Forum

Thoracic epidural anaesthesia for coronary artery bypass
graft surgery  Effects on postoperative complications

D. J. Turfrey,1 D. A. A. Ray,1 N. P. Sutcliffe,1 P. Ramayya,1 G. N. C. Kenny2 and N. B. Scott1*

1 HCI International Medical Centre, Beardmore Street, Clydebank, Glasgow G81 4HX, UK
2 University Department of Anaesthetics, Glasgow Royal Infirmary, 8–16 Alexandra Parade, Glasgow G31 2ER, UK

Summary
We have performed a retrospective analysis of the peri-operative course of 218 consecutive patients
who underwent routine coronary artery bypass graft surgery in this institution. All patients
received a standardised general anaesthetic using target-controlled infusions of alfentanil and
propofol. One hundred patients also received thoracic epidural anaesthesia with bupivacaine and
clonidine, started before surgery and continued for 5 days after surgery. The remaining 118
patients received target-controlled infusion of alfentanil for analgesia for the first 24 h after surgery,
followed by intravenous patient-controlled morphine analgesia for a further 48 h. Using
computerised patient medical records, we analysed the frequency of respiratory, neurological, renal,
gastrointestinal, haematological and cardiovascular complications in these two groups. New
arrhythmias requiring treatment occurred in 18% of the thoracic epidural anaesthesia group of
patients compared with 32% of the general anaesthesia group (p < 0.02). There was also a trend
towards a reduced incidence of respiratory complications in the thoracic epidural anaesthesia
group. The time to tracheal extubation was decreased in the epidural group, with the tracheas of
21% of the patients being extubated immediately after surgery compared with 2% in the general
anaesthesia group (p < 0.001). There were no serious neurological problems resulting from the use
of thoracic epidural analgesia.

Keywords Anaesthetic techniques, regional; epidural, thoracic. Surgery; coronary artery bypass grafts.
Complications; postoperative.

Correspondence to: Dr N. B. Scott
Accepted: 2 March 1997

In recent years there has been a growing interest in the
use of thoracic epidural anaesthesia for coronary artery
bypass surgery. Its potential advantages include excellent
analgesia [1], improved pulmonary function [2], early
tracheal extubation [2, 3] and cardiac protection as a
result of sympathetic blockade [4]. Thoracic epidural
anaesthesia decreases the stress response to sternotomy
and cardiopulmonary bypass. Increased sympathetic activ-
ity may lead to an increase in arterial pressure, tachycardia
and an imbalance between the myocardial oxygen demand
and supply, with increased myocardial oxygen extraction
and the possibility of ischaemic episodes. Moore et al.
showed that plasma concentrations of adrenaline and
noradrenaline did not increase in the first 24 h after cardiac
surgery in patients receiving thoracic epidural anaesthesia
compared with a conventional anaesthetic technique [5].
Other studies have shown that haemodynamic stability
was maintained during and after surgery using thoracic
epidural anaesthesia [6–9].

Thoracic epidural anaesthesia has been shown to
decrease pain and improve the endocardial to epicardial
blood flow ratio, thereby decreasing the number of
ischaemic episodes [10–12]. Thoracic epidural anaesthesia
has also been shown to decrease infarct size after coronary
artery occlusion in dogs [13] and has been used in patients
with unstable angina [11]. However, several concerns have
been raised about the use of thoracic epidural anaesthesia
in cardiac surgery, including the risk of haemodynamic
instability and the risk of epidural haematoma formation in patients who may be receiving aspirin and who will be fully heparinised during cardiopulmonary bypass [1].

Methods

A retrospective analysis of the peri-operative course of all patients who underwent routine coronary artery bypass graft surgery over a 9-month period was performed. There were 118 patients who received general anaesthesia alone (GA group) and 100 patients who received general anaesthesia together with thoracic epidural anaesthesia (TEA group). The decision to perform thoracic epidural anaesthesia was left to the individual anaesthetist. The patients were not formally randomised into either group. Written consent for surgery and anaesthesia was obtained from all patients and the anaesthetic technique was fully explained before surgery. Approval for the study was acquired from the local ethics committee. Premedication for all patients consisted of ranitidine 150 mg, metoclopramide 10 mg and temazepam 30 mg given orally on the evening before surgery and again 2 h before surgery. The same cardiac surgeon performed all of the procedures.

Thoracic epidural anaesthesia was performed immediately before induction of anaesthesia using an 18G epidural needle. The catheter was sited at the T2–3 or T3–4 interspace with the patient in the left lateral position. All patients had routine coagulation studies as part of the pre-operative investigation screen but bleeding time was not measured. Patients with abnormal coagulation studies were not given thoracic epidural anaesthesia but patients who were receiving aspirin were not excluded from the study. A block to the first thoracic dermatome was instituted using 4–12 ml of bupivacaine 0.5% and following induction of anaesthesia an infusion of bupivacaine 0.125% with clonidine 0.6 μg.ml⁻¹ was started. This infusion was continued for 5 days after surgery and the block height was maintained between the first and second thoracic dermatomes by adjusting the infusion rate.

General anaesthesia was then induced and maintained using target-controlled infusions of propofol and alfentanil, achieving initial plasma concentrations of 1.5–3 μg.ml⁻¹ and 100–200 ng.ml⁻¹, respectively. The target concentrations were adjusted as necessary. The patients’ lungs were ventilated following the administration of pancuronium 8 mg intravenously. Additional pancuronium was given as necessary.

The GA group of patients continued with a target-controlled alfentanil infusion after surgery (target blood concentration 80 ng.ml⁻¹) until they were able to use a patient-controlled target-controlled alfentanil infusion system. After 24 h this was changed to patient-controlled intravenous morphine with the following settings: 1 mg bolus, 5 min lockout period, no background infusion. All patients in this series received regular coproxamol and ibuprofen given orally for 7 days after surgery provided the plasma creatinine was normal and bleeding from the drains was minimal.

The fully computerised hospital patient medical record system was used to obtain the pre-operative and post-operative data for each patient. Data from the patient monitoring system (Tramscope 12C, Marquette, USA) were stored automatically during surgery by the Anaesthesia Information Management System (RECALL, Version 2, Informatics, UK) and later retrieved and analysed using Microsoft Excel Version 5.0. Postoperative data and patient demographic data were stored on the electronic hospital medical record (Cerner Corporation, Kansas, USA).

Age, sex, weight, New York Heart Association (NYHA) classification, pre-operative arrhythmias, number of grafts performed and ischaemic time on bypass were recorded. After surgery, we assessed the frequency of new arrhythmias, the time until tracheal extubation, the use of intra-aortic balloon pumps and ventricular assist devices, inotrope use and complications affecting the renal, gastrointestinal, neurological and respiratory systems. All patients underwent continuous ECG monitoring for the first 5 days after surgery. Extubation criteria were standardised and are shown in Table 1.

All complications during the first five postoperative days were recorded. Respiratory complications were divided into proven lower respiratory tract infection (i.e. with positive sputum culture), atelectasis or collapse assessed on postoperative chest X-ray, adult respiratory distress syndrome or respiratory failure requiring intervention such as re-intubation. Arrhythmias were classified as atrial flutter or fibrillation, conduction defects and ventricular arrhythmias. Postoperative myocardial infarction was diagnosed using a combination of ECG analysis and serum creatinine kinase levels. Renal failure was defined as oliguria combined with an increase in the serum creatinine to greater than twice the pre-operative value. Significant bleeding was defined as that which required treatment with blood

<table>
<thead>
<tr>
<th>Extubation criteria following routine coronary artery bypass graft surgery.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Cardiovascular stability without inotropes – systolic blood pressure of &gt; 90 mmHg</td>
</tr>
<tr>
<td>2. Core temperature over 36.5 °C</td>
</tr>
<tr>
<td>3. Spontaneous ventilation with PaO₂ &gt; 12 kPa on FiO₂ of &lt; 0.4 and PaCO₂ &lt; 7 kPa</td>
</tr>
<tr>
<td>4. Blood loss from chest drains &lt; 60 ml.h⁻¹</td>
</tr>
<tr>
<td>5. Urine output &gt; 1 ml.kg⁻¹.h⁻¹</td>
</tr>
<tr>
<td>6. No sedation and patient not in pain or agitated</td>
</tr>
</tbody>
</table>
products or a return to the operating theatre in order to control the haemorrhage surgically.

The data were analysed with the Chi-squared test and Fisher’s exact test using Minitab software (Minitab Ltd, UK).

Results

The two groups of patients were similar when compared for age, sex, NYHA score, weight, ejection fraction and the number of grafts performed (Table 2). There were no differences between the duration of bypass and ischaemic time. There was no difference between the two groups in the use of inotropes or intra-aortic balloon pump devices. No patients in the study period required a ventricular assist device. One patient in the TEA group and two in the GA group died whilst in the operating theatre as a result of intra-operative complications.

New arrhythmias requiring treatment occurred in 18% of patients in the TEA group compared with 32% in the GA group ($p = 0.02$). The majority of these arrhythmias were supraventricular in origin (Fig. 1). Respiratory complications occurred in 21% of the GA group and in 16% of the TEA group. Immediate extubation was defined as extubation within 1 h of return to ITU and was achieved in 21% of the TEA group compared with 2% of the GA group ($p < 0.002$) (Fig. 2). The median time to extubation was 12 h after arrival in the intensive care unit in both groups. Two patients in the TEA group and one in the GA group who were extubated immediately required re-intubation within the next 24 h.

There were no significant differences in the incidence of renal, haematological, neurological or wound complications between the two groups in the postoperative period (Table 3). Of the neurological complications that occurred, none was serious or caused long-term problems. In the TEA group six complications occurred. These included one transient ischaemic attack in the postoperative period, one brachial plexus lesion resulting in temporary weakness in the C7–8 distribution, one acute confusional state, one recurrent laryngeal nerve palsy, one frontal headache and one episode of weakness in the left leg. In the GA group of patients, nine neurological complications occurred. Five of these were acute confusional states, there was one brachial plexus lesion and three cerebrovascular accidents. There was no occurrence of epidural haematoma following epidural insertion in any

Table 2 Patient demographic data, duration of bypass and cardiac ischaemia and ejection fraction in the two groups of patients studied. Values are given as median (range) or mean (SD) where appropriate.

<table>
<thead>
<tr>
<th></th>
<th>TEA group</th>
<th>GA group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of grafts</td>
<td>3 (1–5)</td>
<td>3 (1–5)</td>
</tr>
<tr>
<td>Age; years</td>
<td>58.7 (16.9)</td>
<td>61.3 (14.2)</td>
</tr>
<tr>
<td>Sex ratio; M:F</td>
<td>6:1</td>
<td>5:7:1</td>
</tr>
<tr>
<td>NYHA Class</td>
<td>3 (2–4)</td>
<td>3 (2–4)</td>
</tr>
<tr>
<td>Weight; kg</td>
<td>77.4 (14.7)</td>
<td>71.3 (12.2)</td>
</tr>
<tr>
<td>Bypass time; min</td>
<td>84.8 (23.7)</td>
<td>84.3 (26.9)</td>
</tr>
<tr>
<td>Ischaemic time; min</td>
<td>47.6 (15.7)</td>
<td>53.1 (20.1)</td>
</tr>
<tr>
<td>Ejection fraction; %</td>
<td>45 (35–65)</td>
<td>45 (35–65)</td>
</tr>
</tbody>
</table>

Figure 1 Frequency and type of postoperative arrhythmia in the two groups studied.

Figure 2 Percentage of patients undergoing tracheal extubation in four time periods after surgery.
Table 3 Number and incidence (%) of postoperative complications in the two groups studied.

<table>
<thead>
<tr>
<th></th>
<th>TEA group (n = 100)</th>
<th>GA group (n = 118)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Arrhythmias</td>
<td>18 (18%)</td>
<td>*38 (32%)</td>
</tr>
<tr>
<td>Intra-aortic balloon pump</td>
<td>3 (3%)</td>
<td>7 (6%)</td>
</tr>
<tr>
<td>Postoperative myocardial infarction</td>
<td>1 (1%)</td>
<td>5 (4%)</td>
</tr>
<tr>
<td>Lower respiratory tract infection</td>
<td>7 (7%)</td>
<td>11 (9%)</td>
</tr>
<tr>
<td>Other infection</td>
<td>2 (2%)</td>
<td>7 (6%)</td>
</tr>
<tr>
<td>Neurological complication</td>
<td>6 (6%)</td>
<td>9 (8%)</td>
</tr>
<tr>
<td>Acute renal failure</td>
<td>6 (6%)</td>
<td>6 (6%)</td>
</tr>
<tr>
<td>Dialysis</td>
<td>0 (0%)</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Respiratory complication</td>
<td>16 (16%)</td>
<td>25 (21%)</td>
</tr>
<tr>
<td>Gastrointestinal tract complication</td>
<td>3 (3%)</td>
<td>3 (3%)</td>
</tr>
<tr>
<td>Significant bleeding</td>
<td>8 (8%)</td>
<td>10 (9%)</td>
</tr>
</tbody>
</table>

* Significantly different from GA group, p < 0.02.

patient. The patient with the weak left leg in the TEA group received urgent magnetic resonance imaging to exclude cord compression. No abnormality was found and the weakness resolved when the epidural infusion was discontinued.

In the TEA group, four patients required re-intubation of the epidural in the postoperative period because of inadequate block. One patient had a unilateral block, one epidural ‘fell out’ and the other two patients had no demonstrable block despite repeated bolus doses of bupivacaine. In addition, supplementary doses of intravenous morphine were required in 22 patients in the TEA group to provide analgesia either for leg wounds or for pain related to the chest drains. No patient who received morphine for chest drain pain had more than 10 mg up to the point at which the drains were removed. In all cases the removal of the drains led to relief of the pain. Four patients had acute torticollis on return from the operating theatre, presumably due to sternal retraction, which responded well to a 100 mg diclofenac suppository.

Discussion

Thoracic epidural anaesthesia following coronary artery surgery was first used in our hospital at the beginning of 1996 to provide rescue analgesia for four patients within 24 h of surgery in whom standard intravenous opioid analgesia was resulting in excessive sedation and poor respiratory function. None of these patients required re-intubation and we were encouraged to increase gradually the usage of the technique. All of the consultants in the department are now experienced in the technique and its postoperative management and we have reviewed all 218 patients who have undergone coronary artery surgery in 1996 in order to determine the need for a more comprehensive, large, prospective, randomised study.

Our retrospective data suggest that thoracic epidural anaesthesia is a safe adjunct to general anaesthesia for patients undergoing routine coronary artery bypass graft surgery. This technique may have significant benefits in terms of decreased postoperative morbidity, as demonstrated in this study by the decrease in new arrhythmias and a trend towards a decreased incidence of respiratory complications. Thoracic epidural anaesthesia has been shown previously to be associated with excellent cardiovascular stability [6–8] and our study showed no significant increase in the use of inotropic agents and intra-aortic balloon pump devices in the TEA group of patients, although a more powerful study may demonstrate an increase.

The data demonstrate a significant reduction in the incidence of arrhythmias in patients receiving thoracic epidural anaesthesia. This may be a result of the blockade of cardiac accelerator fibres, improvement of regional myocardial blood flow and attenuation of the stress response during surgery, in conjunction with postoperative sensory blockade preventing endocrine and autonomic responses to pain and trauma [5, 14]. The data support the view that patients receiving thoracic epidural anaesthesia are more likely to undergo early tracheal extubation. This has been shown previously by Joachimson et al. [15] and Liem et al. [2]. Earlier studies have shown that patients receiving thoracic epidural anaesthesia also have an increased PaO2 and lower pain and sedation scores in the postoperative period [2, 16]. Sedation and pain scores were not formally assessed in the present study.

The patients remained in ICU for a median of 2 days and were then transferred to a high-dependency area for a median of 2 days. They were then returned to a general ward for the remainder of the epidural infusion period and were supervised by our Acute Pain Service throughout their hospital stay. All patients in this series received regular coproxamol and ibuprofen orally for 7 days once the postoperative creatinine was normal, usually within 24 h of surgery. Any pain which developed in the epidural group during the 5 days was treated either by an epidural top-up of 4 mL bupivacaine 0.25%, with 2.5 mg morphine intravenously or 100 mg diclofenac orally or rectally.

The Peri-operative Ischaemia Randomised Anaesthesia Trial Study Group has shown that patients undergoing peripheral vascular surgery who required re-operation for graft thrombosis had high levels of circulating noradrenaline in the postoperative period [17]. A recent editorial by Desborough suggests similar effects may be seen in cardiac surgery [1]. In our study, the incidence of postoperative myocardial infarction was 1% (n = 1) in the TEA group and 4.2% (n = 5) in the GA group. Although only small groups of patients were studied, we consider this difference also warrants further investigation in prospective studies.

There was no significant difference in the frequency of
atelectasis and lower respiratory tract infection between the two groups, although the trend was for these complications to be decreased in the TEA group.

The use of thoracic epidural anaesthesia in anticoagulated patients undergoing cardiac surgery is controversial because of the risk of epidural haematoma formation. There are currently no accepted guidelines for the safe time period between insertion of thoracic epidural anaesthesia and full anticoagulation. In studies by Liem et al. [6, 15] and Joachimsson et al. [6, 15] the epidural catheter was sited 24 h before surgery in an attempt to decrease the risk. Kirno et al. sited the thoracic epidural anaesthesia 12 h before surgery in 10 patients with no complications noted [18]. El Baz & Goldin sited thoracic epidural anaesthesia catheters in 30 patients immediately before induction of anaesthesia with no neurological complications noted [19]. In our series, 100 catheters were sited immediately before surgery and we have noted no serious neurological complications. Furthermore, epidural anaesthesia has been used in 1000 anticoagulated patients with no permanent neurological complications [20]. A recent literature review noted that the frequency of haematoma formation associated with either spinal or epidural insertion in obstetric patients was 17 per 180 000 (≈1 in 10 000) [21]. The author of this review comments that as the frequency is so rare, the coincidental occurrence of spontaneous haematoma must be considered even when a neuraxial blockade has been performed.

In the postoperative setting, it is generally believed that outcome can be improved by good pain management and the beneficial effects of sympathetic blockade can also reduce morbidity [22, 23]. This study appears to support this belief. Surgery for coronary artery disease is performed in a high-risk population and carries a significant mortality, in excess of 2%. Thus, on an individual basis, since the exact risk of epidural haematoma is unknown and the published incidence is less than 0.01%, it seems inappropriate to deny patients the benefits of this technique provided that the putative benefits can be established in a large, prospective, randomised study.

References

FORUM

Comparison of nalbuphine and buprenorphine in total intravenous anaesthesia

F. A. Khan, A. Zaidi and R. S. Kamal

Department of Anaesthesia, The Aga Khan University Hospital, Stadium Road, PO Box 3500, Karachi-74800, Pakistan

Summary
Nalbuphine (0.3 mg.kg\(^{-1}\)) and buprenorphine (2.5 \(\mu\)g.kg\(^{-1}\)) were compared as part of a total intravenous anaesthesia regimen using a propofol infusion in 60 patients undergoing laparoscopic cholecystectomy in a randomised double-blind study. Changes in haemodynamic variables greater than 20% from the baseline were noted. No difference was observed in blood pressure but the heart rate was significantly lower in the buprenorphine group. Intra-operative bradycardia (heart rate <60 beat.min\(^{-1}\)) occurred more often in the buprenorphine group. Recovery was fast and comparable with both drugs and no patient reported awareness. Quality of analgesia was similar in both groups. Both drugs provide suitable analgesic supplementation to total intravenous anaesthesia.

Keywords Anaesthetics, intravenous; propofol. Analgesics; nalbuphine, buprenorphine.

Total intravenous anaesthesia (TIVA) is becoming increasingly popular, mainly because of the availability of newer anaesthetic drugs with short half-lives and advances in infusion pump technology which has made the administration of these drugs easier. Currently propofol is regarded as the most suitable anaesthetic agent for TIVA owing to its short duration of action, minimal side-effects and rapid recovery [1, 2]. It has been used in combination with fentanyl, alfentanil and sufentanil [3, 4] but little work has been done with propofol and partial agonist–antagonist narcotic combinations [5].

The objective of this study was to compare nalbuphine and buprenorphine in TIVA using propofol in patients undergoing laparoscopic cholecystectomy, comparing analgesic efficacy, haemodynamic stability, intra-operative complications and recovery profile in the two groups.

---

References

Methods

The study was approved by the Ethics Committee of the Aga Khan University Hospital and informed consent was obtained from the patients. The study was randomised and double-blinded. Sixty patients aged 20–60 years, ASA status 1 and 2, undergoing laparoscopic cholecystectomy were included in the trial. Patients with a history of hypertension, morbid obesity or possible difficult tracheal intubation were not studied. Those enrolled in the trial were randomly divided into two groups; group 1 received nalbuphine 0.3 mg.kg\(^{-1}\), and group 2 received buprenorphine 2.5 mg.kg\(^{-1}\), both intravenously, 5 min before induction of anaesthesia. The investigator was blinded to the analgesic drug by the use of coded syringes. The rest of the anaesthetic technique was standardised.

All patients received 7.5 mg of oral midazolam as premedication approximately 1 h prior to surgery. On arrival in the operating room, base-line observations were taken and the trial drug was administered. Five minutes later pre-oxygenation was started. Anaesthesia was induced with propofol 2 mg.kg\(^{-1}\) over 30 s followed by vecuronium 0.1 mg.kg\(^{-1}\) over 15 s. An infusion of propofol was started immediately after induction according to the following regimen: 10 mg.kg\(^{-1}\).h\(^{-1}\) for the first 10 min, 8 mg.kg\(^{-1}\).h\(^{-1}\) for the next 10 min and 6 mg.kg\(^{-1}\).h\(^{-1}\) thereafter, using an Imed-Gemini PC-1 infusion pump. Tracheal intubation was performed 3 min after vecuronium. Patients’ lungs were ventilated with an air–oxygen mixture maintaining an \(F_{1}\)\(_{\text{O}_2}\) of 0.4. A nasogastric tube was routinely inserted in all patients following tracheal intubation and removed before reversal of neuromuscular blockade.

Depth of anaesthesia was assessed intra-operatively by relying on signs of sympathetic and parasympathetic stimulation. Any variation of more than 20% above or below the baseline in systolic blood pressure or heart rate was noted. Lacrimation, sweating and any movements during anaesthesia were also noted. If any two of the above signs were seen, the rate of infusion was increased again to 8 mg.kg\(^{-1}\).h\(^{-1}\) for another 10 min. If the signs persisted for more than 3 min after giving the bolus, the rate of infusion was increased again to 8 mg.kg\(^{-1}\).h\(^{-1}\) for another 10 min. If the signs still persisted, half the pre-induction dose of analgesic was repeated from a coded syringe. Vecuronium supplementation was given as required, after the use of a nerve stimulator (Ministim). A train-of-four count of two was maintained during the surgical procedure. The ECG was monitored throughout using the CM3 lead. Noninvasive blood pressure, oxygen saturation, \(F_{1}\)\(_{\text{O}_2}\) and end-tidal CO\(_2\) were all monitored using the Datex Cardiocap monitor. The infusion of propofol was stopped at the time of the last suture and muscle relaxation was reversed with neostigmine 0.05 mg.kg\(^{-1}\) and atropine 0.02 mg.kg\(^{-1}\) given intravenously.

Blood pressure and heart rate were noted 5 min before and 5 min after the study drug, 1, 2 and 3 min after induction, every minute for 5 min after tracheal intubation, 1 and 2 min after decreasing the dose of propofol to 8 mg.kg\(^{-1}\).h\(^{-1}\) and then to 6 mg.kg\(^{-1}\).h\(^{-1}\), 1, 2 and 3 min after incision, and after reversal and extubation. The time interval between reversal and extubation was also noted.

In the immediate recovery period, time to eye opening to verbal command and time to correctly stating own name was noted. Patients were kept in the recovery room until they fulfilled our routine discharge criteria, i.e. stable vital signs for at least 30 min, maintaining a good airway, able to breathe deeply and cough, rousable or awake and moving purposefully. Any untoward effects were noted. The time of the first dose of analgesic required in the postoperative period was also noted.

All patients were visited the next day by one of the principal investigators and any complaints noted. At the postoperative visit every patient was questioned specifically regarding recall of operative events. Awareness was assessed by asking three specific questions: What is the last thing you remember before going to sleep? What is the first thing you remember after waking up? Do you remember anything between these events? Patients were also asked to give their opinion about the anaesthetic.

Variables were analysed using the Epi-info-6 statistical package. Analysis of variance was used to compare the mean changes in systolic, diastolic and mean blood pressure and heart rate. The incidence of untoward effects and other qualitative data was assessed by Chi-squared analysis. A p value of less than 0.05 was taken as significant.

Results

Demographic data

Both groups were comparable for age, weight, pre-induction blood pressure, heart rate and duration of anaesthesia (Table 1).

Haemodynamic data

Figure 1 shows the changes in systolic blood pressure (SBP) and diastolic blood pressure (DBP) in relation to time. Both groups showed a decrease in SBP and DBP in the first 3 min after induction which returned to near baseline values 1 min after intubation. This decrease was less than 20% of baseline, except with DBP in the nalbuphine group.

The maximum negative change seen was at 3 min after induction (19% drop in SBP in nalbuphine group and 16% in buprenorphine group). The fall in DBP was slightly more exaggerated, especially in the pre-intubation period.
in the nalbuphine group (24% vs. 17% in the buprenorphine group). No significant rise was associated with tracheal intubation, and the blood pressures remained 10–15% below the pre-induction baseline values during the maintenance period including directly after the surgical incision. No significant difference was observed at any time between the two groups.

Figure 2 shows the changes in heart rate which remained within 2–8% of the baseline values after induction. Tracheal intubation and surgical incision were accompanied by an insignificant rise (less than 5% in both groups). The maximum fall in heart rate was seen in the unstimulated intubated patient before surgical incision. A significant difference was observed in the heart rate between the two groups at 2, 3, 4 and 5 min after tracheal intubation (p < 0.1), in the unstimulated patient before incision (p < 0.01) and also in the readings taken after the surgical incision (p < 0.01), with the heart rate being lower in the buprenorphine group.

### Intra-operative sedative and analgesic drug requirements

Four patients in the nalbuphine group and three patients in the buprenorphine group required additional boluses of 10 mg of propofol. The same four patients in the nalbuphine group required a further increase in infusion rate compared to only two patients in the buprenorphine group. Only one patient in the nalbuphine group required a further analgesic top-up. These differences were not statistically significant.

### Untoward effects seen intra-operatively

Bradycardia was predefined as a heart rate below 60 beat. min⁻¹. Twenty per cent of patients in the nalbuphine group had bradycardia compared to 46% of patients in the buprenorphine group (p < 0.05). Three patients receiving buprenorphine required intravenous atropine for severe bradycardia (<50 beat. min⁻¹) accompanied by hypotension (systolic blood pressure <90 mmHg).

### Table 1 Demographic and baseline haemodynamic data. Mean (SD).

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Sex M:F</th>
<th>SBP</th>
<th>DBP</th>
<th>MAP</th>
<th>HR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nalbuphine</td>
<td>30</td>
<td>39 (10)</td>
<td>64 (10)</td>
<td>7:23</td>
<td>127 (17)</td>
<td>83 (10)</td>
<td>95 (12)</td>
<td>88 (16)</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>30</td>
<td>42 (10)</td>
<td>65 (12)</td>
<td>7:23</td>
<td>126 (14)</td>
<td>81 (8)</td>
<td>95 (12)</td>
<td>82 (10)</td>
</tr>
</tbody>
</table>

SBP, systolic blood pressure; DBP, diastolic blood pressure; MAP, mean arterial pressure; HR, heart rate.

Figure 1 Mean (SD) systolic (S) and diastolic (D) blood pressures during intravenous infusions of propofol/nalbuphine (●) or propofol/buprenorphine (O) at various times during anaesthesia.* Statistical significance (p < 0.05).
Recovery profile

Recovery profile is shown in Table 2. No significant difference was observed between the groups.

Untoward effects seen in the recovery room

Two patients in the buprenorphine group had heart rates between 50 and 60 beat.min\(^{-1}\) in the recovery room but did not require any treatment. No patient in the nalbuphine group had heart rate below 60 beat.min\(^{-1}\). One patient in the nalbuphine group complained of nausea and another vomited compared to one patient in the buprenorphine group who vomited. One patient in the nalbuphine group complained of vertigo during the recovery room stay. No significant difference was observed between the groups.

Extubation to first analgesic dose interval

Analgesia in the postoperative period was given on patient demand. After extubation analgesia was required after a mean period of 352 min in the nalbuphine group and 385 min in the buprenorphine group. This difference was not statistically significant. One patient in the nalbuphine group and five in the buprenorphine group did not require any postoperative analgesia.

Patient feedback

None of the patients complained of awareness in the intra-operative period at their postoperative interview. Forty per cent of patients in both groups graded anaesthesia as excellent and 60% graded it as good or satisfactory. Fourteen patients in each group (46%) experienced nausea or vomiting postoperatively, with the majority having one episode only.

Discussion

Combined infusions of propofol and an opioid were first reported in 1985 by DeGrood et al. [6] using fentanyl. The technique can be used in modern sophisticated situations or in places with the bare minimum of facilities and may have particular advantages in developing countries where there may be problems with availability of compressed

---

**Table 2** Recovery profile. Values are mean (SD) times (min).

<table>
<thead>
<tr>
<th></th>
<th>Reversal to extubation</th>
<th>Extubation to eye opening</th>
<th>Extubation to telling name</th>
<th>Extubation to first analgesic dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nalbuphine</td>
<td>3.2 (2.5)</td>
<td>5.0 (9.9)</td>
<td>22.9 (16.7)</td>
<td>352 (354)</td>
</tr>
<tr>
<td>Buprenorphine</td>
<td>3.36 (2.6)</td>
<td>4.46 (7.2)</td>
<td>29.4 (20.6)</td>
<td>385 (260)</td>
</tr>
</tbody>
</table>
Anaesthesia, 1997, 52, 1090–1113

Buprenorphine is considered to be 30–40 times more potent than morphine [18]. Its intra-operative use during anaesthesia has been reported with doses varying from 2.5 to 15 \( \mu \text{g.kg}^{-1} \) [13, 19, 20]. Most of the studies involving the higher dosages did not use inhalational supplementation. In our previous work using buprenorphine as a component in both balanced anaesthesia and in TIVA we found that 2.5 \( \mu \text{g.kg}^{-1} \) gave good haemodynamic stability with minimal side-effects in recovery [5, 19]. The doses of nalbuphine and buprenorphine used in our study were not equipotent but were based on the recommended dosage requirements in clinical anaesthesia practice.

Buprenorphine has been used as the narcotic component in TIVA [5]. Raftery has commented that its use may present difficulties in TIVA because of its poor response to opioid antagonists and its propensity for causing nausea [21]. Nalbuphine has been used in TIVA only for minor cases in patients undergoing procedures such as cystoscopy or dilatation and curettage [22].

Different regimens have been recommended for the use of propofol in TIVA. A blood propofol concentration of 3 \( \mu \text{g.ml}^{-1} \) combined with an opioid is said to provide adequate anaesthesia [1, 23]. We used Robert’s regimen with the slight alteration of a 2 \( \text{mg.kg}^{-1} \) induction dose instead of 1 \( \text{mg.kg}^{-1} \).

No difference was observed by us in the blood pressure response in the two groups, although the heart rate was significantly lower in the buprenorphine group. This might be of significance in patients with underlying ischaemic heart disease undergoing noncardiac surgery. The effect of buprenorphine on the heart rate may be exaggerated when it is combined with drugs such as propofol [24]. Tempia et al. found a 52% incidence of bradycardia with buprenorphine compared to 32% with fentanyl, probably due to buprenorphine’s morphine-like effect on the vagal nucleus [25]. In our study 46% of patients in the buprenorphine group experienced a fall in heart rate to below 60 beat.min\(^{-1}\) compared to only 20% in the nalbuphine group.

Supplemental doses of narcotic or propofol have both been found to be equally effective in controlling acute haemodynamic and hormonal responses to surgical stimuli during TIVA [26, 27]. In our study, a supplemental propofol bolus was required in 27% of patients in the nalbuphine group compared to 17% of patients in the buprenorphine group. One patient in the nalbuphine group required a further narcotic bolus. Buprenorphine therefore provided more profound intra-operative analgesia. To our knowledge no other study has compared buprenorphine and nalbuphine for intra-operative use in TIVA. This difference might be explained by the difference in the potency of the two drug dosages used. However, in a study by Pugh et al. comparing postoperative requirements for nalbuphine and buprenorphine with a conventional anaesthetic technique, more additional analgesia was required in patients given nalbuphine [28].

The recovery profile was the same in both groups, with no patient needing treatment for respiratory depression. In the recovery room symptomatic bradycardia (heart rate <60 beat.min\(^{-1}\) associated with SBP <90 mmHg) had to be treated with atropine in three patients in the buprenorphine group (10%). The other complication seen in the recovery room was nausea and vomiting. None of the patients had been given a prophylactic anti-emetic. A high incidence of nausea and vomiting has been reported in both laparoscopic and biliary procedures [29], which can be reduced by avoiding nitrous oxide. An incidence of nausea of 30–35% has been reported with buprenorphine anaesthesia by Carl [30], with a figure of 2–22% reported for nalbuphine [31]. The incidence of nausea and vomiting in the recovery room in our study was quite low, and no difference was observed in the emetogenic properties of the two drugs. One of the reasons for the low incidence of nausea and vomiting seen in the recovery room could have been the routine insertion of a nasogastric tube intra-operatively on surgical request. The tube was removed before reversal of anaesthesia. The use of propofol could also have contributed to this low incidence of vomiting.
Forty-six per cent of patients in each group vomited one or more times in the 24 h following surgery. All of these patients had received pethidine for postoperative analgesia on the ward. Seventeen per cent of patients in the nalbuphine group and 20% in the buprenorphine group had nausea/vomiting prior to receiving any pethidine. In the rest of the patients the contribution of pethidine to this high incidence cannot be ruled out.

Awareness was not reported by any of our patients, although we used indirect criteria for monitoring light anaesthesia. These indirect methods have been criticised [32] but no entirely reliable method or monitor for routine use has yet come into standard practice. Although awareness is a problem particularly highlighted in TIVA it has been pointed out that it is not specifically related to TIVA but to the use of muscle relaxants in general [30]. The reported incidence in TIVA is comparable to that reported with standard balanced anaesthesia techniques using muscle relaxants [33].

In conclusion, both drugs were found to be satisfactory for use in TIVA, in situations where short-acting narcotics which are used as an infusion are not available, and are able to provide adequate analgesia in combination with propofol. The major side-effect seen was intra-operative bradycardia requiring treatment with atropine in the buprenorphine group.

References

FORUM

Analgesic and respiratory effect of nalbuphine and pethidine for adenotonsillectomy in children with obstructive sleep disorder

W. Habre1 and B. McLeod2

1 Division of Paediatric Anaesthesia, Hospital des Enfants, 6, rue Willy Donze, 1211 Geneva 14, Switzerland
2 Department of Anaesthesia, Princess Margaret Hospital for Children, GPO Box D184, Perth WA 6001, Western Australia

Summary

Opioids may depress respiration and contribute to airway obstruction after adenotonsillectomy for obstructive sleep disorder. We compared the respiratory and analgesic effects of nalbuphine, which has a ceiling effect for respiratory depression, and pethidine in 90 children (aged 2–12 years) with a history of obstructive sleep disorder undergoing adenotonsillectomy. Children were scored for their obstructive sleep disorder history and were randomly allocated to receive intravenously at induction of anaesthesia either nalbuphine 0.1 mg.kg⁻¹ (group N) or pethidine 1 mg.kg⁻¹ (group P). End-tidal carbon dioxide was measured in the recovery period using a nasopharyngeal catheter and oxygen saturation whilst breathing air; pain and sedation scores were recorded for 6 h postoperatively. Both groups were similar with respect to the demographic data and respiratory measurements: mean (SD) oxygen saturation on air in the recovery area (96.2% (1.2) vs. 96.5% (1.1) in group N and P, respectively) and mean (SD) end-tidal carbon dioxide (46.4 (5.5) mmHg vs. 47.7 (4) mmHg in group N and P, respectively). High obstructive sleep disorder score, history of apnoea, hyperactivity and loud snoring were found to be the best predictors of early postoperative oxygen desaturation in both groups.

Keywords Analgesics; nalbuphine; pethidine. Complications; sleep apnoea syndromes. Surgery; paediatric; adenotonsillectomy.

Correspondence to: Dr W. Habre
Accepted: 14 April 1997
Tonsillectomy or adenotonsillectomy in children for the treatment of obstructive sleep disorder is increasing [1]. The most common serious postoperative complication following this procedure is upper airway obstruction [2, 3]. Many factors may contribute including mechanical swelling, disruption of pharyngeal receptors from surgery and the effects of general anaesthesia [4]. There is also concern that opioids may depress respiration and precipitate airway obstruction after surgery. Nalbuphine, an agonist/antagonist opioid analgesic, has been shown to provide effective analgesia [5] and to possess a ceiling effect for respiratory depression [6, 7]. These properties may make nalbuphine a useful and safe analgesic for children undergoing tonsillectomy or adenotonsillectomy for obstructive sleep disorder.

We compared the analgesic and respiratory depressant effects of intravenous pethidine and nalbuphine for tonsillectomy or adenotonsillectomy in children with a history of obstructive sleep disorder.

**Methods**

After institutional Ethics Committee approval and parental informed written consent, we studied 90 children (ASA physical status 1 or 2), aged 2–12 years. All children were in-patients scheduled for tonsillectomy or adenotonsillectomy with a history of varying degrees of obstructive sleep disorder. Children with craniofacial abnormalities, previous upper airway trauma, failure to thrive or cor pulmonale were not studied.

Parents were questioned about their child’s symptoms. Severity of obstructive sleep disorder was scored using the presence or absence of each of the following symptoms: snoring, apnoea, nocturnal sweating, nocturnal awaking, restlessness during sleep, odd sleeping position, feeding problems or daytime hyperactivity. A score of 0 was given if the symptom was absent. Snoring was scored as either 1 and considered as minor (when the parents must be in the same room to hear it), or scored 2 and considered as loud (when the parents can hear it from outside their child’s bedroom). All other symptoms were scored as 1, if present. Apnoea was defined as the occurrence of breath-holding during the child’s sleep which was of concern to the parents. Odd sleeping position was recorded if the child’s head was noted to be in hyperextension during sleep.

All children were given paracetamol 15 mg.kg\(^{-1}\) orally and had topical anaesthetic cream (EMLA\textsuperscript{TM}) applied to both hands 1 h before surgery. Anaesthesia was induced with thiopentone 5 mg.kg\(^{-1}\) intravenously and maintained with halothane and nitrous oxide 70% in oxygen. Atracurium 0.5 mg.kg\(^{-1}\) was given to facilitate tracheal intubation. Children were then randomly assigned to receive on induction either nalbuphine 0.1 mg.kg\(^{-1}\) intravenously (Group N) or pethidine 1 mg.kg\(^{-1}\) intravenously (Group P). These opioids were prepared by another anaesthetist who was not involved in the procedure. Further intra-operative analgesic supplements were not given and muscle relaxation was reversed prior to tracheal extubation with atropine 0.02 mg.kg\(^{-1}\) and neostigmine 0.05 mg.kg\(^{-1}\). At the end of the procedure, and prior to tracheal extubation, the surgeon introduced a 10F feeding tube in the nasopharynx to allow end-tidal carbon dioxide measurement in the recovery period. The tube tip was positioned under direct vision to be visible in the oropharynx just below the soft palate.

Heart rate, arterial blood pressure and administered halothane concentration were recorded every 3 min during the surgical procedure. In the recovery area, oxygen saturation on air, end-tidal carbon dioxide, respiration rate, heart rate and sedation score (1 = alert, 2 = alert, calm, 3 = drowsy, sleeping but easily awake and 4 = asleep, no response to minor stimuli) were recorded on arrival and every 15 min. Oxygen saturation and end-tidal carbon dioxide were recorded on a regularly calibrated gas analyser (OSCAROXY\textsuperscript{TM}, Datex) which displays a capnograph. The same monitor was used for all children. End-tidal carbon dioxide was sampled continuously in the recovery area and values were recorded after 1 min of a steady-state capnograph trace. Pain was also assessed at the same intervals using our institution pain scoring system (PMH pain score), which is based on four criteria (facial expression, position in bed, vocalisation and nurse’s assessment) [8]. Each criterion was scored 0–2 making a maximum pain score of 8. A total score of less than 4 was considered to indicate adequate analgesia. If analgesia was required, children received the same opioid as given on induction from a syringe labelled with their name. Recovery nursing staff were therefore blinded to the choice of opioid. The children thus received either nalbuphine 25 \(\mu\)g.kg\(^{-1}\) or pethidine 250 \(\mu\)g.kg\(^{-1}\) intravenously every 3 min if required.

On the ward, the same variables, except end-tidal carbon dioxide, were recorded every 30 min for the first 2 h then hourly for a further 4 h. The incidence of vomiting and the time of first analgesic administration were also recorded.

Groups of 45 subjects can been shown to have, respectively, 80% and 90% power to detect a 30% difference in pain score less or more than 4 and a 1% difference in mean oxygen saturation breathing air in the recovery area between the two groups. Parametric data were compared using unpaired two-tailed \(t\)-tests. Chi-squared analysis was used to compare sedation scores at each time. Linear regression analysis was used to compare the effect of obstructive sleep disorder scores on postoperative oxygen.
saturation and end-tidal carbon dioxide in each group. A p value < 0.05 was considered as statistically significant.

**Results**

There was no difference between the two groups with respect to age, weight, gender, number of children undergoing tonsillectomy or adenotonsillectomy and duration of surgery (Table 1). The symptoms of sleep disorder were also comparable in both groups (Fig. 1). Although most of the children snored, only 44% of them had a history of apnoea noted by parents.

**Analgesia**

Haemodynamic data during surgery were comparable between groups (heart rate: 107 (14) beat.min⁻¹ in group N vs. 105 (16) beat.min⁻¹ in group P; systolic blood pressure: 109 (11) mmHg in group N vs. 117 (13) mmHg in group P; diastolic blood pressure: 65 (11) mmHg in group N vs. 65 (13) mmHg in group P) and there was no difference in halothane requirements (0.84 (0.25)% in group N vs. 0.85 (0.27)% in group P).

In the recovery area, children in group N had higher pain scores only at 15 min after admission (Table 2). In addition, they required more doses of analgesia during their stay in the recovery area (1.9 (1.5) doses in group N vs. 1.2 (1.2) in group P (p = 0.012)). Eighteen children in group N and six children in group P required three or more additional doses of opioid. However, there was no statistical difference in heart rate and sedation scores (Table 2).

On the ward, there was no difference in pain scores between the two groups. Although children in group N required additional analgesia later than children in group P, this difference did not reach statistical significance. Furthermore, there was no difference between them with respect to the requirement of further opioid administration (Table 3).

**Respiration**

In the recovery area, the difference between the two groups in mean oxygen saturation on air and mean end-tidal carbon dioxide reached statistical significance at 15 min. However, respiration rate, mean oxygen saturation on air and mean end-tidal carbon dioxide were comparable between the two groups at all other periods of measurement (Table 2). Moreover, total mean oxygen saturation breathing air during all the stay in the recovery area (96.2 (1.2)% vs. 96.5 (1.1)% in group N and P, respectively) and total mean end-tidal carbon dioxide

---

**Table 1** Demographic data. Results are expressed as number, mean (SD) or median [interquartile range].

<table>
<thead>
<tr>
<th></th>
<th>Nalbuphine</th>
<th>Pethidine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Surgery; adenotonsillectomy: tonsillectomy</td>
<td>43.2</td>
<td>40.5</td>
</tr>
<tr>
<td>Age; months</td>
<td>67 (29)</td>
<td>76 (29)</td>
</tr>
<tr>
<td>Gender; male:female</td>
<td>26:19</td>
<td>18:27</td>
</tr>
<tr>
<td>Weight; kg</td>
<td>20.8 (6.7)</td>
<td>23.9 (8.1)</td>
</tr>
<tr>
<td>Duration of surgery; min</td>
<td>17.1 (7.3)</td>
<td>17.1 (8)</td>
</tr>
</tbody>
</table>

---

**Figure 1** Distribution of obstructive sleep disorder symptoms within subjects.
Using a linear regression analysis, low oxygen saturation breathing air in the recovery period (less than 95%) was strongly associated with a high obstructive sleep disorder score in both groups (p < 0.01). Low oxygen saturation on air in the recovery period was significantly associated only with a history of loud snoring, apnoea and hyperactivity (Table 4). However, there was no correlation between high values of end-tidal carbon dioxide and obstructive sleep disorder. There was also no correlation between high end-tidal carbon dioxide and low oxygen saturation. Both were comparable in children who received three or more doses of opioids and those who did not.

On the ward, there was no statistical difference in mean oxygen saturation breathing air and respiration rate (Table 3). We did not observe any obstructive event or need for supplemental oxygen during the study period.

**Discussion**

This study shows that nalbuphine and pethidine have similar respiratory effects in children with a history of obstructive sleep disorder undergoing tonsillectomy or adenotonsillectomy. Furthermore, this study provides information on the effect of the different obstructive sleep symptoms on the risk of postoperative oxygen desaturation. The ideal study would have included pre-operative polysomnography to confirm and document the severity of the obstructive sleep disorder and correlate this with the patient’s history and the postoperative measurements. However, a role for routine polysomnography prior to adenotonsillectomy in children has yet to be established and the economic impact of such a practice observed in both groups in the recovery area and on the ward. Two patients in group N may have experienced psychomimetic effects, one child appeared to be frightened and another complained of bad dreams. The incidence of postoperative vomiting was identical in both groups (58%) during the study period.

**Table 4** Linear regression analysis for single variable models of obstructive sleep disorder risk factors associated with oxygen saturation on air less than 95% in the recovery area.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Coefficient</th>
<th>SE</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hyperactivity</td>
<td>-0.68</td>
<td>0.27</td>
<td>p = 0.012</td>
</tr>
<tr>
<td>Apnoea</td>
<td>-0.55</td>
<td>0.25</td>
<td>p = 0.028</td>
</tr>
<tr>
<td>Loud snoring</td>
<td>-0.54</td>
<td>0.27</td>
<td>p = 0.046</td>
</tr>
<tr>
<td>Odd sleeping position</td>
<td>-0.43</td>
<td>0.26</td>
<td>p = 0.1</td>
</tr>
<tr>
<td>Feeding problems</td>
<td>-0.41</td>
<td>0.27</td>
<td>p = 0.14</td>
</tr>
<tr>
<td>Nocturnal awaking</td>
<td>-0.37</td>
<td>0.26</td>
<td>p = 0.16</td>
</tr>
<tr>
<td>Sweating</td>
<td>-0.35</td>
<td>0.25</td>
<td>p = 0.17</td>
</tr>
<tr>
<td>Restless sleeping</td>
<td>-0.26</td>
<td>0.26</td>
<td>p = 0.32</td>
</tr>
<tr>
<td>Overall: obstructive sleep disorder score</td>
<td>-0.17</td>
<td>0.05</td>
<td>p = 0.002</td>
</tr>
</tbody>
</table>

---

* p < 0.05.
would be significant. In practice, most doctors rely on data such as medical history, physical examination and intraoperative findings to assess risk and determine anaesthetic technique and postoperative management.

Postoperative respiratory complications after tonsillectomy or adenotonsillectomy are multifactorial and opioids may further compromise respiratory patterns [4]. A ceiling effect for respiratory depression has been demonstrated for nalbuphine [7] which could have been of importance in children with obstructive sleep disorder, as they may already have impaired ventilatory responses to carbon dioxide [9].

We used a dose of nalbuphine 0.1 mg.kg\(^{-1}\) at the induction of anaesthesia which is considered to be equianalgesic to morphine 0.1 mg.kg\(^{-1}\) [10] and pethidine 1 mg.kg\(^{-1}\). The mean blood pressure, heart rate and halothane requirements during the procedure were comparable in both groups, further suggesting that nalbuphine 0.1 mg.kg\(^{-1}\) is equipotent to pethidine 1 mg.kg\(^{-1}\). However, although respiratory and heart rates were comparable, pain scores were different 15 min after arrival in the recovery area and children in the nalbuphine group required more analgesia despite comparable sedation scores. Furthermore, the number of patients who required further analgesia in the recovery area was comparable in both groups (31 in group N vs. 26 in group P).

Pain was assessed using the routine pain tool at our institution [8] and our policy is to give intravenous analgesia if the pain score is 4 or more in the recovery area. The reasons for higher pain scores at one stage in the nalbuphine group may be multifactorial. These may include the possible psychotomimetic effects of nalbuphine, the higher incidence of intolerance to the nasopharyngeal catheter and the possibility that nalbuphine at a dose of 0.1 mg.kg\(^{-1}\) was less potent than expected.

The monitoring of end-tidal carbon dioxide through a nasal cannula and its correlation to arterial carbon dioxide tension has already been validated in children [11]. However, this method could be limited by entrainment of room air or by mouth breathing particularly in the presence of a Guedel airway. We modified this technique by using a nasopharyngeal catheter which samples the gas from the posterior nasopharynx [12]. This decreases dead space and also allows suction of the airway if postoperative bleeding occurs. Thirteen out of the 90 children were upset by the presence of the nasopharyngeal catheter; two of them pulled out their catheter immediately on arrival in the recovery area and 11 after 20 min. However, in the remaining children, we were able to obtain a normal capnograph tracing with consistent measurements within 1 min. We observed, 15 min after arrival in the recovery area, a significant difference between the two groups in the mean value of end-tidal carbon dioxide. However, this slight difference could not be considered as clinically relevant, and similarly for the difference in oxygen saturation. Furthermore, there was no correlation between the severity of the symptoms of obstructive sleep disorder and the values of end-tidal carbon dioxide in the recovery area. Although airway obstruction or alveolar hypoventilation may induce an elevation in arterial carbon dioxide, our measurements through a nasopharyngeal catheter would have detected it, as demonstrated during polysomnography in children [13]. Recently, children with sleep-induced respiratory obstruction were shown to have higher end-tidal carbon dioxide then nonobstructers before surgery, but this difference disappeared after adenotonsillectomy [14]. In our study, mean end-tidal carbon dioxide in the recovery area was also similar in children with and without a history of loud snoring and apnoea.

A ceiling effect for respiratory depression has been reported after repeated doses of nalbuphine [6]. Eighteen patients in group N required three or more doses of analgesic in the recovery area compared with only six in group P. Despite this small number, we did not find a difference in end-tidal carbon dioxide and oxygen saturation on air in the recovery area between children who received repeated doses of opioids and those who did not.

A simple linear regression analysis was carried out using the medical history of obstructive sleep disorder as a risk factor for low values of oxygen saturation in the recovery area (less than 95%). In our model including all symptoms of obstructive sleep disorder a high score was significantly associated with postoperative oxygen desaturation. The symptoms of obstructive sleep disorder considered in our study were similar to those found in selected patients with sleep apnoea [15, 16]. Considering each symptom, we found only apnoea, loud snoring and hyperactivity to be significantly associated with low oxygen saturation in the recovery area.

Stradling et al. found pre-operative hyperactivity in 60% of the children with obstructive sleep disorder which significantly decreased to less than 10% 6 months after the operation [15]. In our study, 30% of the children were considered by their parents to have pre-operative daytime hyperactivity. Children less than 4 years old were found to be at high risk of postoperative respiratory compromise because of anatomical factors such as small mandible and large tongue [3]. Although 25% of the children in our study were 4 years old or younger, only three of them had oxygen saturation on air in the recovery area of less than 95%.

In conclusion, this study shows that postoperative sedation and respiratory patterns were similar after nalbuphine or pethidine. In addition, our model showed that the severity of obstructive sleep disorder could be suggested by the medical history and predict a risk of
postoperative oxygen desaturation. Among all risk factors, a history of apnoea, loud snoring and daytime hyperactivity should be considered when assessing these patients as these symptoms were more important determinants of postoperative respiratory problems than the choice of opioid. Pre- and postoperative polysomnography in a selected group of children could more accurately delineate the risk factors for postoperative oxygen desaturation.

References


FORUM

The site of airway irritation during induction of anaesthesia

A. K. Dashfield, S. E. Bree, A. M. Weiss and J. A. Langton*

Department of Anaesthesia, Derriford Hospital, Derriford, Plymouth PL6 8DH, UK

Summary

The aim of this investigation was to study the role of the nasal airway in mediating upper airway reflexes during induction of anaesthesia when the commonly used irritant inhalational anaesthetic agent enflurane is used. In a prospective randomised study, 40 ASA 1 & 2 day-case patients undergoing body surface surgery were recruited. Following intravenous induction using propofol,
20 patients received enflurane administered via a laryngeal mask airway (LMA), the anaesthetic vapour therefore bypassing the nasal airway. In the other group, 20 patients received enflurane anaesthesia administered using a face mask, the nasal airway therefore being exposed to inhalation anaesthetic. We were unable to demonstrate any significant (p < 0.05) differences between the two groups in relation to upper airway complications (cough, breath holding, laryngeal spasm, bronchospasm and excitement). Previous work has identified the nose as a possible important reflexogenic site for upper airway reflexes in humans during anaesthesia. We have been unable to demonstrate any difference in upper airway complications when the nasal airway was included or excluded from exposure to irritant anaesthetic vapours, when administered in a clinical setting.

**Keywords** Complications; airway irritation. Anaesthesia; induction.

It is generally accepted that inhalational anaesthetic agents may cause irritation to the upper airway when inhaled, as demonstrated by coughing, laryngospasm and breath holding [1]. This may impair the smooth induction of anaesthesia and lead to airway obstruction and associated hypoxaemia and hypercarbia, which may in some cases be associated with serious complications.

A recent study [2] has proposed that the site involved in mediating upper airway reflexes to inhaled anaesthetic vapours is located within the nasal airway. Previous work has shown that stimulation of the nasal mucosa can cause various airway reflexes in humans [3–6]. Nasal irritation more commonly causes apnoea rather than a sneeze under experimental conditions. The apnoeic reflex is part of the complex diving response [7], the physiological stimulus being water applied to the face or into the nose. Associated with the apnoea are cardiovascular changes and complete laryngeal closure occurs as part of the diving response [8]. The receptors that initiate the apnoeic reflex have not been identified but probably include nerve endings in the nasal mucosa. It is unlikely, however, that the diving reflex is initiated by a single modality of receptor. Nasal irritant stimuli that mimic the diving response also close the larynx by the same nervous pathways [9]. The nature and strength of the stimulus used determines to some extent the type of reflex response obtained.

Enflurane has been found to be especially irritant to the upper airway causing an increase in laryngeal wall tension and prolongation of expiratory time during nasal insufflation [2]. If the site which modulates upper airway reflexes during induction of anaesthesia were to be identified, therapies could be designed to reduce and eliminate these problems. It may also be possible to identify particular chemical structures involved in causing irritation to the upper airway during induction of anaesthesia. This may be of particular importance in paediatric anaesthesia, where the incidence of upper airway complications is known to be higher and may rapidly lead to life-threatening complications.

To investigate this question, we performed a prospective randomised, controlled study of enflurane anaesthesia administered via a laryngeal mask airway (LMA), thus bypassing the nasal airway compared with enflurane anaesthesia administered using a face mask and Guedel airway, therefore including the nasal airway.

**Methods**

Following local Ethics Committee approval, patients presenting for routine day-case, body surface surgery were recruited into this study. They were ASA 1 or 2, aged 18–65 years, nonsmokers and had no history of asthma, nasal polyps or upper respiratory tract infection within the previous 2 weeks. Patients with risk factors for regurgitation of gastric contents were not studied. Written informed consent was obtained prior to participating in the study. No premedication was administered and, prior to anaesthesia, patients were randomly allocated to one of two groups according to a sealed envelope random number system.

A standardised general anaesthetic technique was used. Anaesthesia was induced using propofol administered via an Ohmeda 9000 syringe pump (Ohmeda, Steeton, West Yorks., UK) set at an infusion rate of 1200 ml.h⁻¹ following formal pre-oxygenation for 2 min. The end-point of induction was judged to be when the patient dropped a 20 ml water-filled syringe [10] held between thumb and index finger of the right hand. In the laryngeal mask airway (LMA) group the airway was then inserted in the recommended manner [11] and anaesthesia was maintained using 2% enflurane in 66% nitrous oxide in oxygen using a Mapleson D breathing system. The
The concentration of enflurane was increased by 0.5% every 30 s in the spontaneously breathing patient until 5% was reached. In the face mask group a Guedel airway was inserted following intravenous induction and anaesthesia was maintained in the same manner using a close-fitting black rubber Ohmeda anaesthetic face mask.

The anaesthetic induction was scored for upper airway complications until 5% enflurane with 66% nitrous oxide in oxygen was reached. The incidence and severity of the following upper airway complications were scored: (i) coughing, (ii) breath holding after spontaneous ventilation had commenced, (iii) laryngeal spasm, (iv) bronchospasm and (v) excitement. We defined excitement as spontaneous movement of the head, arms or legs by the patient during induction. Breath holding was defined as periods of apnoea in the absence of upper airway obstruction measured from after the commencement of spontaneous ventilation, as initial apnoea postinduction was likely to have been due to the respiratory depressant effect of propofol.

These complications were each scored using a four-point scale, none, occasional, frequent and continuous, and then pooled to obtain a total score. We defined occasional coughing as up to two coughs, frequent up to 10 coughs and continuous more than 10 coughs during the induction period. Mild laryngospasm was defined as lasting 15 s or less with the patient able to maintain adequate spontaneous ventilation. Moderate laryngospasm lasted greater than 15 s but could be overcome with continuous positive airways pressure. Complete laryngospasm was defined as inability to ventilate the patients’ lungs requiring muscle relaxation to restore a clear airway. The total complication score could therefore range from a minimum of 5 to a maximum of 20 points (Table 1). This scoring system has been used in previous work [12] investigating upper airway complications during induction of anaesthesia.

In a previous study [2] nasal insufflation of enflurane triggered upper airway reflexes in 46% of patients. We calculated that 20 patients would be required in each group to give the study a power of >0.75 [13]. Statistical analysis of our nonparametric data was performed using the Macintosh Instat version 2.01 statistical computer software program. The Chi-squared test was applied to individual upper airway complication scores and the Mann–Whitney U-test to total upper airway complication scores between both groups. A p value of < 0.05 was considered statistically significant.

Results
Forty patients were successfully enrolled into the study. Table 2 shows the demographic data, both groups being closely matched for weight, age and gender.

Statistical analysis revealed that there were no significant differences between the two groups in relation to individual upper airway complications (cough, breath holding, laryngeal spasm, bronchospasm or excitement) or in the total upper airway complication scores (Table 3).

Table 1 Scoring system for upper airway complications during induction of anaesthesia.

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough</td>
<td>None</td>
<td>Occasional</td>
<td>Frequent</td>
<td>Continuous</td>
</tr>
<tr>
<td>Breath holding</td>
<td>None</td>
<td>&lt; 15 s</td>
<td>15–30 s</td>
<td>&gt; 30 s</td>
</tr>
<tr>
<td>Laryngeal spasm</td>
<td>None</td>
<td>Mild laryngospasm</td>
<td>Moderate laryngospasm</td>
<td>Complete laryngospasm</td>
</tr>
<tr>
<td>Bronchospasm</td>
<td>None</td>
<td>Expiration only</td>
<td>Inspiration and expiration</td>
<td>Difficult to ventilate</td>
</tr>
<tr>
<td>Excitement</td>
<td>None</td>
<td>&lt; 30 s</td>
<td>30–60 s</td>
<td>&gt; 60 s</td>
</tr>
</tbody>
</table>

Table 2 Demographic data.

<table>
<thead>
<tr>
<th></th>
<th>Laryngeal mask group</th>
<th>Face mask group</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of patients</td>
<td>20</td>
<td>20</td>
</tr>
<tr>
<td>Age [median (range)]; years</td>
<td>41.8 (18–59)</td>
<td>40.5 (19–65)</td>
</tr>
<tr>
<td>Sex (M/F)</td>
<td>12/8</td>
<td>9/11</td>
</tr>
<tr>
<td>Weight [mean (SD)]; kg</td>
<td>75.4 (14.1)</td>
<td>72.1 (13.3)</td>
</tr>
</tbody>
</table>

Table 3 Upper airway complication scores for individual airway complications and total scores for each group. Median (range).

<table>
<thead>
<tr>
<th></th>
<th>Laryngeal mask group (n = 20)</th>
<th>Face mask group (n = 20)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cough</td>
<td>1 (1–3)</td>
<td>1 (1–4)</td>
</tr>
<tr>
<td>Breath holding</td>
<td>1 (1–4)</td>
<td>2 (1–3)</td>
</tr>
<tr>
<td>Airway obstruction</td>
<td>1 (1–2)</td>
<td>1 (1–3)</td>
</tr>
<tr>
<td>Bronchospasm</td>
<td>1 (1)</td>
<td>1 (1)</td>
</tr>
<tr>
<td>Excitement</td>
<td>1 (1–3)</td>
<td>2 (1–4)</td>
</tr>
<tr>
<td>Total score</td>
<td>122</td>
<td>151</td>
</tr>
</tbody>
</table>
Discussion

In recently published work by Nishino and co-workers, it has been suggested that the nasal airway plays an important role in mediating the reflex response of the upper airway to irritant anaesthetic vapours [2]. The aim of this study was to investigate this hypothesis in the clinical setting. The investigation was designed to exclude the nasal airway in one group from exposure to anaesthetic vapour during induction by the use of the laryngeal mask airway. The laryngeal mask airway, by conducting the anaesthetic gases directly to the larynx, effectively excludes the nasal airway from exposure to anaesthetic vapours. In the face mask group the nasal airway will be exposed to anaesthetic vapour. It was not possible to blind this study as there was no means of disguising whether the laryngeal mask or face mask was being used whilst observing the patient and scoring upper airway complications.

Although the laryngeal mask may have caused direct physical stimulation during insertion we felt that this was not significantly greater than the stimulation caused by insertion of a Guedel airway. Previous work found no difference in the cardiovascular response of laryngeal mask insertion when compared with Guedel airway insertion [14].

We attempted to standardise the induction as far as possible and we chose to use enflurane as this had been previously identified as the most irritant anaesthetic vapour to the nasal airway [2]. It would therefore have been most likely to reveal any differences between the two groups. Patients with blocked noses resulting from upper respiratory tract infections or polyps were excluded from entering the trial. Although the percentage of fresh gas flow taken through the nose in the Guedel airway and face mask group is unknown it is likely that, during the 3 min trial period, anaesthetic gas containing enflurane will have diffused into the nasal passages. Previous work identified the position of an apparently well positioned Guedel airway to be very variable with a high incidence of partial or complete obstruction [15]. It seems likely that the ratio of nasal to oral fresh gas flow will similarly vary.

Our results did not demonstrate a difference between the two groups in relation to upper airway complications occurring during induction of anaesthesia. The possibility of a type 2 error cannot be discounted, however, as our study only had a relatively low power of 0.75.

There were several important differences in methodology between our study and that employed in previous work. The previous workers utilised a double-cuffed tracheal tube designed to measure laryngeal and tracheal cuff pressures. We chose to measure clinical indicators of upper airway complications and not solely movement of vocal cords, tidal volume, inspiratory time and expiratory time. There were also differences in the method of delivering enflurane. We introduced a relatively low concentration of anaesthetic vapour, which was increased in a standardised incremental manner. This is in keeping with the techniques most frequently employed in clinical anaesthetic practice.

In the previous work the nasal airway was exposed by direct insufflation of 5% enflurane in oxygen. This may have produced very high local concentrations of inhalational agent within the nasal airway.

In the LMA group in our investigation the larynx and laryngeal inlet would have been exposed to anaesthetic vapour directly. The larynx is richly supplied with sensory receptors mediating upper airway complications including laryngospasm and coughing. It is possible that this area of the upper airway provides an equal contribution to airway problems occurring during induction of anaesthesia.

In our investigation we used propofol as the induction agent. This is in contrast to the previous work [2] where flunitrazepam (0.04 mg.kg\(^{-1}\)) and pentazocine (0.03 mg. kg\(^{-1}\)) were used. It is known that propofol has a depressant effect on upper airway reflexes [16–20]. However, in this study propofol was used in both groups and therefore would be expected to exert an equal effect.

In conclusion, although previous work has proposed that the nasal airway may be an important reflexogenic site mediating upper airway reflexes in humans during anaesthesia, we have been unable to demonstrate a difference in the incidence of upper airway complications when the nasal airway was included or excluded from exposure to inhalational anaesthetic agents in a clinical setting.

References

8. Banting FG, Hall GE, James JM, et al. Physiological studies...


---

**FORUM**

**Comparison of caudal block using bupivacaine and ketamine with ilioinguinal nerve block for orchidopexy in children**

D. Findlow, L. M. Aldridge and E. Doyle

*Department of Anaesthesia, Royal Hospital for Sick Children, Sciennes Road, Edinburgh EH9 1LF, UK*

**Summary**

Forty boys weighing less than 25 kg undergoing unilateral orchidopexy were randomly allocated to receive one of two analgesic regimens. Group C received a caudal epidural block with 0.25% bupivacaine 1 ml kg\(^{-1}\) and preservative-free ketamine 0.5 mg kg\(^{-1}\); Group L received an ilioinguinal nerve block with 0.25% bupivacaine 0.5 ml kg\(^{-1}\) and infiltration of the wound with 0.25% bupivacaine 0.5 ml kg\(^{-1}\). All subjects received diclofenac sodium 1–2 mg kg\(^{-1}\) as a rectal suppository. Postoperative pain was assessed by means of a modified Objective Pain Score and analgesia was administered if this exceeded a value of 4. The median duration of analgesia was 10 h (range 2.6 to >24 h) in Group C and 2.9 h (range 0.7 to >24 h) in Group L (p < 0.05). There were no differences between groups in the incidence of motor block, urinary retention, postoperative vomiting or postoperative sedation. Subjects in Group L required significantly more doses of postoperative analgesia than those in Group C (p < 0.05).

**Keywords** Anaesthesia; paediatric. Pain; postoperative. Anaesthetic techniques, regional; caudal, ilioinguinal nerve block.

---

**Correspondence to:** Dr D. Findlow

**Accepted:** 14 April 1997

© 1997 Blackwell Science Ltd
Caudal epidural blockade is a common local anaesthetic technique in paediatric anaesthesia. When used for caudal analgesia in children, bupivacaine 2.0–2.5 mg.kg$^{-1}$ has a duration of action of 2–4 h. Over 60% of children undergoing orchidopexy with this technique require further analgesia during the postoperative period [1].

It has previously been demonstrated that the addition of ketamine 0.5 mg.kg$^{-1}$ to bupivacaine for caudal epidural blockade in children prolongs the duration of postoperative analgesia and reduces the requirement for analgesics to a greater extent than the addition of adrenaline or clonidine [2–4]. Ketamine produces potent analgesia when given in subanaesthetic doses. It exerts its anaesthetic and analgesic actions by binding to a subset of glutamate receptors stimulated by the agonist N-methyl d-aspartate (NMDA receptors). These are found throughout the central nervous system including the lumbar spinal cord. As well as a general anaesthetic effect produced by systemic administration, ketamine exerts profound analgesic actions at a spinal cord level in animal preparations [5, 6]. In clinical practice ketamine injected into the lumbar epidural space after abdominal surgery has been shown to have a potent analgesic effect [7, 8].

Surgical procedures in the inguinal region are common in paediatric surgery and analgesia may be provided either by a caudal epidural block or by an ilioinguinal nerve block. These techniques have been shown to have similar efficacy in orchidopexy [9] and in inguinal hernia repair [10]. Ilioinguinal nerve block is free of the side-effects of lower limb motor block and urinary retention [10, 11] which may be caused by caudal blockade and, for this reason, an ilioinguinal nerve block is often the preferred analgesic technique. This study was designed to compare these two techniques when ketamine is used to prolong the duration of the caudal epidural block and to determine the duration of analgesia and incidence of side-effects when they are used to provide analgesia after orchidopexy. Ilioinguinal nerve blocks were supplemented by infiltration of the wound under direct vision by the surgeon in a bid to improve efficacy and diclofenac was used in both groups since it has been shown to prolong the effects of both caudal epidural and ilioinguinal nerve blockade in paediatric practice [12].

**Methods**

The study was approved by the local Ethics Committee and written informed parental consent was obtained for each subject. The study group comprised 40 boys weighing up to 25 kg undergoing unilateral orchidopexy as day cases. Children in whom there were contraindications to caudal blockade and/or whose parents were unable or unwilling to perform objective pain assessments were not studied. At the time of recruitment, parents were instructed in the use of the modified Objective Pain Score [1, 9, 13] for the assessment of postoperative pain and the requirement for analgesia. This is an observational pain scoring system which has been validated for use by parents [13]. The score uses five criteria: crying, agitation, movement, posture and localisation of pain. Each criterion scores from 0 to 2 to give a total score of 0–10.

Premedication was not used but EMLA cream was applied at least 1 h pre-operatively. Anaesthesia was induced with propofol 3–4 mg.kg$^{-1}$ followed by placement of a laryngeal mask. Anaesthesia was maintained with halothane 0.5%–2% with 70% nitrous oxide in oxygen.

After induction of anaesthesia, patients were allocated, by a computer-generated randomisation scheme, to receive either caudal epidural blockade (Group C) ($n = 20$) or ilioinguinal nerve block and local infiltration (Group L) ($n = 20$). Children in Group C were positioned on their side and a caudal injection was performed by a consultant anaesthetist using an aseptic technique. This consisted of 0.25% bupivacaine 1 ml.kg$^{-1}$ with preservative-free ketamine (10 mg.ml$^{-1}$) 0.5 mg.kg$^{-1}$ to a maximum volume of 20 ml. In Group L, an ilioinguinal nerve block was performed by a consultant anaesthetist using a 22G short-bevelled regional block needle to inject 0.25% bupivacaine 0.5 ml.kg$^{-1}$. At the end of the operation the surgeon infiltrated the wound with 0.25% bupivacaine 0.5 ml.kg$^{-1}$ to a maximum volume of 20 ml. All boys had occlusive dressings placed over both of the possible sites of local anaesthetic injection before leaving the operating theatre. A suppository containing diclofenac sodium 1–2 mg.kg$^{-1}$ was inserted after instituting the regional block and before the start of surgery.

Anaesthetic agents were discontinued at skin closure and the time from discontinuation of anaesthesia to spontaneous eye opening was noted. Before discharge, analgesia was requested by parents (who were unaware of the local anaesthetic technique employed) when their estimate of the Objective Pain Score reached 4 or more. This consisted of paracetamol 15 mg.kg$^{-1}$ by mouth four-hourly as required. The duration of motor blockade was assessed by determining when the children began to move their legs. The time of first micturition was noted. The time of first micturition was noted. The degree of sedation was assessed at 1 and 4 h postoperatively using an objective score based on eye opening (eyes open spontaneously = 0; eyes open in response to verbal stimulation = 1; eyes open in response to physical stimulation = 2).

After discharge home, parents were asked to assess the child regularly and to give analgesia if the Objective Pain Score reached 4 or more. Parents were contacted by telephone the morning following surgery to determine the requirement for analgesia after discharge from hospital.
the timing of micturition and any evidence of nightmares, hallucinations or odd behaviour. The total requirement for postoperative analgesia in the first 24 h was recorded.

Statistical analysis was performed using Student’s t-testing for normally distributed data and the Mann–Whitney U-test and Chi-squared test for nonparametric data.

**Results**

Two subjects in Group C and two in Group L were not included in the analysis. In two cases the subject did not undergo the scheduled surgery and in two it proved impossible to obtain follow up data. The two groups were similar in respect of physical characteristics and operative details (Table 1).

The median duration of action of the technique employed, as indicated by the time to first analgesia, was significantly longer in Group C (10 h) than in Group L (2.9 h) (p < 0.05) (Fig. 1).

The number of subjects pain free (OPS ≤ 4) at hourly intervals up to 24 h is shown for each group. Significant differences between the two groups occurred between 3 and 8 h (p < 0.01) (Fig. 2). Significantly fewer doses of analgesia were required over the first postoperative 24 h by subjects in Group C compared with Group L (p < 0.05) (Fig. 3).

There were no significant differences between the groups either in the time taken to spontaneous eye opening after the cessation of anaesthesia or in sedation scores 4 h postoperatively. The times to first micturition and spontaneous leg movements were similar in the two groups. One subject in Group C and none in Group L vomited during the postoperative period (Table 2). There were no reports of nightmares, hallucinations or odd behaviour in any subject.

**Discussion**

This study was designed to compare the efficacy and incidence of side-effects of caudal epidural analgesic
The incidence of side-effects is very low in both groups. However, hallucinations and nightmares have been reported after the use of epidural ketamine. Although hallucinations occurred in one child in group C. Although some degree of motor block occurred in this group this was of little clinical importance. Previous work has shown that the degree of block with ketamine supplementation of bupivacaine prolongs the duration of caudal epidural blockade [2, 3]. Our study has also shown a significant difference in the duration of postoperative analgesia and the requirement for subsequent analgesia between the two techniques. There was no difference in postoperative sedation between the groups as shown by the time to spontaneous eye opening and sedation scores at 4 h postoperatively. We found no evidence of prolonged motor block in Group C. Although some degree of motor block occurred in this group this was of little clinical importance. Previous work has shown that the degree of block with ketamine supplementation is no greater than when bupivacaine is used alone [3]. It was notable that postoperative vomiting only occurred in one child in group C. Although hallucinations or nightmares have been reported after the use of epidural ketamine they appear to be very rare when a dose of 0.5 mg.kg$^{-1}$ is used compared with 1 mg.kg$^{-1}$ when the incidence is higher [4]. Parents’ assessments of pain and the need for analgesia using the Objective Pain Score has been shown to be comparable to pain assessments made by a doctor [13].

In summary, when ketamine 0.5 mg.kg$^{-1}$ is used to prolong the duration of caudal epidural blockade with bupivacaine in children, the duration of analgesia is longer and the requirement for postoperative analgesia less than that seen with ilioinguinal nerve block with bupivacaine supplemented by wound infiltration. The incidence of side-effects is very low in both groups.

**Acknowledgment**

Dr Findlow was supported by the Sir Jules Thorn Charitable Trust.

### Table 2 Recovery characteristics in boys undergoing orchidopexy with caudal (group C) or ilioinguinal (group L) methods of pain relief. Values are median [range] where appropriate.

<table>
<thead>
<tr>
<th></th>
<th>Group C (n = 18)</th>
<th>Group L (n = 18)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Spontaneous eye opening: min</td>
<td>29 [17–50]</td>
<td>27 [4–45]</td>
</tr>
<tr>
<td>4h sedation score</td>
<td>0 [0]</td>
<td>0 [0]</td>
</tr>
<tr>
<td>Micturition: h</td>
<td>4.8 [2.5–8.3]</td>
<td>3.7 [1.5–7.0]</td>
</tr>
<tr>
<td>Leg movements: h</td>
<td>&lt;1 [0–5]</td>
<td>&lt;1 [0–5]</td>
</tr>
<tr>
<td>Postoperative vomiting: n</td>
<td>1</td>
<td>0</td>
</tr>
</tbody>
</table>

References