice, this is not the case and the operators were able to carry out other duties whilst intermittently visiting the patient, to wash and process the collected blood.

We believe that with current costs, which now favour the use of any recycling equipment, providing one unit of blood is salvaged, almost all systems on the market are cost-effective. As we have a red cell salvage service in place with operating department personnel experienced in the operation of centrifugal devices, this is a standard of care we wish to continue. The use of simple re-infusion has, of course, been used with good results and may well suit the smaller orthopaedic units. Use of such devices for large volumes of recycled blood is unlikely to prove cost effective and may cause a greater inflammatory reaction.

D. W. Thomas
Swansea
UK

Numbness a greater problem than pain?

Editor—in his article ‘Analgesia for day-case surgery’, Rawal$^1$ suggests that bupivacaine and ropivacaine are suitable solutions for brachial plexus blockade in patients having day-case surgery. These drugs last from 12 to 15 h$^2$ and most patients who have ever had a ‘local anaesthetic’ at the dentist will agree that the sensation of numbness and heaviness is extremely unpleasant.

Brachial plexus block performed using 1.5% lidocaine 35–40 ml with 1/100 000 epinephrine will produce analgesia lasting 3–6 h.$^3$ Most people having day-case surgery will be relieved when this block wears off. If patients are warned that their surgical site will be painful and, in anticipation of this, begin treatment with paracetamol or NSAIDs, I believe they would have greater satisfaction than if they were to return home to nurse a ‘dead arm’ for more than 12 h.

In my opinion, bupivacaine or ropivacaine should only be used to perform brachial plexus blocks on inpatients having extensive surgery and returning to a unit where there are experienced staff to help them cope with their numb limb.

Anthea Hatfield
Walsall
UK

1 Rawal N. Analgesia for day-case surgery. Br J Anaesth 2001; 87: 73–87
2 Klein SM, Greengrass RA, Steele SM et al. A comparison of 0.5% bupivacaine, 0.5% ropivacaine, and 0.75% ropivacaine for interscalene brachial plexus block. Anesth Analg 1998; 87: 1316–9

Ondansetron after Caesarian section

Editor—I was surprised to see a study in which ondansetron had been given to mothers in the post-partum period,$^4$ since I am aware that manufacturers caution against its use in breast-feeding women. Were the women in the study made aware of the potential

2 Murphy DB, Chan VWS. Upper extremity blocks for day surgery. Tech Reg Anesth Pain Mgmt 2000; 4: 19–29

1 Klein SM, Greengrass RA, Steele SM et al. A comparison of 0.5% bupivacaine, 0.5% ropivacaine, and 0.75% ropivacaine for interscalene brachial plexus block. Anesth Analg 1998; 87: 1316–9


Ondansetron after Caesarian section

Editor—I was surprised to see a study in which ondansetron had been given to mothers in the post-partum period,$^4$ since I am aware that manufacturers caution against its use in breast-feeding women. Were the women in the study made aware of the potential
for unknown adverse effects on their baby? In my experience, women will tolerate far more serious symptoms than nausea if the health of their baby depends on it. I am not belittling the distress caused by nausea, but I am concerned that it is unethical to subject women to a drug that may harm their baby. Thalidomide was also an antiemetic. The days of vague explanations and administration of potentially toxic drugs to uninformed patients should be consigned to the past.

Leyla Sanai
Glasgow
UK

Editor—Thank you for allowing me to respond to this letter. The issue of breast feeding is always difficult, due to the lack of objective data for most drugs. A great many drugs are present in breast milk, although generally in small amounts. For the few that are not, the manufacturer is in the fortunate position of being able to declare their product safe while breast feeding. With the remaining drugs, either trace amounts are excreted in breast milk, or the fate of the drug is unknown. For obvious reasons, most pharmaceutical companies do not study the effect of maternally administered drug on breast-fed babies, choosing instead to simply issue a blanket caution. This is the current position for both ondansetron and prochlorperazine; there is no evidence that they are actually harmful, simply no proof that they are not. Thalidomide was altogether different, being given during the crucial period of organogenesis.

Despite the understandably cautious approach of the pharmaceutical industry, it is often necessary to administer drugs which are excreted in breast milk to lactating mothers. Both prochlorperazine and ondansetron are widely used in the postpartum period and, to date, there have been no reports or suggestions of any harm to newborn babies, despite their widespread use. Both drugs are routinely used in our institution (prochlorperazine as first line, ondansetron as rescue, for ‘high-risk’ patients or where the former is contraindicated). While we accept the need to warn breastfeeding mothers receiving antiemetics, and dol, several codeine preparations, and many NSAIDs.

Asystole during anaesthetic induction with remifentanil and sevoflurane

Editor—Severe bradycardia has been reported in patients receiving boluses of remifentanil.1–4 Pre-existing cardiac disease, beta-blocking and calcium channel blocking drugs have been suggested as risk factors,1,4 most of the cases being described in patients scheduled for fast track cardiac surgery.1,2,5 The use of sevoflurane may also be a predisposing factor.1 We report a case of asystole in a patient who was given remifentanil and sevoflurane for induction of anaesthesia. This 78-yr-old man was suffering from laryngeal cancer, and was scheduled for laryngeal endoscopy. He was a heavy smoker. He had been treated with 5-fluorouracil and cisplatin 1 month previously, but did not receive any medication at the time of anaesthesia. Pre-anaesthetic examination noted a basal heart rate of 62 beats min⁻¹.

In the operating theatre, after placement of an arterial pressure cuff, ECG, and pulse oximeter, the patient was preoxygenated with 100% oxygen. Anaesthesia was induced through a face mask with 8% sevoflurane in a mixture of 60% nitrous oxide and 32% oxygen. Loss of consciousness occurred after four vital capacity breaths and the patient was manually ventilated until the end-tidal sevoflurane concentration reached 5%. Mivacurium 0.2 mg kg⁻¹ was given followed by an i.v. bolus of remifentanil 0.5 µg kg⁻¹ over 1 min, and a 0.5 µg kg⁻¹ h⁻¹ continuous infusion. Within 1 min, heart rate decreased from 50 beats min⁻¹ to asystole, unresponsive to i.v. atropine 1 mg. The remifentanil infusion was stopped immediately and 100% oxygen administered. Cardiac sinus rhythm resumed after two precordial thumps. The trachea was intubated and the laryngeal endoscopy was performed under 2% sevoflurane and a remifentanil 0.15 µg kg⁻¹ h⁻¹ infusion. No other episode of bradycardia or cardiac arrest occurred during anaesthesia or in the following 24 h of continuous heart rate monitoring. Postoperative ECG, plasma tropinin concentrations and echocardiography were all normal.

Only one case of asystole has been previously reported in a patient given sevoflurane and remifentanil,1 but several cases of severe bradycardia have been documented in patients receiving remifentanil.1–4 In this case, where no other risk factor was documented, we postulate that the rapid sequence induction of anaesthesia with sevoflurane blunted sympathetic tone,3 and allowed uncompensated parasympathetic activation by remifentanil. We thus recommend that induction of anaesthesia with sevoflurane should be avoided in patients about to receive remifentanil.

Okba Kurti
Arnaud Deleuze
Emanuel Marret
Francis Bonnet
Paris
France

4 De Souza G, Lewis MC, TerRiet MF. Severe bradycardia after remifentanil. Anaesthesia 1997; 87: 422–3