Abstracts

Confirmation of tracheal intubation using a chemical device

J.P. O'Callaghan, R.T. Williams
Department of Anaesthesia, Foothills Hospital at the University of Calgary, Calgary, Alberta

Oesophageal intubation is a major cause of anaesthetic-related morbidity and mortality. In 1980, Pollard and Junius[1] showed that the commonly accepted techniques for verifying correct tracheal tube placement could not be relied on. There were certain patients in whom oesophageal intubation could "mimic" the signs of tracheal intubation. They concluded that direct visualization of the tube entering the larynx was the only sure certain method correct placement. Since that time, monitors which can detect carbon dioxide in the expired gas have become available. In a recent review[2] of techniques for detecting oesophageal intubation, it was found that end-tidal CO_2 measurement and direct cord visualisation were the only two techniques with no documented incidents of failure. It is now recommended that expired gases be monitored for carbon dioxide in all cases where the vocal cords have not been visualized.

This involves the use of electrically powered, often expensive, quantitative carbon dioxide analysers. We would like to describe a new, inexpensive and easy to use device for the detection of carbon dioxide in the expired gases.

Description of device

The device consists of a pH-sensitive chemical indicator which is enclosed in a replaceable disc housing and connected to the gas stream between the endotracheal tube and the anaesthetic circuit. Exposure to more than one per cent carbon dioxide results in an instantaneous colour change from white to a deep blue. At present, the active chemical is hydrazine hydrate on alumina with crystal violet as the indicator dye, but investigations are currently underway to find a less toxic active chemical. The amount of hydrazine used per test is less than 1.0 mg and the alumina-hydrazine-crystal violet mixture is adhered to tape to prevent any contamination of the patient. The device has been used in 100 patients (thiopentone and succinylcholine induction), ranging in age from 4–78 years. In these apnoeic patients, one push on the chest or inflation of the lungs is sufficient to produce a dramatic colour change if the trachea is intubated; "deliberate" intubation of the oesophagus resulted in no colour change at all. Occasionally, mask ventilation before an attempted intubation may force some of the patient's own exhaled gas into the stomach. Once this has occurred, carbon dioxide can be detected in the expired gases following an oesophageal intubation, but continued ventilation eliminates the carbon dioxide within one minute. Under these circumstances, an initial colour change occurs in the chemical indicator, but this fades quickly as the carbon dioxide concentration falls. Any fade in indicator colour necessitates the insertion of a new indicator disc, which would immediately confirm that an oesophageal intubation has occurred.

Discussion

The quantitative carbon dioxide analyser is one of the most valuable monitors we have in clincial anaesthesia. Unfortunately, because of their size and cost, these monitors are not available in all areas where tracheal intubation is carried out. Few hospitals can supply every operating room, emergency room or "crash cart" with one of these devices, and they are not practical for use by medical personnel in "field" settings or by paramedics in their work environment. The pH-sensitive chemical indicator, on the other hand, is small, inexpensive and easy to use, and would be ideal for all the above situations. It is not a replacement for the quantitative carbon dioxide analyser, but if used in conjunction with the usual methods of detecting correct tube placement, it should reduce the number of undiagnosed oesophageal intubations.

References

1 *Pollard BJ, Junius F.* Accidental intubation of the oesophagus. Anaesth Intensive Care. 1980; 8: 195–8.
2 *Birmingham PK, Cheney FW, Ward RJ.* Esophageal intubation: a review of detection techniques. Anesth Analg 1986; 65: 886–91.

Preoxygenation and denitrogenation

C.J.E. Cruise, R.R. Crago, S.M. Paluck, F.J. Carmichael
Department of Anaesthesia, Toronto Western Hospital, Toronto, Ontario

Preoxygenation of patients prior to anaesthetic induction replaces nitrogen in the lungs with oxygen and facilitates a subsequent period of apnoea without the development of hypoxia. Various techniques of preoxygenation have been recommended, varying from four maximal breaths to ten minutes of normal breathing of 100 per cent oxygen. Previous authors[1] have assessed the adequacy of preoxygenation by measuring the saturation of haemoglobin and the time taken for desaturation to occur during a period of apnoea. However, saturation alone does not indicate the lung's oxygen reserve.

The purpose of this study was to determine whether four inspiratory capacity breaths and three minutes of tidal breathing of 100 per cent oxygen provide the same degree of denitrogenation of the lungs as measured by mass spectrometry.

Methods

Nine ASA physical status class I, non-smoking volunteers were studied under two conditions. In a supine position, breathing from a circle system with a fresh gas flow rate of 10 $L \cdot min^{-1}$, using a tight-fitting mask, each subject (i) breathed 100 per cent

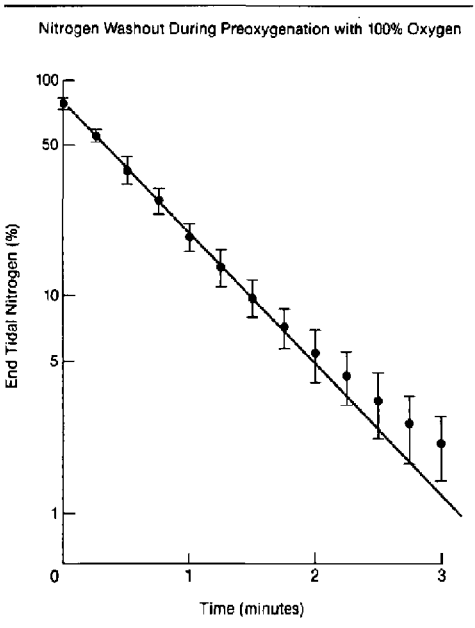


FIGURE Log_{10} end-tidal nitrogen concentration (per cent) vs time (minutes) for tidal volume breathing of 100 per cent oxygen (n = 9).

oxygen at tidal volume for three minutes or (ii) took eight inspiratory capacity breaths exhaling to FRC. End-tidal nitrogen and carbon dioxide, sampled at the face mask, were continuously analyzed using a Perkin-Elmer Advantage 2000 mass spectrometer.

Results

End-tidal nitrogen concentration (log_{10}) for nine subjects during three minutes of tidal breathing is plotted against time in the Figure. The end-tidal nitrogen content was 18.9 ± 2.8 per cent after one minute, 5.5 ± 1.5 per cent after two minutes, and 2.1 ± 0.7 per cent after three minutes. The time required to reach an end-tidal nitrogen concentration of one per cent ranged from two minutes 15 seconds to six minutes 30 seconds.

The end-tidal nitrogen concentration (per cent) following inspiratory capacity breathing was: control 79.3 ± 1.2; two breaths 38.2 ± 4.5; four breaths 16.9 ± 2.5; six breaths 8.7 ± 1.8; eight breaths 6.1 ± 1.3.

Discussion

The replacement of nitrogen in the lungs with oxygen is more complete following three minutes of tidal breathing than following four inspiratory capacity breaths. Eight inspiratory capacity breaths would be required to provide an oxygen reserve equivalent to two minutes of normal tidal breathing of 100 per cent oxygen.

Mass spectrometry, which is becoming increasingly available,[2] is a valuable aid in assessing the adequacy of denitrogenation of the lungs prior to anaesthetic induction in subjects breathing 100 per cent oxygen. This technology would be most advantageous when applied to patients with lung disease where rates of denitrogenation could be markedly altered.

References

1 *Gambee AM, Hertzka RE, Fisher DM.* Preoxygenation techniques: comparison of three minutes and four breaths. Anesth Analg 1987; 66: 468–70.

2 *Ozanne GM, Young WG, Mazzei WJ, Severinghaus JW.* Multiple anesthetic mass spectrometry: rapid analysis of data stored in long catheters. Anesthesiology 1981; 55: 62–70.

Cardiopulmonary effects of the volume recruitment manoeuvre for measuring respiratory mechanics during anaesthesia

D. Shulman, G. Volgyesi, K. Brown, J. Lerman, S. England, A.C. Bryan

The Research Institute and the Department of Anaesthesia, The Hospital for Sick Children, University of Toronto, Toronto, Ontario

Respiratory mechanics can be measured by a new, non-invasive technique termed "volume recruitment" in spontaneously breathing animals and infants.[1] In order to study the applicability of this test to anaesthetized subjects, we examined the effects of this manoeuvre during anaesthesia on lung volumes, respiratory rate, arterial blood gases, and on cardiac output.

Methods

Seven 2.8 to 7.0 kg infant swine, ten days to three weeks of age, were studied supine and spontaneously breathing during halothane in N_2O/O_2 anaesthesia delivered via a cuffed 3.5 mm endotracheal tube. The end-tidal fraction of halothane was 0.7–1.23 per cent. The femoral artery and vein were cannulated and the electrocardiogram and blood pressure were continuously recorded on a four-channel chart recorder. Attached to the endotracheal tube was a heated Fleisch pneumotachograph connected to a differential pressure transducer whose signal was amplified and integrated. The flow (V), volume (V) and airway pressure (P) signals were recorded on magnetic tape and on chart paper (Figure). Fresh gas flow entered the inspiratory port of a unidirectional valve connected to the pneumotachograph, and expiratory gas was vented to the room via the expiratory port.

During spontaneous breathing tidal volume (V_t) and frequency (f) were measured and arterial to alveolar oxygen gradient ($D(A\text{-}a)PO_2$) was calculated using the alveolar gas equation. In six swine cardiac output (CO) by indocyanine green dilution was also determined. In three animals functional residual capacity (FRC) was measured by closed circuit Helium dilution. The expiratory port of the unidirectional valve was then occluded which prevented expiration but allowed inspiration for three consecutive breaths. After the third inspiration, the occlusion was released (Figure). The above measurements were then repeated. Statistical analysis was by the Wilcoxon signed rank test using each subject as his own control.

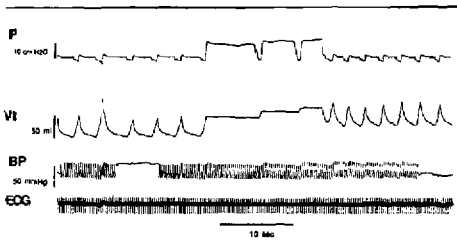


FIGURE Volume (V_t) and pressure (P) signals from an infant swine during the volume recruitment manoeuvre. Expiration is occluded while the subject progressively recruits volume during three unoccluded inspirations. V_t and f increase following the release of the occlusion while there is no change in the blood pressure (BP) or in the ECG. Blood sampling resulted in the interruptions in the BP signal.

Results

V_t and f increased significantly following the volume recruitment manoeuvre (Figure), and CO decreased to a small but significant extent (1.03 ± 0.37 L·min^{-1} before vs 0.97 ± 0.36 L·min^{-1} after the manoeuvre, $p = 0.02$). There was no significant change in D(A-a)PO_2. $PaCO_2$ increased following the manoeuvre but the change failed to reach statistical significance ($p = 0.06$). FRC increased by a mean of 41.4 ± 18.1 ml which represented a 32.9–62.6 per cent increase in baseline FRC.

Discussion

The technique of volume recruitment was simply applied and non-invasive. We conclude that the volume recruitment manoeuvre can be applied to spontaneously breathing anaesthetized subjects with no deleterious cardiopulmonary effects.

Reference

1 *Grunstein MM, Springer C, Vilozni D, Bar-Yishay E, Godfrey S.* Expiratory volume clamping (EVC), a new method to assess respiratory mechanics in infants. J Appl Physiol 1987; 62: 2107–14.

The effect of nitrogen balance on weaning from mechanical ventilation

H.R. Wexler, J. Pikul
Departments of Anaesthesia and Clinical Nutrition Services, University Hospital, University of Western Ontario, London, Ontario

Patients suffering from hypermetabolic states such as sepsis, trauma or multi-organ failure often require prolonged mechanical ventilation. During the hypermetabolic period there is obligatory skeletal muscle catabolism which usually cannot be reversed by nutritional support and appears to affect respiratory muscle function, metabolic demands, and weaning from mechanical ventilation.[1] Serveral studies indicate that overall nutritional status may predict potential success in weaning, but none have studied the effect of nitrogen balance per se on weaning.[1–5] We therefore compared the 24-hour nitrogen balance with the ability to wean from mechanical ventilation.

We studied 21 ICU patients who required mechanical ventilation for more than five days. All patients had a negative nitrogen balance on admission and received enteral nutritional support of ≥110 per cent of their energy expenditure measured in calories/day, and ≥1.0 g protein per kg body weight/day. The average length of mechanical ventilation was 33.2 days (6–69 days). At the time of successful weaning, 19/21 patients (90.5 per cent) had a positive nitrogen balance averaging +4 g (0 ± 8.2 g). Two/21 (9.5 per cent) were able to be weaned with negative nitrogen balances of −2.3 g and −2.1 g ($p < 0.002$). Both of these patients required relatively short periods of mechanical ventilation (10 and 12 days), and one presented following gastroplasty for morbid obesity.

We suggest that ventilator-dependent patients are more likely to wean with a positive nitrogen balance, i.e., an anabolic state, and that aggressive weaning of catabolic patients may not be feasible and should be delayed until successful repletion can occur.

References

1 *Bassili H, Deitel M.* Nutritional support in long term intensive care with special reference to ventilator patients: a review. Can Anaesth Soc J 1981; 286: 17–20.
2 *Bassili H, Deitel M.* Effect of nutritional support on weaning patients off mechanical ventilators. JPEN 1981; 161–3.
3 *Larca L, Greenbaum D.* Effectiveness of intensive nutritional regimes in patients who fail to wean from mechanical ventilation. Crit Care Med 1982; 10: 297–300.
4 *Rochester, D.* Malnutrition and the respiratory muscles. Clin Chest Med 1986; 7: 91–9.
5 *Kelly S, Rosa A, Field S et al.* Inspiratory muscle strength and body composition in patients receiving total parenteral nutrition therapy. Am Rev Respir Dis 1984; 130: 33–7.

The efficacy of three different modes of percutaneous trans-tracheal ventilation in hypoxic, hypercarbic swine

T.C. Thomas, M.H. Zornow, M.S. Scheller, R.J. Unger
University of California at San Diego, San Diego, California

Acute upper airway obstruction constitutes a true medical emergency. When mask ventilation or tracheal intubation is impossible, percutaneous trans-tracheal ventilation (PTTV) may provide oxygenation, ventilation, and prove to be a life-saving manoeuvre. Numerous techniques of PTTV have been proposed,[1–3] but their relative ability to reverse existing hypoxia and provide ventilation has not been studied. The purpose of this study was to examine the ability of three different techniques of PTTV to provide ventilation and reverse pre-existing hypoxia in swine.

Methods

The three methods of PTTV tested were as follows: (1) *Jet* – the tracheal catheter (see below) was connected to the hospital piped oxygen supply (50 psi) via high-pressure tubing with flow

regulated by a thumb-operated valve.[4] This valve was activated at a rate of 15/min; (2) *Flush* – the tracheal catheter connected to the fresh gas outlet on the anaesthesia machine via a length of high-pressure tubing and activated by intermittently depressing the oxygen flush button at a rate of 15/min; (3) *Circle* – the tracheal catheter connected to a standard anaesthesia circuit via a 3 mm endotracheal tube connector and vigorous attempts made at manual ventilation with circle pressures consistently greater than 80 cm H_2O.

After obtaining institutional animal care committee approval, five healthy pigs (approximately 22 kg) were sedated with intramuscular ketamine (20 mg·kg^{-1}) and allowed to spontaneously breathe 3.5 per cent isoflurane. Following oral endotracheal intubation, the pigs were ventilated in the supine position with two to three per cent isoflurane in oxygen to achieve a normal end-tidal CO_2. Pancuronium (0.2 mg·kg^{-1}) was intermittently injected via an ear vein catheter to ensure muscle paralysis. Oxygen saturation was measured with a pulse oximeter probe (Nellcor N-25) placed on the animal's ear. Following infiltration with 0.25 per cent bupivacaine, a groin incision was made for the insertion of a femoral artery cathether for continuous monitoring of blood pressure and intermittent sampling of arterial blood for blood gas determinations (ABG). A 2 in 14 gauge IV catheter was then inserted (percutaneously) into the trachea caudad to the tip of the endotracheal tube. Following the surgical preparation, a stabilization period ensued to ensure that $PaCO_2$ was in the normal range (33–40 mmHg) and that PaO_2 was greater than 350 mmHg, as demonstrated by two separate ABG determinations at least five minutes apart (B1 and B2). At this point the O_2 saturation was 100 per cent in all cases. The endotracheal tube was then disconnected from the ventilator and a one-way valve was attached to the end of the tube to prevent the entrainment of room air while allowing for unrestricted exhalation. The animals were left apnoeic until the oxygen saturation fell to 60 per cent (t = 0) at which time ventilation was begun with one of the three methods of PTTV (*Jet*, *Flush* or *Circle*). Arterial blood gas samples were drawn at t = 0 and at one minute intervals thereafter, for the remainder of the ventilation period (five minutes). Expired volumes and oxygen saturation were recorded as the ABG samples were drawn. After five minutes, conventional ventilation via the endotracheal tube was resumed and the sequence was repeated using one of the other modes of PTTV (each animal was ventilated with all three modes of ventilation). Upon completion of the experiment, the animals were killed with an intravenous bolus of KCl and the tracheas were removed and examined for evidence of injury. Data was obtained from the five animals and the mean values (± SD) for PaO_2 and $PaCO_2$ at each sample time were calculated. The PaO_2 and $PaCO_2$ for each mode of ventilation were compared using a repeated measures ANOVA followed by Scheffe's test for multiple comparisons, when appropriate.

Results

At t = 0 (oxygen saturation = 60 per cent), there were no significant differences in $PaCO_2$ or PaO_2 before application of any of the three PTTV methods. Figure 1 demonstrates that the PaO_2 increased with all three modes of PTTV. At t = 1 minute,

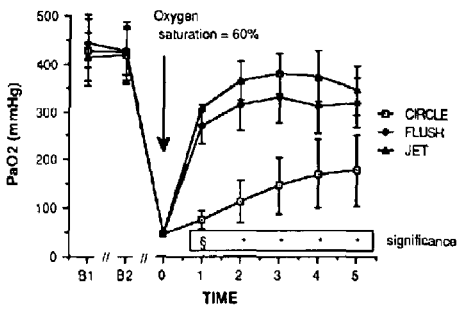


FIGURE 1 Arterial PaO_2 with each of the three modes of PTTV (B1 = baseline 1, B2 = baseline 2). Significance – § = Jet > Flush > Circle, $p < 0.05$; * = Jet > Circle and Flush > Circle, $p > 0.05$. No significant difference between Jet and Flush modes.

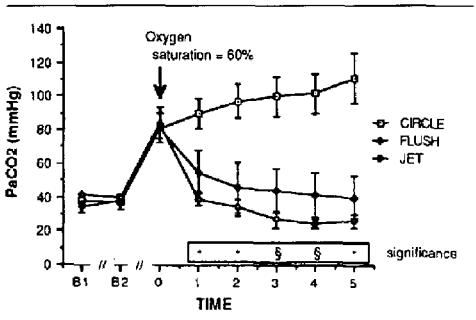


FIGURE 1 Arterial PaO_2 with each of the three modes of PTTV (B1 = baseline 1, B2 = baseline 2). Significance – § = Jet > Flush < Circle, $p < 0.05$; * = Jet < Circle and Flush < Circle, $p < 0.05$. No significant difference between Jet and Flush modes.

the mean PaO_2 was significantly different for all three intergroup comparisons (i.e., Jet > Flush > Circle, $p < 0.05$). At each subsequent time point, the mean PaO_2 in the *Jet* and *Flush* groups were greater than the mean PaO_2 in the *Circle* group, but no differences existed between the *Jet* and *Flush* modes. Figure 2 demonstrates that both the *Jet* and *Flush* modes resulted in lower mean $PaCO_2$ values compared to control (t = 0) at all measurement points. At t = 3 and t = 4 minutes the *Jet* $PaCO_2$ was less than that of the *Flush* $PaCO_2$. The *Circle* mode was associated with increasing $PaCO_2$ at all points beyond control ($p < 0.05$, *Circle* vs *Flush* and *Circle* vs *Jet* modes). Gross and microscopic inspection of the tracheas demonstrated full-thickness erosion of the posterior tracheal mucosa at the tip of the tracheal catheter. These lesions were relatively uniform in size and shape (≈0.8 × 1.2 cm in diameter).

Discussion

These data demonstrate that the *Flush* and *Jet* modes of PTTV are superior to the *Circle* mode in their ability to both oxygenate

and ventilate hypoxic, hypercarbic swine. While it may be possible to reverse pre-existing hypoxia with the *Circle* mode, it is not possible to provide adequate ventilation. Damage to the tracheal mucosa, presumably from the high velocity jets of oxygen generated by the *Jet* and *Flush* modes, was demonstrated in each of the animals.

References

1 *Spoerel WE et al.* Transtracheal ventilation. Br J Anaesth 1971; 43: 932–9.
2 *DeLisser EA, Muravchick S.* Emergency transtracheal ventilation. Anesthesiology 1981; 55: 606–7.
3 *Reich DL, Schwartz N.* An easily assembled device for transtracheal oxygenation. Anesthesiology 1987; 66: 437–8.
4 *Millar WL.* Management of a difficult airway in obstetrics. Anesthesiology 1980; 52: 523–4.

A comparison of five metallic tapes for protection of endotracheal tubes during CO_2 laser surgery

M. Sosis, S. Heller
Department of Anesthesia, Indiana University Medical Center, Indianapolis, Indiana

The use of the laser in proximity to the airway during general endotracheal anaesthesia may cause a disastrous endotracheal tube fire.[1] It has been recommended that combustile endotracheal tubes be wrapped with metal tape to prevent this catastrophy.[1] However, it has been suggested that some tapes may themselves be flammable.[2] We evaluated the protection offered by five commercially available metal tapes.

Methods

Size 8 ID polyvinylchloride endotracheal tubes were wrapped with 1/4″ self-adhesive foil tape. The wrapping was started at the distal (cuffed) end and was applied in an overlapping spiral fashion. The tapes used were: 3M (St. Paul, MN) #425,[3] #1430, #433, Radio Shack (Tandy Corp., Ft. Worth, TX) #44-1155 and copper foil tape (Venture Tape Corp., Rockland, MA). Five litres of oxygen per minute were made to flow through the tubes. A Laser Sonics model LS880 CO_2 laser and Zeiss operating microscope employing a 400 mm lens and 0.68 mm beam diameter were used. The beam was directed onto the foil-wrapped tubes at the point of overlapping of the tape. Seventy watts of power in the continuous mode of laser operation was used. The time necessary until the occurrence of smoke, flames or perforation and "blow torch" ignition were noted. Finally, a segment of tape was wrapped adhesive-side outward and the same procedure undertaken.

Results

3M #425, #433 and copper tape were unaffected by 25 seconds of laser impact. Penetration and blow torch fire of the tube occurred in 7 and 14 seconds respectively with the Radio Shack and 3M #1430 tapes.

The adhesive backing of the 3M #433 and Radio Shack tapes were ignited and the tape perforated within 0.1 second of the laser's impact. Flaming occurred at one second without penetration of the 3M #1430 and copper tape. 3M #425 tape could be made to smoke at two seconds; however, no flaming occurred and there was no perforation.

Discussion

The high-energy density of the laser can easily perforate combustible endotracheal tubes. The anaesthetic gases will then promote the combustion of the tube turning it into a blow torch. Wrapping endotracheal tubes with foil tape has been advocated to protect them from the laser.[1] Most authors do not specify which tape to use[1] and some have even recommended tape shown to be inadequate in this study.[4]

3M #1430 and Radio Shack tapes offer inadequate protection of flammable endotracheal tubes and should not be used for this purpose. 3M #433 tape has a flammable backing and also should not be used. Copper foil and 3M #425 tapes provided excellent protection of the endotracheal tubes when a meticulous technique of wrapping is used and are thus recommended for use during CO_2 laser surgery in proximity to the airway. We have gas sterilized tubes wrapped with these tapes without affecting the tape or its adherence to the tube. The copper foil is malleable, resulting in a smoother exterior than the 3M #425 tape.

References

1 *Hermans JM, Bennett MJ, Hirshman CA.* Anesthesia for laser surgery. Anesth Analg 1983; 62: 218–29.
2 *Cork RC.* Anesthesia for otolaryngologic surgery involving use of a laser. Brown BR Jr. (Ed). Contemporary anesthesia practice. F.A. Davis, 1987 pp. 127–40.
3 *Carruth JAS, McKenzie AL, Wainwright AC.* The carbon dioxide laser: safety aspects. J Laryngol Otol. 1980; 94: 411–7.
4 *Lim RY, Kenney CL.* Precaution and safety in carbon dioxide laser surgery. Otolaryngol head Neck Surg 1986; 95: 239–41.

Assessment of preoperative gastric emptying using a marker dye

J.R. Maltby, N. Koehli, A. Ewen, E.A. Schaffer
Departments of Anaesthesia and Gastroenterology, Foothills Hospital at University of Calgary, Calgary, Alberta

Residual gastric fluid volume (RGV) is measured commonly by aspiration through a multi-orifice Salem sump tube.[1] However, previously ingested fluid cannot be distinguished from gastric secretions using this technique. A marker dye which is not absorbed from the stomach may be used to make this distinction, and to assess gastric emptying in the interval between ingestion and sampling. Phenol red is the standard inert marker used to measure volume in gastric emptying studies.[2,3]

Methods

Informed consent was obtained from 120 healthy, non-pregnant, elective surgical inpatients, age 18–70 yr, who were randomly allocated to one of four groups. Patient characteristics of age, sex, weight, duration of overnight fast and history of heartburn and smoking were recorded. Those receiving drugs known to affect gastric secretion or motility were

TABLE Results

Group (n)	Fast (hr)	Total fluid aspirate (ml)	Ingested fluid (ml)	Gastric secretion (ml)
1 fast (30)	14.6 ± 1.7	24(0–90)	0(0–1)	24(0–90)
2 fast (30)	14.0 ± 1.5	13(0–70)	0(0–2)	13(0–70)
3 fluid(29)	2.7 ± 0.8	17(0–133)	2(0–17)	15(0–131)
4 fluid(31)	2.6 ± 0.6	14(0–51)	2(0–16)	12(0–43)

Fast values: mean ± SD. Other values: mean (range).

excluded. Between 2 and 3 hr before the scheduled time of surgery, all patients ingested phenol red 50 mg in 10 ml water, with placebo tablet alone (Groups 1 and 2), placebo tablet with 150 ml oral fluid (Group 3) or oral ranitidine 150 mg with 150 ml oral fluid (Group 4). Patients chose coffee, tea, fruit juice or water. Those in Group 1 received oral diazepam 5–15 mg 1–1.5 hr preoperatively or no premedication. Those in Groups 2, 3 and 4 received IM morphine 7.5–15 mg or meperidine 75–100 mg, with atropine 0.6 mg 1 hr preoperatively. Following induction of anaesthesia, gastric contents were aspirated through a #18 Salem sump tube and volume was recorded. Phenol red concentration was measured using a Beckman U-50 spectrophotometer whose lower limit of detection was 5 $\mu g \cdot ml^{-1}$. From this value and RGV the percentage dye retrieval was calculated, and thus the proportion of RGV which represented ingested fluid.

Results

Patient characteristics were similar in all groups. In only ten per cent of all patients was more than five per cent of ingested dye retrieved. In Groups 3 and 4 the highest volume of ingested fluid at the time of sampling was 17 ml (Table).

Discussion

The use of a marker dye demonstrates that gastric emptying of ingested fluid is almost complete within two hours. Therefore, even in patients who receive narcotic-atropine premedication, residual gastric fluid at induction of anaesthesia is predominantly gastric secretions.

References

1 *Hardy JF, Plourde G, Lebrun M, Coté C, Dube S, Lepage Y.* Determining gastric contents during general anaesthesia: evaluation of two methods. Can J Anaesth 1987; 34: 474–7.

2 *Bloom DS, Jacobson ED, Grossman MI.* Validation of dilution indicators in the stomach. Gastroenterology 1967; 52: 205–10.

3 *George JD.* New clinical method for measuring the rate of gastric emptying: the double sampling test meal. Gut 1968; 9: 237–42.

Intraoperative prochlorperazine for prevention of postoperative nausea and vomiting

R.J. Cramb, A. Fargas-Babjak
McMaster University Medical Centre, Hamilton, Ontario

Prochlorperazine (Stemetil) has been acknowledged as the "gold standard" for treatment of nausea and vomiting during chemotherapy.[1–3] The purpose of this study was to evaluate the effectiveness of prochlorperazine (10 mg) in preventing postoperative nausea and vomiting. The antiemetic and antinauseant benefits of this drug have not been critically evaluated in the postanaesthetic patient population. Prochlorperazine was given intraoperatively so that an adequate drug concentration should have been present at the time of the patient's recovery from anaesthesia. All patients selected for the study were having surgical procedures (abdominal, head and neck) known to have a high incidence of nausea and vomiting.

Methods

One hundred ASA physical status I or II patients, 85 women and 15 men, aged 18–70 years participated in the study after giving informed consent. Human ethics committee approval was obtained. Each patient was randomized to a treatment (prochlorperazine) or control (sterile water) group, each containing 50 patients. One patient in the control group did not complete the study as a full dose of medication was not administered. The anaesthetists, patients, and nursing staff were blinded to the nature of the treatment received.

Each patient received a standardized anaesthetic consisting of thiopental 1–5 $mg \cdot kg^{-1}$, succinylcholine 1–2 $mg \cdot kg^{-1}$, fentanyl less than 2 $\mu g \cdot kg^{-1}$, isoflurane, nitrous oxide (70 per cent), muscle relaxant and reversal of choice. After the induction of anaesthesia, with vital signs stable either the treatment drug or placebo was given intravenously. Heart rate and blood pressure were recorded prior to injection, then at one- and five-minute intervals. Postanaesthetic observations on patient nausea and vomiting were gathered by the nursing staff in the recovery room. Nausea and vomiting was scored twice, once before and then after receiving narcotics for pain relief during the stay in the recovery room. Nausea was scored either present or absent as judged by the patient after achieving a full Glasgow coma scale score. Vomiting was separated into four categories; 0 – no vomiting, 1 – retching only, 2 – vomiting once only, 3 – vomiting more than once. The minimum stay in the recovery room was one hour. Statistical analysis was by Chi square or Students t test where appropriate.

Results

The two groups were well matched for age, sex, height and

TABLE Results

	Nausea		Vomiting			
	None	Present	0	1	2	3
Before narcotics						
Placebo	35	14	39	7	2	1
Stemetil	37	13	36	11	2	1
After narcotics						
Placebo	31	10	31	7	2	1
Stemetil	30	9	29	9	1	0

Vomiting scale: 0 = none; 1 = retching; 2 = vomiting once; 3 = vomiting more than once.

Eight patients in the placebo group and 11 patients in the Stemetil group did not receive narcotics.

weight. All but one patient were intubated and mechanically ventilated although there were no study restrictions regarding airway management and ventilation. There was no statistically significant change in heart rate or blood pressure between the two groups. The data for nausea and vomiting in each group are presented in the Table. The difference between groups both before and after receiving narcotics did not reach statistical significance. We conclude that prochlorperazine is not an effective anti-nauseant or antiemetic for the postanaesthetic patient population.

TABLE Distribution of females vomiting after laparoscopy

		Metoclopramide	
	Control	*20 mg*	*50 mg*
Number in group	30	30	30
Nausea/vomiting	16	10 (NS)	7 ($p = 0.027$)

References

1 *Frejtak S, Moestel CG.* Management of nausea and vomiting in the cancer patient. JAMA 1981; 245: 393–7.

2 *Moertel CG, Rutemeier E.* A controlled clinical evaluation of antiemetic drugs. JAMA 1963; 186: 116–9.

3 *Sallan SE, Cronin C.* Antiemetics in patients receiving chemotherapy for cancer. N Enol J Med 1980; 302: 135–9.

The Gender-dependent pharmacodynamic difference in the antiemetic action of metoclopramide is a dose-related phenomenon

M.J. Diamond, D. Bailey, A. McPhee

Department of Anaesthesia, Victoria Hospital, University of Western Ontario, London, Ontario

Previous work on the antiemetic action of metoclopramide demonstrated that not only was there a reduction in postoperative nausea and vomiting, but also that the overall response was attibutable to the male component of the patient population.[1] The difference between male and female response prompted the present study bearing in mind the safe and effective reduction of chemotherapy-induced emesis by high-dose metoclopramide therapy.[2] It was felt that by increasing the dose of metoclopramide a beneficial effect would occur in females undergoing surgery. To test this hypothesis, a randomised double-blind placebo-controlled parallel study was undertaken in premenopausal, A.S.A. physical status I or II non-pregnant females undergoing out-patient laparoscopic surgery to evaluate the efficacy and safety of metoclopramide 50 mg administered intravenously at induction of anaesthesia. This design would allow a non-parametric analysis.

Accordingly, 90 informed consenting females were randomly assigned to three groups.

Group I received 10 ml in normal saline in a numbered ampoule. Group II received 20 mg of metoclopramide and Group III received 50 mg of metoclopramide each in numbered ampoules.

Standard anaesthetic technique was employed and patients were monitored postoperatively until discharged. Any complaint of nausea or sign of vomiting was counted as a failure.

After the 90 cases were completed, the code was broken and the results analysed by Chi-square (Table). No dystonias were noted in the series.

This study strongly suggests that there is a dose-dependent pharmacodynamic difference in the anitemetic action of metoclopramide, and that 50 mg in females is equivalent to 20 mg in males.

References

1 *Diamond, MJ, Keeri-Szanto M.* Reduction of postoperative vomiting by preoperative oral metoclopramide. Can Anaesth Soc J 1980; 27: 1.

2 *Gralla R et al.* High dose metoclopramide. N Engl J Med 1981; 305: 905–9.

Evaluation of transdermal scopolamine for the reduction of nausea associated with epidural morphine in postoperative patients

K. Loper, B. Dorman, L.B. Ready

Department of Anesthesiology, University of Washington, Seattle, Washington

Nausea is a commonly reported side effect of epidural morphine for postoperative analgesia. Scopolamine, given intramuscularly, is an effective postoperative antiemetic against morphine.[1] The rapidity of intramuscular absorbtion, however, leads to a high incidence of dose-related adverse effects. Further, scopolamine's short half-life results in the rapid dissipation of its action. The transdermal unit delivers scopolamine at a slow, steady rate, thus reducing the incidence of its adverse effects and prolonging the duration of its therapeutic action.[2] This study evaluates the use of transdermal scopolamine for the reduction of nausea associated with epidural morphine in postoperative patients.

Methods

The patch is a laminated structure comprised of a backing layer, a drug resevoir containing scopolamine, 1.5 mg, a rate-controlling mebrane, and an adhesive layer (which contains 0.14 mg of scopolamine as a priming dose). The unit is designed to release scopolamine at 5 $\mu g \cdot hr^{-1}$ over a three-day period. Placebos are identical, but contain no scopolamine.

Twenty healthy adult women, undergoing major gynaecological surgery, were studied in a double-blinded, randomized manner. Following institutional approval, informed consent was obtained. The patients were instructed to request antiemetic medication if they experienced nausea.

Prior to surgery, a patch was placed on the postauricular skin and a T_{4-6} block established via a lumbar epidural catheter with lidocaine 2 per cent. General anesthesia was then induced with thiopentone (4–6 $mg \cdot kg^{-1}$), and maintained with isofulurane (0.5–0.9 per cent). Nitrous oxide and atropine were avoided. The first dose of epidural morphine (3–6 mg) was administered intraoperatively; subsequent doses were administered postoperatively every six to eight hours. Patients were prescribed, prn for nausea, metoclopromide 10 mg IV two-hourly, four times; then droperidol, 1.25 mg IV, two-hourly, two times.

After 24 hours, nausea was scored by having the patient mark an "X" on a 100 mm line between "No Upset Stomach" (0) and "Extremely Upset Stomach" (100). The administration of antiemetic drugs were noted. Statistical analysis utilized the t test and Pearson's Correlation for parametric data, and the Mann-Whitney test and Spearman Rank for nonparametric data. Significance was assigned at $p < 0.05$.

Results

In the scopolamine and placebo groups, the respective mean values (±SD) were: age, 40 (±15) and 48 (±16) years; weight, 76 (±25) and 69 (±7) kg; duration of operation, 243 (±103) and 201 (±90) minutes; per cent isoflurane concentration, 0.5 (±0.3) and 0.6 (±0.3); morphine dosage, 4 (±1) and 4 (±1) mg. There are no statistical differences in any of these categories.

The nausea scores were significantly different with scopolamine patients reporting 1 (±2) vs 29 (±40) for placebo ($p < 0.05$). There was a similar significant difference in the respective number of antiemetic medications requested: 0.2 (±0.4) vs 1.6 (±2.4) ($p < 0.05$). The nausea score and the number of medications requested was the only significant correlation ($p < 0.01$).

Discussion

There are numerous factors that affect both the incidence and severity of postoperative nausea and vomiting.[3] In comparing the patients who received a scopolamine patch to those who received a placebo patch, there were no demographic or procedural differences. Thus this study demonstrates that transdermal scopolamine significantly reduces the nausea reported by postoperative patients receiving epidural morphine.

References

1 *Clarke et al.* Studies of drugs given before anaesthesia. Br J Anaesth 1965; 37: 772–7.
2 *McCauley et al.* Effect of transdermally administered scopolamine in preventing motion sickness. Aviat Space Environ Med 1985; 56: 777–82.
3 *Palazzo et al.* Anaesthesia and emesis. Can Anaesth Soc J 1984; 31: 178–87.

Full stomach: assessing the risk of regurgitation in the ferret

A. Hutchinson, P.I. Collman, A. Ewen, J.R. Maltby
Departments of Anaesthesia and Gastroenterology, Foothills Hospital at the University of Calgary, Calgary, Alberta

Regurgitation and pulmonary aspiration of gastric contents is an ever-present risk during induction of anaesthesia. Although approximately 40 per cent of elective patients have gastric fluid volume > 25 ml with pH < 2.5, acid aspiration syndrome is rare (0.01 per cent).[2] Patients at increased risk of regurgitation are those with a "full stomach," oesophageal reflux, delayed gastric emptying, and obstetrical patients. Atropine, either as premedication or at induction, may further increase the risk by lowering the tone of the lower oesophageal sphincter. In experiments on cats in which the duodenum was occluded, 8–41 $ml \cdot kg^{-1}$ gastric fluid was required to produce regurgitation under ketamine anaesthesia.[3] In this study, we observed the effect of anaesthesia immediately after filling the stomach with radiopaque material in ferrets in which the duodenum was not occluded.

Methods

Under general anaesthesia two gastrostomy catheters, 0.75 mm internal diameter, and an indwelling central venous line were inserted into healthy adult ferrets ($n = 7$). One week later the animal was sedated with IV diazepam 0.5–2.0 mg, and 50 ml barium emulsion was infused over a period of five minutes through one of the gastrostomy catheters. Through the other, the baseline intragastric pressure was recorded using a two-channel Gould recorder. IV atropine 0.3 mg was then given to produce total gastrointestinal stasis. Using a plastic infusor bag around the ferret's abdomen, external abdominal compression to 100 mmHg was exerted to maintain an intragastric pressure greater than 30 mmHg for 15 sec, and then released. Anaesthesia was induced using halothane in oxygen (day one) and thiopentone (day two) to a depth at which there was no reponse to a painful stimulus and the pressure again exerted. Each animal was observed fluroscopically throughout the procedure.

Results

Gastric emptying and intestinal propulsive activity began within one minute from the start of gastric filling. There was a minimal rise in intragastric pressure during filling. Peristalsis stopped abruptly and completely one minute after the atropine injection. No regurgitation was observed in any animal under any circumstances. In two animals an emetic stimulus of intragastric hypertonic saline produced neither vomiting nor regurgitation at surgical levels of anaesthesia. However, these animals actively vomited during emergence from anaesthesia but no barium entered the tracheo-bronchial tree.

Discussion

During general anaesthesia without neuromuscular blockade or tracheal intubation the lower oesophageal sphincter effectively prevented regurgitation of stomach contents in the presence of gastric distension and external compression. These results suggest that additional factors may be present when passive regurgitation occurs during general anaesthesia.

References

1 *Sutherland AD, Maltby JR, Sale JP, Reid CRG.* The effect of preoperative oral fluid and ranitidine on gastric fluid volume and pH. Can J Anaesth 1987; 34: 117–21.
2 *Coombs DW.* Aspiration pneumonia prophylaxis. Anesth Analg 1983; 62: 1055–8.
3 *Plourde G, Hardy JF.* Aspiration pneumonia: assessing the risk of regurgitation in the cat. Can Anaesth Soc J 1986; 33: 345–8.

The effect of autotransfusion of mediastinal shed blood following cardiac surgical operations of blood transfusion requirements

R.F. Seal, G.T. Manning, G.H. Growe, L.C. Jenkins
Department of Anaesthesia, University of British Columbia and Vancouver General Hospital, Vancouver, British Columbia

The current AIDS epidemic has increased physician awareness of transfusion medicine and the risks of homologous blood transfusion. Additionally, the medical profession has become more cost-conscious and efforts are being made to use valuable resources such as banked blood wisely. Previous studies of the ability of autotransfusion (AT) of mediastinal shed blood (MSB) following cardiac surgery to reduce the use of banked blood have yielded conflicting results.[1-4] To attempt to demonstrate an advantage of routine postoperative transfusion of MSB as determined by a protocol over our present selective utilization of autologous MSB, a comparison was made between our current practice and that during time periods when trials were made of two autotransfusion devices (Pleur-evac ATS,* and Sorenson Receptaseal†).

Methods

Three groups of consecutive cardiac surgical patients were studied: 28 in the baseline group, 37 in the Pleur-evac group, and 25 in the Sorrenson group. Patients were to be autotransfused with MSB if more than 200 ml had been collected in a four-hour period. Recovered blood which remained in the container for more than four hours was discarded and a new container inserted. The volume of MSB and the autotransfused volume were recorded for the first four hours, second four hours, and the first 24 hours postoperatively. Homologous blood product usage for the operative day (packed cells only) and for the entire hospital stay were obtained from the blood bank records. Haemoglobins were analyzed preoperatively, immediately postoperatively, on the morning of the first postoperative day, and at time of discharge. Analysis of variance was used for intergroup comparisons.

Results

Data from the three groups is presented in Table I which showed them to be comparable in terms of age, percentage of revascularization procedures, incidence of prior cardiac operation, perfusion time, cross-clamp time, and postoperative mediastinal blood loss. Additionally, there were no differences in the haemoglobin determinations between the three groups (see Table II). Table III indicates the percentage of patients autotransfused, the average volumes of autotransfusion for each time period, and the use of homologous blood products for each group. Although the mean number of units of packed red blood cells (PRBC) used in first 24 hours in the autotransfused groups were less than in our baseline group, these differences did not achieve statistical significance. As well, comparison of total PRBC, frozen plasma, stored plasma, and platelets use showed no statistically significant difference between the groups.

TABLE I Group characteristics

	Baseline group	*Pleurevac group*	*Sorrenson group*
Number in group	28	37	25
AGE mean	59 ± 9	56 ± 17	58 ± 14
range	36–74	17–79	21–84
Males	24 (86%)	27 (73%)	16 (64%)
Females	4 (14%)	10 (27%)	9 (36%)
Revascularization	20 (71%)	24 (65%)	17 (68%)
Other procedures	8 (29%)	13 (35%)	8 (32%)
Valves	6	2	4
Combined/other	2	11	4
Prior cardiac sx	4 (14%)	4 (11%)	4 (16%)
Perfusion time	154 ± 69	156 ± 65	164 ± 55
Cross-clamp time	100 ± 52	107 ± 48	133 ± 46
Mediastinal blood loss (ml)	1295 ± 2023	1108 ± 1092	1078 ± 846

TABLE II Results. Haemoglobin concentrations ($g \cdot L^{-1}$)

	Baseline group	*Pleurevac group*	*Sorrenson group*
Preoperative	138 + 11	139 + 14	137 + 13
Postoperative	98 + 15	98 + 20	107 + 14
Post-op day 1	97 + 13	102 + 19	103 + 16
Discharge	102 + 10	107 + 23	111 + 14

TABLE III Results. Transfusion volumes

	Baseline group	*Pleurevac group*	*Sorrenson group*
Per cent autotransfused	18	81	72
Volume autotransfused			
First 4 hours (ml)	59 ± 156	283 ± 261	323 ± 310
Second 4 hours (ml)	7 ± 37	150 ± 300	163 ± 240
First 24 hours (ml)	70 ± 203	585 ± 1065	634 ± 640
Homologous blood			
Packed RBC's (units)			
Operative day	5.3 ± 6.7	4.1 ± 4.6	2.8 ± 2.7
Entire admission	6.3 ± 7.1	5.6 ± 5.2	3.6 ± 3.4
Frozen plasma (units)	3.2 ± 6.5	2.2 ± 2.2	2.1 ± 3.5
Stored plasma (units)	4.2 ± 3.7	4.3 ± 4.3	2.4 ± 2.8

Discussion

In our study we were unable to demonstrate that the protocolized AT of mediastinal shed blood reduced utilization of blood bank products when compared to our pre-existing selective system. Previous studies demonstrated a saving of banked blood when comparing postoperative AT to no AT.[1,2] Ability to demonstrate a saving of blood bank products may be difficult in the face of the many methods of conservation used during the perioperative period in cardiac patients.[3] Johnson *et al.*[4] using case matched

* Deknatel Division of Pfizer Hospital Products Group, Inc. Floral Park, New York.

† Sorrenson Research Corporation, Salt Lake City, Utah.

controls failed to demonstrate a reduced requirement for banked blood when they examined an entire group receiving postoperative AT according to a protocol, but did demonstrate as saving when they selected only the patients who actually received MSB. Perhaps a similar examination in our study would have been helpful. While previous studies support the efficacy of using postoperative AT it remains unknown if routine connection of an autotransfusion device has an advantage over more selective use.

References

1 *Schaff HV, Hauer JM, Brawley RK.* Autotransfusion in cardiac surgical patients after operation. Surgery 1978; 84: 713–8.

2 *Schaff HV, Hauer J, Gardner TJ et al.* Routine use of autotransfusion following cardiac surgery: experience in 700 patients. Ann Thor Surg 1979; 27: 493–9.

3 *Thurer RL, Lytle BW, Cosgrove DM, Loop FD.* Autotransfusion following cardiac surgical operations: a randomized prospective study. Ann Thor Surg 1979; 27: 500–7.

4 *Johnson RG, Rosenkrantz KR, Preston RA, Hopkins C, Daggett WM.* The efficacy of postoperative autotransfusion in patients undergoing cardiac operations. Ann Thor Surg 1983; 36: 173–9.

The safety of nitrous oxide for patients with coronary artery disease: a laboratory investigation

H.J. Nathan

Department of Anaesthesia, University of Ottawa Heart Insitute, Ottawa Civic Hospital, Ottawa, Ontario

A previous study from this laboratory compared two equipotent anaesthetics in the dog; 50 per cent nitrogen with 1.8 per cent isoflurane (ISF) and 50 per cent N_2O with 1.4 per cent ISF. The use of N_2O resulted in increases of five per cent in heart rate (HR) and eight per cent in systolic BP (SBP). In an ischaemic region of the left ventricle systolic shortening (SS) was diminished by 19 per cent and subendocardial/subepicardial blood flow (I/O) ratio fell by 30 per cent. The present study was designed to determine the mechanism whereby N_2O worsened ischaemia during isoflurane anaesthesia.

Methods

In 12 open-chest ISF anaesthetized dogs the proximal LAD coronary artery was cannulated and connected to an autoperfusion circuit on which an artificial coronary stenosis could be imposed. Subendocardial SS in the left anterior descending (LAD) and circumflex (CCx) regions was measured with a sonomicrometer. Regional myocardial blood flow was measured with radiolabelled microspheres. Measurements were made during the imposition of a stenosis sufficient to decrease SS by 30–50 per cent. The identical stenosis was imposed during three treatments (50 per cent nitrogen + 1.8 per cent ISF, 50 per cent N_2O + 1.4 per cent ISF, 50 per cent N_2O + 1.8 per cent ISF) in a randomized and balanced design. Using atrial pacing, a Fogarty catheter in the aorta, a femoral arteriovenous fistula, and a pressurized blood reservoir, HR, SBP and left atrial pressure (LAP) were held constant to match myocardial oxygen "demand" during the three anaesthetics.

TABLE Effect of N_2O with HR and SBP constant (mean ± SD)

Treatment	*CCx SS normal*	*LAD SS ischaemic*	*CCx I/O normal*	*LAD I/O ischaemic*
A N_2 + 1.8	18.1 ± 8.3	11.8 ± 7.2	1.08 ± 0.13	0.46 ± 0.20
B N_2O + 1.4	18.5 ± 8.0	11.8 ± 7.5	1.02 ± 0.17	0.45 ± 0.18
C N_2O + 1.8	14.4 ± 8.4	8.4 ± 5.9	1.07 ± 0.14	0.46 ± 0.18
A–B %diff	2%	0%	6%	2%
A–C %diff	23%†	34%*	1%	0%

See text for abbreviations.
*$p < 0.01$, †$p < 0.001$ by paired t test.

Results

The Table shows the values of SS and I/O ratio for the three treatments during which HR, SBP, and LAP were matched.

Discussion

In the present study, HR, SBP, and LAP were held constant, and there was no change in SS or I/O ratio comparing 50 per cent nitrogen with 1.8 per cent ISF and 50 per cent N_2O with 1.4 per cent ISF. This indicates that the worsening of ischaemia observed in the previous study was caused by increased myocardial oxygen demand and not by a direct effect on N_2O on the heart or coronary circulation.

When N_2O was added to 1.8 per cent ISF there was a decrease in SS of similar magnitude in both ischaemic and normally perfused myocardium with no change in I/O ratio. This indicates that, at a constant level of ISF, the addition of N_2O caused myocardial depression not specific to ischaemic myocardium and without adverse effect on myocardial blood flow distribution.

If extrapolated to humans these findings suggest that, so long as blood pressure and heart rate are maintained at an appropriate level, nitrous oxide can safely be used for patients with coronary artery disease.

The treatment of hypertension following coronary artery surgery: a comparison of intravenous nifedipine and sodium nitroprusside

H.J. Nathan, M. Harrison, L. Dubé, J. Sweet, C. Foster, M. Bourke, C. Cattran, G. de la Salle, J. Robblee

Department of Anaesthesia, University of Ottawa Heart Institute, Ottawa Civic Hospital, Ottawa, Ontario

Calcium entry blockers, having anti-anginal action, seem ideally suited for the treatment of hypertension in patients with coronary artery disease. Although interest is sufficient that IV preparations may be released for general use, studies comparing these agents to standard treatment are lacking. We present results from a randomized clinical trial comparing the safety and efficacy of IV nifedipine (NIF) and sodium nitroprus-

side (SNP) when used to control hypertension following coronary artery bypass surgery (CABG).

Methods

Eighty-five consenting patients with good LV function scheduled for elective CABG received a fentanyl-based anaesthetic. Of the 39 who became hypertensive postop, 24 received IV NIF and 15 SNP. Hypertension was defined as a MAP over 100 mmHg. The goal was to maintain MAP at 80 mmHg; if MAP rose to 90 mmHg despite maximal dose the other drug was added. SNP was infused up to 8 $\mu g \cdot kg^{-1} \cdot hr^{-1}$. NIF was given in stepwise increments according to response. The maximum was three five-minute infusions of 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$ followed by 1 $\mu g \cdot kg^{-1} \cdot min^{-1}$ until the hypertension remitted. This regimen was arrived at after seven of the first 11 patients who received a lower maximal dose required the addition of SNP.

Haemodynamic variables, leads II and V_5 on Holter monitor, and NIF plasma concentrations ([NIF]) were measured. Two and 24 hr postop ECG and serial CK-MB fractions were collected. (NIF) was measured by gas–liquid chromatography with electron capture detection. The pharmacokinetic profile was determined from steady-state values.

Results

Of 13 patients treated with the final dose regimen of NIF, ten were controlled with NIF alone (rate ± 95 per cent confidence interval = 77 ± 23 per cent); three required the addition of a low dose of SNP (mean = 0.80 $\mu g \cdot kg^{-1} \cdot min^{-1}$ ± SD = 0.55) to keep MAP below 90 mmHg. One of 15 patients in the SNP group required addition of NIF (response rate = 93 ± 13 per cent).

The mean plasma concentration of NIF at steady state in responders was 118 $ng \cdot ml^{-1}$ (CV = 28 per cent). Differences in response were not explained by failure to reach the expected plasma level. Plasma clearance (Clp) ranged from 0.29 to 0.54 $L \cdot hr^{-1} \cdot kg^{-1}$, the volume of distribution (Vd-beta) ranged from 0.47 to 1.35 $L \cdot kg^{-1}$ and the elimination half-life was 1.5 ± 0.6 hr (mean ± SD).

The haemodynamic profile was similar for both groups. The level and variability of MAP was significantly less when NIF was used ($p < 0.05$, ANOVA). There were no episodes of hypotension requiring treatment in either group.

ST-segment analysis showed a trend toward less ischaemia in patients treated with NIF. No patient in either group required inotropic support. There were no deaths.

Discussion

We have developed an intravenous dose regimen for NIF that is safe and effective in maintaining MAP below 90 mmHg in the majority of patients. The haemodynamic profile was similar when SNP was used in the same way. The pharmacokinetics of NIF in this group of patients were similar to those of normal volunteers. These findings suggest that IV NIF can be a practical treatment for acute hypertension. Our early results encourage further study to determine with confidence if NIF has a more favourable effect on myocardial blood flow than SNP in these patients.

Incidence of cell saver contamination in patients undergoing cardiopulmonary bypass

I.M. Schwieger, C.J. Gallagher, D.C. Finlayson, J.A. Bryan, W. Daley

Departments of Anesthesiology and Pathology-Laboratory Medicine, Emory University School of Medicine, Atlanta, Georgia

Infections are a major complication in cardiac surgical patients. Regular bacteriologic surveillance of all apparatus used to treat patients undergoing cardiopulmonary bypass (CPB) is necessary to ensure that the patients are not exposed to risks of infection. The heart-lung machine has been studied as a source of infection. In contrast, the cell saver apparatus (CSA) (Hemonetics® – Hemonetics Corporation, 400 Wood Road, Braintree, Massachusetts 02184) has not been evaluated. The purpose of this study was to determine if the CSA added to the risk of infection in cardiac surgical patients.

Methods

As part of our institutional bacteriologic surveillance of cardiac surgical patients, the CSA was studied in 19 patients undergoing procedures requiring CPB. After each operation, the effluent from the CSA was sterilely sealed and sent to Microbiology for culture, where 50 ml of fluid from each bag of effluent was aspirated using sterile technique, emptied into a sterile 50 ml plastic centrifuge tube, and spun at 2000 rpm for ten minutes.

The sediment was used to inoculate one sheep blood agar plate and one chocolate agar plate which were then incubated at 35° C in the presence of CO_2. Also inoculated were one MacConkey agar and one thioglycollate broth plate which were incubated at 35° C. These inoculates were examined daily for four days. The patients were examined for clinical signs of infection during their hospital stay and were cultured if clinical signs of infection appeared.

Nineteen patients, ages 35 to 77 years, were studied. Fifteen required coronary artery bypass grafting, and four valve replacements. The CSA was in use for the duration of the operation (309 ± 88 minutes mean ± SD), with suction pressures of 120 mmHg and CPB lasted 128 ± 34 minutes. All patients received prophylactic antibiotics, 17 1.0 g cefazolin and 2 1.0 g vancomycin. Two to 12 units (mean 4.5 ± 2.4) of autologous blood from the CSA were transfused to all 19 patients. A total of 21 bags of effluent from the CSA were sent for culture, one bag from each of 17 patients, and two bags from each of two patients. Since two anaerobic cultures and two aerobic cultures were made from each bag, a total of 42 aerobic and 42 anaerobic cultures were studied.

Results

Three of 42 anaerobic cultures were positive for diphtheroids, and one of 42 aerobic cultures positive for staphylococcus epidermidis. None of these four patients with positive cultures of the CSA effluent had infectious complications during their hospital stay. Five out of the remaining 15 patients developed infectious complications while hospitalized. Three of these had lower respiratory infections manifested clinically by a productive purulent cough. Sputum cultures grew Klebsiella in one and

normal oral flora in the other two patients. These episodes resolved and the patients were discharged with no sequelae. One patient suffered a stroke, required prolonged ventilatory support and developed a candida albicans pneumonia. One other died in septic shock on the third postoperative day. Though clinical parameters indicated septic shock, blood cultures were negative. Of these 5/15 patients who developed clinical signs of infection, none had positive cultures from the CSA.

Discussion

Patients undergoing cardiac procedures can suffer significant morbidity with infection. The CSA is used in cardiac surgical patients to provide autologous blood for transfusion. These autologous transfusions are well tolerated haemodynamically, avoid the risk of isoimmunization to blood components, and reduce the transmission of blood borne diseases.[1] None of the patients with positive inoculates developed infectious complications. None of the patients with infectious complications had positive inoculates. We conclude that the CSA does not appear to contribute to infectious risk in cardiac surgical patients, and that the CSA is a safe adjunct to cardiac surgery.

Reference

1 *Council of Scientific Affairs of the AMA*. Autologous Blood Transfusions. JAMA 1986 256: 2378–80.

Comparative study of labetalol and sodium nitroprusside in the treatment of hypertension post coronary bypass surgery

Y. Skrobik, R. Webster, A. Marquez-Julio, T. David, C. Cruise

Departments of Anesthesia, Internal Medicine and Cardiovascular Surgery, Toronto Western Hospital, Toronto, Ontario

Labetalol is a new antihypertensive drug with combined alpha- and beta-adrenergic blocking properties. The effectiveness and safety of this drug in the control of hypertension post-coronary artery bypass grafting (CABG) is controversial.[1,2] Sodium nitroprusside (SNP) has been shown effective in this situation, owing to its rapid onset, short duration of action and ease of titration.[3] In this study, we compared the haemodynamic effects of labetalol with sodium nitroprusside post-CABG.

Methods

Following institutional aproval, informed consent was obtained preoperatively from 42 consecutive patients scheduled for CABG at Toronto Western Hospital over an eight-week period. No attempt was made to influence pre- or intraoperative anaesthetic or surgical management.

Postoperatively, following a 30-minute stabilization period, anti-hypertensive therapy, if any, was discontinued and the blood pressure allowed to rise over a subsequent 30-minute period.

If the systolic blood pressure (SBP) exceeded 140 mmHg or the mean arterial pressure (MAP) exceeded 90 mmHg, control

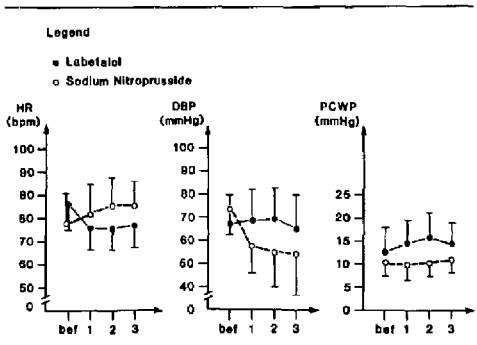


FIGURE Values for HR, DBP, and PCWP before administation of either drug ("bef") and at 10 (1) 20 (2) and 30 (3) minutes after initiation of drug infusion.

haemodynamic measurements were obtained including SBP, MAP, diastolic blood pressure (DBP), heart rate (HR), cardiac output (CO), cardiac index (CI), stroke volume (SV), systemic vascular resistance (SVR), pulmonary capillary wedge pressue (PCWP), central venous pressure (CVP), and left ventricular stroke work index (LVSWI). Patients were randomly allocated to one of two treatment groups. Group I (ten patients) received SNP (50 mg in 250 ml D5W) intravenously by infusion (initial dose 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and adjusted in 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ increments), while Group II (ten patients) received labetalol (200 mg in 200 ml D5W) by intravenous infusion at 2 $mg \cdot min^{-1}$ to a maximum of 300 mg.

When adequate blood pressure control was obtained (SBP less than 120 mmHg or MAP less than 80 mmHg), haemodynamic measurements were repeated in triplicate, at ten-minute intervals to ensure stability of control. The data were analyzed using Student's test, analysis of covariance and repeated measures analysis of variance, where appropriate. p values <0.05 were deemed significant.

Results

There were no significant differences between the groups with respect to age, cross-clamp time, LV grade or number of vessels grafted. There were no statistically significant differences in the control haemodynamic data between Groups I and II, except cardiac output. Comparison of the groups post-antihypertensive treatment showed significant differences in HR, DBP and PCWP (Figure) as well as CI, and SVR ($p < 0.05$). SBP, LVSWI, and SV did not differ significantly.

Discussion

We experienced no difficulties attributable to administration of either drug in our patients. Systolic or mean blood pressure end points were easily achieved with both drugs. Preload, as measured by PCWP did not change over time in either group. HR, however, was significantly lower in the labetalol group and SBP was equally controlled in the two groups. These findings

suggest that labetalol may be preferred for decreasing myocardial oxygen consumption postoperatively. In addition, labetalol may allow for improved myocardial oxygen supply owing to the significantly higher DBP and lower HR, compared with the SNP group.

References

1 *Morel DR, Forster A, Suter PM.* I.V. labetalol in the treatment of hypertension following coronary-artery surgery. Br J Anaesth 1982; 54: 1191–6.

2 *Meretoja OA, Allonen H, Arola M, Laaksonen VO.* Combined alpha- and beta-blockade with labetalol in post heart surgery hypertension-reversal of hemodynamic deterioration with glucagon. Chest 1980; 78: 810–5.

3 *Stinson EB, Holloway EL, Derby G et al.* Comparative hemodynamic responses to chlorpromazine, nitroglycerin, and trimethaphan immediately after open-heart operations. Circulation 1975; 51 (Suppl): I-26–33.

Effect of volatile anaesthetic agents on the coronary circulation of the isolated heart

G.A. Blaise, S.G. Lenis, D. Girard, C. Hollmann, R. Meloche

Department of Anesthesiology, University of Montreal, Notre-Dame Hospital, Montreal, Québec

Coronary arteriolar dilators can interfere with autoregulation and redistribute myocardial flow away from a potentially ischaemic area.[1] The effects of the volatile anaesthetic agents on the coronary circulation are still controversial. We determined the direct effects of three volatile anaesthetic agents at different concentrations on the coronary flow of an isolated non-working heart.[2] In this preparation the preload, the afterload, the heart rate and coronary perfusion pressure are controlled and constant. As the volatile anaesthetic agents depress myocardial contractility, myocardial oxygen consumption is reduced and coronary flow should diminish. Any increase in coronary flow in the preparation would be due to the direct dilatory effect of the anaesthetic agents on the coronary arterioles which regulate coronary flow.

Methods

A rat's heart was isolated and the ascending aorta was cannulated. The coronary arteries were perfused with an oxygenated Krebs-Ringer bicarbonate solution maintained at 37° C. The perfusion pressure was maintained at 85 cm H_2O and the heart rate was controlled with a pacemaker at 250/min.

After stabilization of coronary flow, the hearts were perfused with Krebs-Ringer bicarbonate solution containing one of the volatile anesthetic agents. Coronary flow was measured again. At the end of the experiments, we determined the effect of adenosine (10^{-6} M) on the coronary flow of the hearts perfused with Krebs Ringer solution containing a volatile anaesthetic.

Results

Each of the three volatile anaesthetic agents increased the coronary flow in a dose differentiated fashion. However, halothane at 2 MAC concentration was a much weaker coronary dilator than enflurane and isoflurane (Figure). At a 2 MAC concentration, isoflurane and enflurane increased the coronary flow 115 per cent and 85 per cent respectively and adenosine was unable to significantly increase the dilatation of the coronary vasculature. Halothane at 2 MAC increased the coronary flow by 50 per cent and adenosine was able to dilate the coronary vasculature thus preserving some coronary reserve (data not shown).

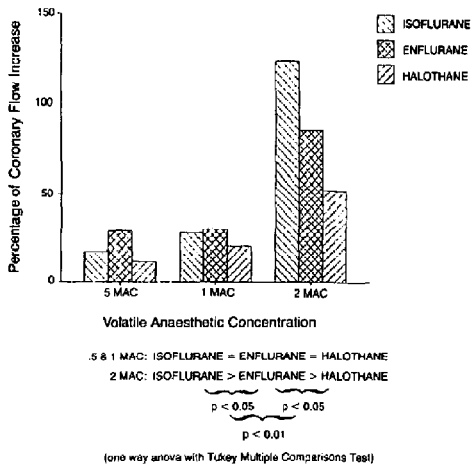


FIGURE Results.

Discussion

In normal hearts, 95 per cent to 98 per cent of the resistance to the coronary flow is at the arteriolar level.[3] Any increase in the coronary flow in our preparation was due to a direct effect of the anaesthetic agents on the coronary arterioles. The data shows that at a 2 MAC concentration, isoflurane and enflurane are potent coronary arteriolar dilators (isoflurane being more potent than enflurane). On the other hand, halothane is a much weaker coronary dilator and does not exhaust coronary reserve. These data suggest that isoflurane and enflurane at high concentrations have the potential to redistribute flow and thus should not be used at a concentration higher than 1.0 MAC in patients with coronary artery disease. At low concentrations, each of the volatile anaesthetic agents have an equally weak dilatory effect on the coronary arterioles.

References

1 *Warltier DC et al.* Am J Cardiol 1980; 46: 83–90.

2 *Ytrehus K et al.* Cardiovasc Res 1986; 20: 597–603.

3 *Young M et al.* Circ Res 1986; 59: 579–593.

Halothane and enflurane attenuate the K^+ induced contractile response of isolated canine coronary artery rings

G.A. Blaise, S.G. Lenis, D. Girard, C. Hollmann, R. Meloche
Department of Anesthesiology, University of Montréal, Notre-Dame Hospital, Montréal, Québec

A high concentration of extracellular K^+ depolarizes the cellular membrane and opens the action potential-operated calcium channel. Ca^{++} then flows into the cell, activates the contractile proteins and induces contraction. One of the actions of the calcium antagonists is to block the calcium channel and inhibit K^+ induced contraction.[1] It has previously been shown that isoflurane is not a calcium antagonist.[2] This protocol was designed to measure the effect of halothane and enflurane on the K^+ induced contraction of isolated coronary artery rings.

Methods

Epicardial coronary rings (4 mm long) were removed from the left circumflex coronary arteries of anaesthetized dogs; placed in organ chambers filled with Krebs-Ringer Solution at 37° and ventilated with a 95 per cent O_2 and 5 per cent CO_2 gas mixture. Rings were connected to a strain gauge for measurement of isometric contractions. Half of the rings were treated with halothane 1.5 MAC or enflurane 1.5 MAC. The other half served as control. Forty-five minutes later, we measured the contractile response to increasing concentrations of KCl. Data were expressed as a percentage of the maximal response to a previously administered standardized K^+ challenge (40 mM). Paired Student's t-test was used for analysis.

Results

Tension generated by isolated rings was plotted vs the K^+ concentrations (Figure). Enflurane depressed the contractile response. Halothane also depressed the KCl induced contractile response (data not shown)

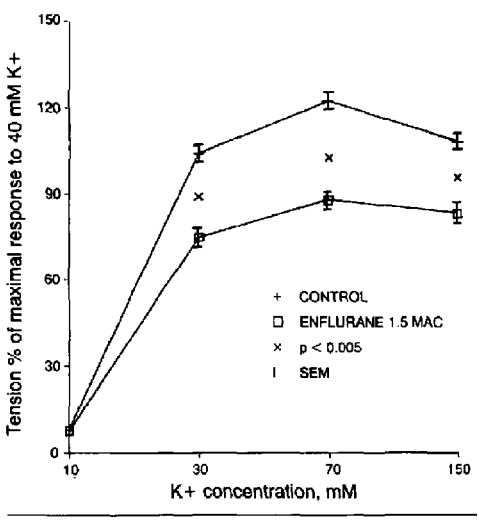


FIGURE Effect of volatile anaesthetics on contractile response.

Discussion

Our data show that contrary to isoflurane,[2] halothane and enflurane interfere with depolarization-induced contraction, acting like calcium antagonists. We are conducting further studies to determine whether halothane and enflurane block the calcium channels or interfere with the intracellular mechanisms regulating the contraction. The effects of halothane and enflurane on KCl-induced contraction is weak compared to nifedipine and nisoldipine. These two calcium blockers at a concentration of 10^{-6} M can completely suppress the contraction induced by membrane depolarization in isolated coronary rings.[2] These preliminary data suggest that halothane and enflurane could reduce coronary artery spasm

References

1 *Godfraind T.* J Pharmacol Exp Ther 1983; 224: 443.
2 *Blaise GA et al.* Anesthesiology 1986; 65: A330.

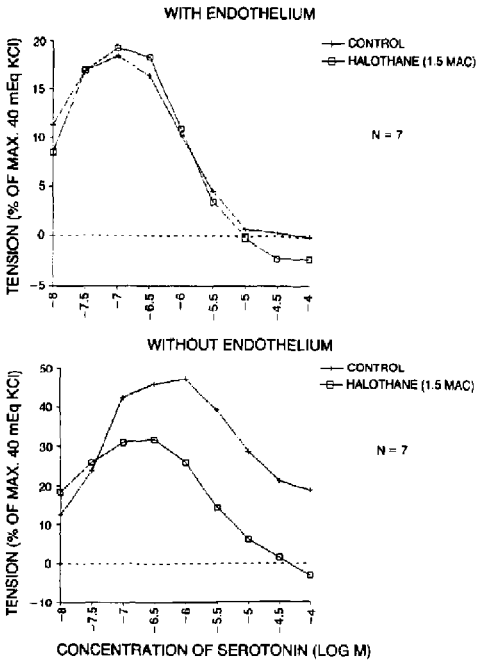
Halothane attenuation of the response of canine coronary artery rings to serotonin: role of the endothelium

G.A. Blaise, D. Girard, C. Hollmann, R. Meloche
Department of Anesthesiology, University of Montréal, Notre-Dame Hospital, Montréal, Québec

Increased coronary tone or coronary spasm are features of coronary artery disease. Coronary artery dilators are commonly used to relieve myocardial ischaemia. However, only direct smooth muscle dilators like nitroglycerine are effective. The indirect ones, such as acetylcholine, bradykinin, etc., which stimulate the release of vasodilators by the endothelium (EDRF, prostacyclin)[1] are ineffective because the endothelium is destroyed or its function is impaired by arteriosclerosis[2] Isoflurane attenuation of the contractile response of canine coronary artery rings stimulated by some spasm mediators is endothelium dependent. It is then difficult to predict its effect on coronary artery tone in patients with coronary artery disease.[3] In this study we determined the effect of halothane on the response of intact and denuded (without endothelium) canine coronary rings stimulated by serotonin (a putative mediator of coronary spasm).

Methods

Hearts were removed from anaesthetized dogs (18–28 kg). The coronary arteries (circumflex and LAD) were dissected and cut into 5 mm rings. The endothelium was mechanically removed from some of the rings. The rings were suspended between two stirrups in a 25 ml organ chamber filled with Krebs-Ringer solution maintained at 37° C. The chambers were ventilated with 95 per cent O_2 and five per cent CO_2. The upper stirrup was connected to a transducer and recorder for tension recording. The vessels were progressively stretched by steps of 2 g to their optimum passive tension for which the active response of the vessels to a stimulation by 20 mM K^+ is maximum. Their



FIGURES Dose–response curve to serotonin. (Top: Figure 1, Bottom: Figure 2.) See Results.

response to 40 mM K^+ was measured and taken as reference for further contractile responses. After this initial preparation, the vessels were washed several times with the Krebs-Ringer solution and allowed to relax for 30 minutes. Halothane, at 1.3 per cent concentration (1.5 MAC in the dog) delivered by a calibrated vaporizer, was added to the gas mixture ventilating some of the chambers. The other chambers served as control. Once the vessels were sufficiently saturated with halothane serotonin was introduced by increasing doses in the organ chamber. Each dose was added at the maximum contraction of the preceeding one. The contractile response of the control intact and denuded rings was measured and compared to the response of the treated intact and denuded rings.

Results

Halothane depresses the contractile response of denuded but not of intact coronary rings to serotonin. In the figures the tension generated by coronary rings has been plotted against serotonin concentrations. Figure 1 shows that halothane has no effect on contractile response of intact vessels. Figure 2 shows halothane's attenuation of serotonin contractile response in vessels without endothelium. Fourteen paired rings (seven treated, seven control) contractile response were recorded. Each area under the curve was calculated and averaged in the control and treated group. A paired T test was used to analyse the data ($p < 0.05$).

Discussion

The data obtained show that halothane has a direct depressing effect on the coronary smooth muscles' contractile response to serotonin. Halothane does not attenuate the response of intact vessels to the mediator. This would suggest that halothane interferes with the release or the action of relaxing substances (EDRF, prostacyclin)[1] released by endothelium. Halothane may reduce coronary spasm at the arteriosclerotic lesion in patients with coronary artery disease.

References

1 *Vanhoutte PM et al.* Ann Rev Physiol 1986; 48: 307.
2 *Freiman PC et al.* Circ Res 1986; 58: 783–789.
3 *Blaise GA et al.* Anesthesiology 1987; 67; 61–65.

The efficacy of esmolol in the treatment of hypertension after aortic reconstructive surgery

F.E. Ralley, J.R. Ramsay, J.E. Wynands, J.P. O'Connor, J. Bilodeau
Department of Anaesthesia, McGill University and Royal Victoria Hospital, Montréal, Québec

Following aortic reconstructive surgery patients frequently require attenuation of hypertension and tachycardia with beta-adrenergic antagonists and/or vasodilators. Potential drawbacks to the use of vasodilators such as nitroprusside include reflex tachycardia, pulmonary venous admixture, decreases in diastolic blood pressure and augmentation of the pulse pressure. Esmolol, an ultra-short acting $beta_1$-adrenergic antagonist, has been shown to attentuate effectively tachycardia and hypertension associated with intubation. This study was designed to determine the efficacy of esmolol vs placebo in controlling hypertension and tachycardia in patients immediately after aortic reconstructive surgery.

Methods

With Ethics Committee approval and informed consent 13 patients scheduled for elective aortic reconstructive surgery were entered in the study. Patients with asthma, COPD, cardiac failure or recent myocardial infarction were excluded. Patients were premedicated with morphine and scopolamine and anaesthetized with fentanyl, N_2O and enflurane. Postoperatively if systolic blood pressure (SBP) was greater than 140 mmHg and heart rate (HR) greater than 60 beat · min^{-1} patients were randomized to receive either esmolol or placebo in a double-blind fashion. Three titration steps were employed, lasting 5 min each. A bolus of 25 mg of drug was given over 30 sec at the start of each titration period, followed by an infusion of 8 mg · min^{-1}, increasing to 16 mg · min^{-1}, then 24 mg · min^{-1} at the end of each 5 min period. HR and SBP were recorded every 1 min during the infusion and at 5, 15, 30 and 60 mins after the infusion was stopped. Further haemodynamic measurements were recorded prior to the start of the infusion, at the end of each titration period

and at 5, 15, 30, 45 and 60 mins post-infusion. If the SBP was not reduced by 15 per cent from control (pre-infusion) by the end of the third titration period a nitroprusside infusion was started. Data were analyzed with ANOVA for repeated measurements.

Results

Six patients received esmolol and seven placebo. There was no difference in the demographic data between the two groups. There was a significant decrease in HR in the esmolol group only at 1 min after the first two titrations, but the SBP was significantly lower in this group from 7 min into the infusion until the end of the third titration period. By the end of the infusion the SBP had decreased by 11 per cent in the esmolol group compared to an increase of 12 per cent in the placebo group (NS). This reduction in SBP was associated with a significant decrease in SVI (13 per cent) and CI (14 per cent) from control values. There were no changes in SVI or CI in the placebo group during the same period. SVR increased by 19 per cent in the esmolol group compared to only three per cent in the placebo group. All cases were considered therapeutic failures and immediately started on nitroprusside. Patients who had received esmolol had significantly lower heart rates for the 60 min following the titration period.

Discussion

Although esmolol was effective in preventing any further rise in SBP during the titration period, the magnitude of the reduction in SBP was not clinically satisfactory. Therefore esmolol is ineffective as the sole agent to control hypertension post-aortic surgery. Despite a reduction in SVI and CI, patients who received esmolol maintained their SBP by increasing their SVR, thus demonstrating a lack of any vasodilatory properties of esmolol. These results suggest that esmolol may not be a pure $beta_1$ adrenergic antagonist, but may also have some effect on $beta_2$ receptors causing the increase in SVR. The significantly lower HR seen in the esmolol group during the infusion of nitroprusside immediately post-titration suggests that esmolol may be a useful drug in combination with nitroprusside by reducing the reflex tachycardia often associated with this drug.

References

1 *Hertzer N.* Myocardial ischemia. Surgery 1983; 93: 97–101.
2 *Newsome LR, Roth JV, Hug CC, Nagle D.* Esmolol attenuates demodynamic responses during fentanyl-pancuronium anesthesia for aortocoronary bypass surgery. Anesth Analg 1986; 65: 45–6.

Comparison of nifedipine and nitroprusside in the treatment of post-coronary bypass hypertension

G. O'Leary, P. Birnbaum, R.D. Weisel, J.C. Mullen, P. Young, M. Madonik, J. Ivanov, S.J. Teasdale
University of Toronto, Toronto General Hospital, Toronto, Ontario

A prospective randomized trial was performed to compare nifedipine (NIF) and nitroprusside (SNP) for the treatment of hypertension (HT), defined as mean arterial pressure (MAP) >95 mmgH, after aorto-coronary bypass surgery (ACB).

Methods

Forty-four patients undergoing elective ACB with a preoperative ejection fraction (EF) >40 per cent gave written informed consent to the institutionally aproved protocol. Patients were maintained on their usual medications until the morning of surgery. Anaesthesia was induced and maintained with fentanyl (75–100 $\mu g \cdot kg^{-1}$), pancuronium and oxygen with ventilation adjusted to normocarbia. After arrival in ICU each patient received additional sedation and relaxants to stabilize and standardize postoperative conditions. Monitoring included radial arterial, Swan-Ganz and coronary sinus catheters, a Millar intraventricular micromanometer and nuclear angiograms. Left ventricular pressure volume loops were constructed. End–systolic pressures (ESP) and volumes (ESVI) and end–diastolic pressures (EDP) and volumes (EDVI) were obtained to assess systolic and diastolic function. After baseline measurements (B1) the response to volume loading (V1) (with five per cent albumin to raise the left atrial pressure (LAP) 2 mmHg) was assessed prior to treatment. When hypertension (HT) developed NIF (n = 25) was administered intranasally (20–80 mg) or SNP (n = 19) was administerd at an initial rate of 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Both drugs were titrated to lower MAP to 90 mmHg. The response to treatment (Rx) and repeat volume loading (V2) was assessed. The data were analyzed using analysis of variance, co-variance (ANOCOVA) and Duncan's multiple range tests.

Results

Treatment with both agents effectively reduced MAP (Figure 1). Heart rate (Figure 2) was equally increased ($p < 0.01$). SNP significantly increased EF and decreased ESVI before and after

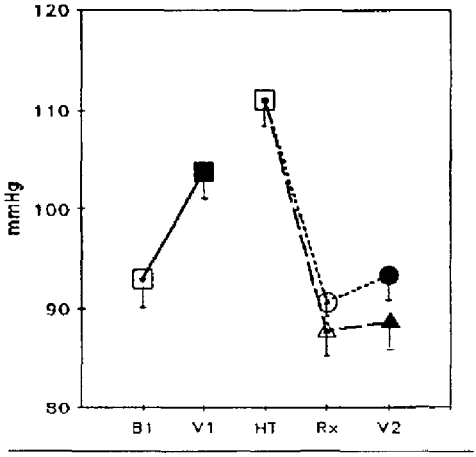


FIGURE 1 Mean arterial blood pressure (see figure 3 for definition of symbols).

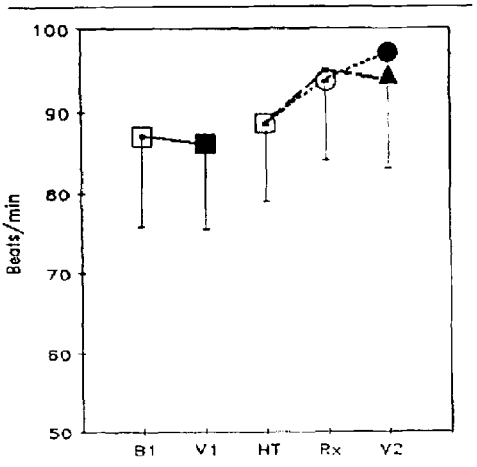


FIGURE 2 Heart rate (see figure 3 for definition of symbols).

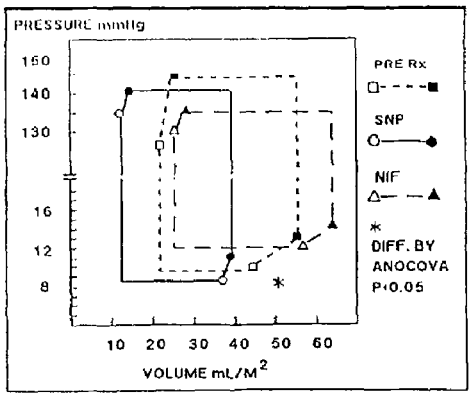


FIGURE 3 Systolic function.

volume loading, however NIF did not increase EF but increased ESVI. Therefore systolic function (Figure 3) remained elevated despite SNP therapy but was depressed with NIF. Diastolic compliance was significantly decreased with SNP because EDVI was lower with SNP than NIF at a similar EDP. No significant difference was seen between groups in coronary sinus blood flow, oxygen consumption or lactate flux (MVL); however, there was a trend towards lower MVL with SNP.

Conclusion

Both agents effectively treated post-ACB hypertension and failed to control reflex tachycardia. NIF decreased contractility. SNP induced an ischaemic decrease in diastolic compliance.

Myocardial performance after repair of congenital heart defects in infants and children: response to volume loading

F.A. Burrows, W.G. Williams, K.H. Teoh, J. Edmonds, R.D. Weisel

Departments of Anaesthesia, Cardiovascular Surgery and the Research Institute, The Hospital for Sick Children, University of Toronto, Toronto, Ontario

Intracardiac repair of congenital cardiac defects (CCD) using cardiopulmonary bypass (CPB) is performed with an acceptably low morbidity and mortality, but the procedure may affect immediate or long-term cardiac function. The effect of such repair on myocardial function in the hours immediately following CPB has not been examined. We investigated the haemodynamic response to increasing left atrial pressure (LAP) by volume loading in infants and children during the first 24 hours after repair of CCD.

Methods

After approval from the Committee on Human Research 70 infants and children undergoing elective repair of CCD using CPB were studied. The patients were grouped into four categories: atrial septal defect (ASD, n = 8); ventricular septal defect (VSD, n = 36); complete transposition following Mustard's repair (TGA, n = 13); and tetralogy of Fallot (TOF, n = 13). Radial arterial and pulmonary arterial catheters were inserted. Heart rate, systemic and pulmonary atrial pressures, mean arterial pressure and cardiac output (cardiogreen dye) were measured. Derived measurements were: cardiac index (CI), left ventricular stroke work index (SWI), and systemic vascular resistance (SVR) by standard formulae. Volume loading was performed by infusing 5–10 $ml \cdot kg^{-1}$ of a colloid solution to raise the LAP 5–10 mmHg over 20–30 minutes.[1] Measurements were made at 2, 4, 8, 12, and 24 hours postoperatively. The relationship between CI or SWI and LAP was evaluated by analysis of covariance. Differences between time periods and operative groups were specified by Duncan's multiple range and least squared means t tests. Multiple linear regression analysis was employed on the VSD group to determine the factors influencing maximum SWI attained by volume loading four hours post-CPB. Statistical significance was accepted as $p \leqq 0.05$.

Results

Within two hours of CPB both CI and SWI were adequate and increased appropriately with volume loading in the four groups. The ASD group had a similar response to volume loading 4 and 24 hours post-CPB. The other three groups had higher filling pressures, lower CI and SWI and a depressed response to increasing preload four hours postoperatively which was maximal between 4 and 12 hours post-CPB (Figure). Recovery was evident at 24 hours post-CPB. The TGA group had a more profound depression in cardiac performance than the other groups. Aortic cross-clamp time and smaller size (BSA < 0.36 m^2) were identified as independent predictors of maximum SWI.

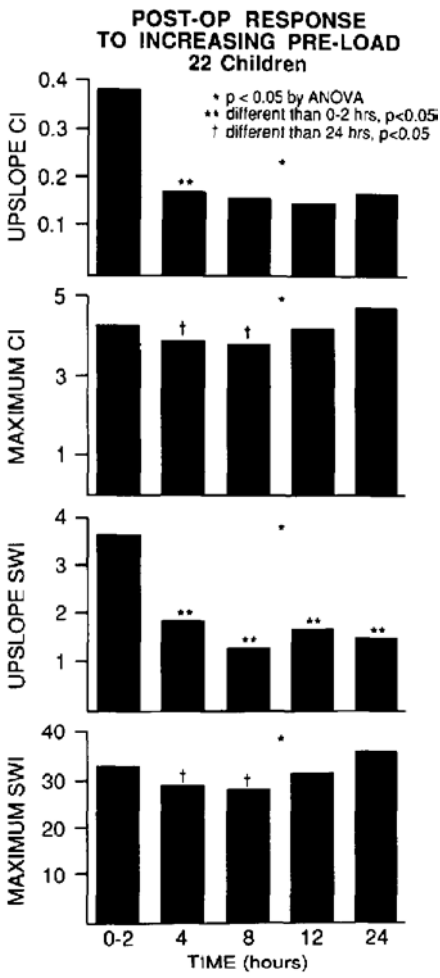


FIGURE Average values are plotted to show trends in performance in 22 children post-VSD repair. Maximum depression of cardiac performance is demonstrated between 4 h and 24 h post-CPB with subsequent improvement at 24 h but persistant depression compared to 2 h.

Discussion

These results demonstrate a significant alteration in cardiac performance during the first 24 hours after repair of CCD. These changes should be considered when planning postoperative management.

Reference

1 *Benzing G, Helmsworth J, Schrieber JT, Kaplan S.* Cardiac performance and oxygen consumption during intracardiac operations in children. Ann Thorac Surg 1976; 22: 176–81.

Accepted for publication by the Journal of Thoracic and Cardiovascular Surgery.

The effect of halothane on the stunned myocardium

S. Belo, C.D. Mazer
St. Michael's Hospital, University of Toronto, Toronto, Ontario

The term "stunned myocardium" has been coined to describe the reversible dysfunction that persists following reperfusion of a transiently ischaemic heart.[1] This study examines the effect of halothane on post-ischaemic ventricular dysfunction in an open-chest dog model using left anterior descending artery (LAD) occlusion. Regional myocardial function was assessed by measurement of systolic shortening using ultrasonic crystals.

Methods

Five mongrel dogs (20–30 kg) were anaesthetized with intravenous sodium pentobarbitone (30 $mg \cdot kg^{-1}$ bolus plus 2 $mg \cdot kg^{-1} \cdot hr^{-1}$ infusion) and fentanyl (23 $\mu g \cdot kg^{-1}$ bolus plus 1 $\mu g \cdot kg^{-1} \cdot hr^{-1}$). The animals were paralyzed with pancuronium and ventilated to maintain normocapnia. A left thoracotomy was performed and the LAD was dissected distal to the first major diagonal branch. Two pairs of piezo-electric ultrasonic crystals were implanted in the subendocardium – one pair in the area supplied by the LAD to be stunned, and the other in the distribution of the circumflex (CIRC) artery as a control. Systolic shortening (SS) was determined by the equation SS = (EDL – ESL)/EDL, where EDL equals end–diastolic length and ESL equals end–systolic length. All values are expressed as per cent of the pre-ischaemic control. To produce an area of stunned myocardium, the LAD was occluded for 15 minutes and then reperfused. The effect of two doses of halothane (0.75 MAC and 1.5 MAC) on stunned and normal myocardium was measured during reperfusion in each animal. The end–tidal concentration of halothane was held constant for at least ten minutes prior to data acquisition. Heart rate and arterial blood pressure were held near control levels during the experiment by atrial pacing and variable occlusion of the descending aorta with umbilical tape. Data were analyzed using repeated measures analysis of variance and Duncan's multiple range test.

Results

LAD occlusion produced significant decreases in systolic shortening in the LAD area which persisted during reperfusion ("stunned" myocardium) – see Table. The amount of systolic dysfunction produced was variable between animals, with some showing systolic bulging (or negative systolic shortening). Halothane caused decreases in systolic shortening in both the LAD (stunned) and CIRC (normal) regions in every experiment. The amount of myocardial depression of the stunned myocardium caused by halothane did not correlate with either the degree

TABLE Systolic shortining (per cent control) – mean ± SD

	15 Min reperf	*Hal 0.75 MAC*	*Hal off*	*Hal 1.5 MAC*	*Hal off*
LAD	51.2 ±48.0	15.4* ±28.4	71.3 ±53.9	3.0* ±19.6	60.6 ±52.9
CIRC	118.7 ±33.1	88.4* ±27.8	111.7 ±21.6	78.8* ±21.2	106.4 ±18.0

*$p < 0.01$ vs 15 min reperf (n = 5).

of dysfunction during occlusion or the amount of recovery at 15 minutes of reperfusion.

Discussion

Halothane causes a decrease in systolic shortening of the stunned myocardium. This myocardial depressant effect is dose-dependent, but does not seem to be related to the amount of dysfunction during or immediately after LAD occlusion. Although it appears that the myocardial depressant effects of halothane may be greater in the stunned myocardium, because of the small number of animals studied, further experiments are required to support these conclusions.

Reference

1 *Braunwald E, Kloner RA.* The stunned myocardium: prolonged post-ischemic ventricular dysfunction. Circulation 1982; 66: 1146–9.

Delayed hypoxaemia during recovery from anaesthesia

G. Lim, M.I. Skinner, E.A. Rose, R.L. Knill
University of Western Ontario, London, Ontario

Following inhalational anaesthesia, patients breathing air may have a reduced arterial oxygen tension for up to 2 hr. The purposes of this study were to (1) determine the time course of this impairment of oxygenation and (2) assess its relation to ventilation, ventilatory pattern and state of consciousness.

Methods

We studied ten ASA physical status I non-smoking patients, age 31 ± 6 yr, wt 66 ± 8 kg and ht 168 ± 8 cm; six females undergoing tuboplasty or hysterectomy and four males undergoing orthopaedic knee surgery. All gave informed consent. Preoperatively, oxygen saturation (SaO_2) was measured by ear oximetry (Biox IIA) with the patient supine. Anaesthesia included thiopentone, N_2O, isoflurane and fentanyl, and pancuronium as required. Ventilation was controlled to keep end-tidal CO_2 close to 40 mmHg. Procedures lasted 85 to 180 min. Commencing about 10 min after arousal (first response to command) in the recovery room and throughout the subsequent 2 hr, we monitored (1) SaO_2 by ear oximetry, (2) transcutaneous CO_2 (tCO_2) with a Radiometer TCM2O, and (3) ventilation ($\dot{V}_E$), tidal volume (V_T), and breathing frequency (f) with a calibrated Respitrace, and (4) wakefulness/sleep by EEG (C4-A1) and EOG. Morphine 1–2.5 mg was given IV q. 5 min p.r.n. for pain. Oxygen was discontinued at arousal and then given only if SaO_2 fell to 85 per cent. Patients were otherwise cared for in the usual fashion, remaining in a lateral and/or supine position. Values of SaO_2, tCO_2, $\dot{V}_E$, V_T and f were averaged for every 30 sec epoch of the study period. Per cent sleep time was determined for each 10 min epoch, using standard EEG/EOG criteria. Statistical assessment was by analysis of variance.

TABLE Results (n = 10, mean ± SD)

	Pre-op	*T0*	*T1*	*T2*
Time (min)			22 ± 12	43 ± 20
Mean SaO_2 (%)	97 ± 1	95 ± 2†	94 ± 3†*	90 ± 3†*
tCO_2 (mmHg)		50 ± 7	51 ± 2	49 ± 6
$\dot{V}_E$ ($L \cdot min^{-1}$)		10.4 ± 3.2†	6.8 ± 2.1	7.0 ± 3.4
V_T (ml)		590 ± 220	450 ± 190	430 ± 210
f (breaths · min^{-1})		19 ± 7	16 ± 6	17 ± 6
Time asleep (%)		42 ± 38‡	64 ± 33	73 ± 20

	Pre-op	*T3*	*T4*	*T5*
Time (min)		68 ± 18	91 ± 5	111 ± 14
Mean SaO_2 (%)	97 ± 1	94 ± 2†	94 ± 3†	95 ± 3†
tCO_2 (mmHg)		50 ± 4	50 ± 5	50 ± 4
$\dot{V}_E$ ($L \cdot min^{-1}$)		7.7 ± 2.3	8.1 ± 3.8	7.7 ± 3.1
V_T (ml)		500 ± 190	490 ± 250	490 ± 170
f (breaths · min^{-1})		17 ± 8	16 ± 6	16 ± 4
Time asleep (%)		82 ± 29	88 ± 11	85 ± 17

†Different from pre-op ($p < 0.05$).
*Different from T0, T1, T3, T4, T5 ($p < 0.05$).
‡Different from T1–T5 ($p < 0.05$).

Results

Upon arousal, SaO_2 was slightly less than preop. With time, it decreased further and progressively to a nadir and then slowly recovered. Individual times to the nadir varied from 17 to 70 min. For analysis, we selected the data of epochs at the outset and end of the study period (T0, T5) at the nadir (T2), and at points halfway between T0 and T2 (T1), and 1/3 and 2/3 between T2 and T5 (T3, T4). SaO_2 at T2 was significantly less than at other recovery times. $\dot{V}_E$ was greater and sleep time less at T0. No other significant differences were observed.

Discussion

To our knowledge, this is the first study in which SaO_2 has been monitored continuously after arousal during anaesthetic recovery. The results reveal two phases of SaO_2 change – first, a gradual reduction over periods of up to about 1 hr, and then a progressive recovery. The minimum SaO_2 values were less than expected in healthy young patients during recovery from anaesthesia. The cause of this pattern of SaO_2 change is not immediately apparent. It appears unrelated to ventilation, ventilatory pattern, PCO_2 or sleep. There were no obvious clinical correlates. We conclude that, after arousal from general anaesthesia, the period of maximum impairment of oxygenation may be delayed for up to an hour.

The role of amrinone in weaning patients from hypothermic cardiopulmonary bypass

J.M. DeJesus, J.E. Wynands, J.R. Ramsay, J.P. O'Connor, F.E. Ralley, G.R. Robbins, J. Bilodeau
Departments of Anaesthesia, McGill University and Royal Victoria Hospital, Montreal, Quebec

The need exists for a cardiotonic drug which will enhance cardiac performance and facilitate weaning from CPB without detrimental effect to the heart. Amrinone, a relatively new, non-glycoside, non-catecholamine agent with both positive inotropic and vasodilator effects may be such a drug. This study was undertaken to determine the efficacy of amrinone vs placebo in facilitating weaning from cardiopulmonary bypass.

Methods

With Ethics Committee approval and informed consent a randomized double-blind study was conducted. Twenty-eight ASA physical status III & IV patients scheduled for coronary artery bypass surgery with LVEF > 40 per cent were studied. All patients were premedicated with morphine, diazepam and scopolamine and their usual cardiac medication. Monitoring was established and patients were anaesthetized with sufentanil, pancuronium, O_2 and enflurane. Haemodynamics. $S\bar{v}O_2$. SaO_2 and haematocrits were determined prior to induction, 5 min post-induction and at pericardiotomy for measurement of oxygen consumption ($\dot{V}O_2$). After rewarming to a nasopharyngeal temperature of 37° C and bladder temperature of 34° C and prior to weaning, amrinone 0.75 $mg \cdot kg^{-1}$ (Group A) or an equal volume of placebo (Group C) was infused over two minutes. CPB flow and MAP were recorded before drug administration and every 2 min for a total of 10 min, at which time the patient was weaned from CPB. Haemodynamic profiles were recorded at 1, 10, 20 and 30 min post-bypass and thereafter, half hourly for five measurements. Following CPB, phenylephrine, noradrenalin plus phentolamine (16 $\mu g \cdot ml^{-1}$ and 20 $\mu g \cdot ml^{-1}$), and where necessary an IABC were used as required for cardiovascular support. Anova and discriminatory tests were used for statistical analysis.

Results

Thirteen patients received amrinone and 15 placebo. There were no significant differences in haemodynamic values between groups at any time. There was a trend for amrinone patients to receive less noradrenaline with phentolamine and ephedrine, but more phenylephrine was required (Table I). The mixed venous oxygen saturations were significantly higher during the 1, 10, 20 and 30 min periods in those patients who received amrinone leading to lower whole body $\dot{V}O_2$ at these times (Table II).

Discussion

While Group A patients did not have significantly different cardiac performance from those in Group C, when the inotrope requirement findings are subjected to power analysis it appears that a sample of 100 patients would show significantly reduced requirements with amrinone. The unexpected and intriguing finding is the higher $S\bar{v}O_2$ giving a lower $\dot{V}O_2$ in Group A. In these patients an equivalent amount of cardiac work was being performed at a lower $\dot{V}O_2$. Thus, while measured haemodynamics appeared unaffected by amrinone, energy utilization may have been more efficient. This suggests measurement of myocardial $\dot{V}O_2$ may shed further light on the role of amrinone in this setting. These preliminary findings suggest possible benefits from amrinone in weaning patients from CPB.

TABLE I Drug requirements (means ± SEM)

	Phenylephrine (μg)	*Ephedrine (mg)*	*Noradrenalin (μg)*
Group A			
0–30 (min)	115 ± 64	0.38 ± 0.38	0
0–180	192 ± 86	1.5 ± 1.1	0.003 ± 0.002
Group C			
0–30 (min)	35 ± 19	0.71 ± 0.45	0.169 ± 0.141
0–180	35 ± 19	4.2 ± 1.7	0.132 ± 0.112

P = NS.

TABLE 2 $S\bar{v}O_2$ and $\dot{V}O_2$ 1–30 min post-CPB (mean ± SEM)

	$S\bar{v}O_2$	*$\dot{V}O_2$*
Group A		
1 (min)	76 ± 2*	104 ± 6*
10	75 ± 2*	120 ± 7*
20	70 ± 2*	116 ± 7*
30	75 ± 2*	135 ± 7
Group C		
1 (min)	67 ± 3	135 ± 11
10	65 ± 3	157 ± 12
20	63 ± 2	154 ± 10
30	61 ± 2	177 ± 13

*$p < 0.05$ (Group A vs Group C).

Reference

1 *Mancini D, LeJemtel T, Sonnenblick EH.* Intravenous use of amrinone for the treatment of the failing heart. Am J Cardiol, 1985; 56: 8B–15B.

Comparison of fentanyl and sufentanil anaesthesia for abdominal aortic surgery

E.T. Crosby, D.R. Miller, P.P. Hamilton
Department of Anaesthesia, Ottawa General Hospital, University of Ottawa, Ottawa, Ontario

Fentanyl is established as an anaesthetic agent in patients with limited cardiac reserve.[1] Fentanyl has been shown to be inadequate in preventing hyperdynamic responses to perioperative stimuli in aortic surgery.[2] The purpose of this study was to test the hypothesis that equipotent doses of sufentanil, compared to fentanyl, would result in a haemodynamically more stable

operative course in patients undergoing abdominal aortic surgery.

Methods

Twenty-two patients undergoing elective abdominal aortic-aneurysm resection or aortobifemoral bypass for occlusive disease were entered into this prospective randomized, double-blind trial. All patients gave written informed consent to the protocol approved by the Hospital Ethics Committee. Excluded were patients with a history of myocardial infarction (MI) within six months of surgery or severe systemic disease. Patients were premedicated with morphine 0.15 $mg \cdot kg^{-1}$ IM and lorazepam 0.04 $mg \cdot kg^{-1}$ 90 minutes preoperatively. All patients were beta blocked with either their usual dose of beta blocker given up to the morning of surgery, or with metoprolol 100 mg PO in two divided doses prior to surgery. Anaesthesia was induced and maintained with either fentanyl (FEN) 125 $\mu g \cdot kg^{-1}$ or sufentanil (SUF) 25 $\mu g \cdot kg^{-1}$ administered in four divided doses from coded syringes. Pancuronium 0.04 $mg \cdot kg^{-1}$ and metocurine 0.16 $mg \cdot kg^{-1}$ provided muscle relaxation and patients were ventilated to an end–tidal PCO_2 of 35–40 with 100 per cent oxygen throughout the procedure. Intravenous nitroglycerin (NTG) was given if needed to maintain systolic pressure within 20 per cent of control. Isoflurane was added if greater than 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of NTG was required. Haemodynamic indices were obtained to measure the responses to induction (IND), intubation (INT), skin incision (SI), aortic cross clamping (XC) and declamping (DEC). Continuous Holter monitoring of lead V_5 was used to detect myocardial ischaemia. Results were analyzed using repeated measures analysis of variance (ANOVA) and paired Student's t tests.

Results

There were no significant differences ($p < 0.05$) between the two groups with respect to age, ASA physical status Class, duration of surgery or blood loss. No patient had any recollection of surgery. One patient (1/10) in the FEN group suffered a postoperative MI (new Q wave), and one patient (1/12) in the SUF group developed ischaemic ST segments (>0.1 mm ST depression) but did not develop an MI. There were no deaths in either group. There were no differences ($p < 0.05$ by repeated measures ANOVA) between groups during IND, INT, SI, XC or DEC with respect to any of the haemodynamic variables. The important haemodynamic changes (deltas) within each group during the critical anaesthetic and surgical interventions are presented in the Table. The FEN group required 1.02 ± 1.11 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of NTG and 0.03 ± 0.06 MAC units · $hour^{-1}$ of isoflurane compared to 1.72 ± 2.8 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of NTG and 0.02 ± 0.03 MAC units · $hour^{-1}$ for the SUF group (n.s., $p < 0.05$). One patient in each group required propranolol 1–2 mg to maintain HR < 90.

TABLE Haemodynamic changes with anaesthetic and surgical interventions

Intervention	*Drug*	*HR*	*MAP*	*PCWP*	*CI*
Induction	FEN	6.9 ± 7.5*	5.3 ± 14.9	1.9 ± 3.7	0.2 ± 0.2
	SUF	6.4 ± 7.5*	−14.0 ± 24.5	1.1 ± 2.9	0.1 ± 0.4
Intubation	FEN	2.4 ± 4.1	4.5 ± 7.7	1.7 ± 2.7	0.1 ± 0.1
	SUF	2.6 ± 4.1*	3.0 ± 12.7	1.2 ± 2.3*	−0.9 ± 0.2
Incision	FEN	−5.0 ± 2.6	−3.3 ± 10.8	−3.0 ± 3.1	−0.3 ± 0.1
	SUF	1.5 ± 3.6	−2.0 ± 12.5	−1.6 ± 2.2	−0.4 ± 0.2
Crossclamp	FEN	−2.5 ± 5.7	0.3 ± 6.3	−2.6 ± 3.5	−0.0 ± 0.1
	SUF	−1.8 ± 4.7	5.2 ± 21.0	−0.5 ± 4.3	−0.4 ± 0.3
Declamp immed	FEN	0.8 ± 4.6	−2.9 ± 16.6	1.4 ± 4.4	0.0 ± 0.3
	SUF	−0.5 ± 6.5	−5.5 ± 14.1	−0.9 ± 2.9	0.2 ± 0.3
Declamp stable	FEN	3.1 ± 4.8	5.2 ± 25.1	0.7 ± 3.9	0.4 ± 0.3
	SUF	2.1 ± 5.1	5.7 ± 20.8	1.8 ± 4.3	0.2 ± 0.3

Intervention	*Drug*	*SVI*	*LVSWI*	*SVR*
Induction	FEN	−1.8 ± 2.7	0.3 ± 0.3	−1.2 ± 141.2
	SUF	−1.1 ± 3.0	0.6 ± 1.2	−291.5 ± 119.8*
Intubation	FEN	−1.7 ± 2.7	0.6 ± 0.6	79.3 ± 160.7
	SUF	−2.4 ± 2.8	0.1 ± 1.2	22.8 ± 96.4
Incision	FEN	−0.8 ± 2.0	0.2 ± 0.5	35.3 ± 127.0
	SUF	−3.0 ± 3.5	−0.4 ± 0.5	−26.0 ± 233.2
Crossclamp	FEN	−5.5 ± 2.2	−1.1 ± 0.4	193.6 ± 80.7
	SUF	−3.2 ± 3.0	−0.3 ± 0.4	290.6 ± 124.1*
Declamp immed	FEN	−0.5 ± 3.3	0.1 ± 0.8	76.1 ± 211.9
	SUF	2.3 ± 3.8	1.7 ± 0.7	−186.0 ± 197.1
Declamp stable	FEN	4.2 ± 3.6	0.8 ± 1.4	−212.4 ± 126.4
	SUF	2.3 ± 3.6	0.0 ± 0.8	−46.8 ± 141.0

*$p < 0.05$.

Discussion

The intense surgical stimulation and impact of aortic clamping and unclamping during abdominal aortic surgery induces significant haemodynamic alterations which may vary considerably from one patient to another. We have demonstrated that FEN and SUF provide a remarkable degree of haemodynamic stability with minimal use of NTG or anaesthetic supplement for patients undergoing aortic reconstructive surgery. Based on a potency ratio of 1:5 (FEN:SUF) we were unable to demonstrate significant differences between these two drugs. We conclude that a safe, effective anaesthetic may be provided with either FEN or SUF for abdominal aortic surgery.

References

1 *Bovill JG, Sebel PS, Stanley TH.* Opioid analgesics in anesthesia: with special reference to their use in cardiovascular anesthesia. Anesthesiology 1984; 61: 731–55.

2 *Friesen RM, Thomson IL, Hudson RJ, Rosenbloom M, Putriss CL, Cannon JE.* Fentanyl oxygen anaesthesia for abdominal aortic surgery. Can Anesth Soc J 1986; 33: 719–27.

Can anaesthesiologists detect preoperative anxiety?

N.H. Badner, W.R. Nielson, S. Munk, A.W. Gelb
Departments of Anaesthesia and Psychology, University Hospital, University of Western Ontario, London, Ontario

Most patients admitted to hospital for elective surgery experience anxiety preoperatively.[1] As part of the pre-anaesthetic visit, many anaesthestists prescribe a premedication to relieve the anxiety.[2] A recent study demonstated that anxiety the afternoon before surgery predicted anxiety preoperatively,[3] thus making the effectiveness of this prescribing practice critically dependent on the anaesthestist's ability to detect anxiety. The

aim of our study was both to assess the ability of anaesthestists to detect and quantify preoperative anxiety, and to substantiate the correlation between anxiety on the afternoon prior to surgery and that found immediately preoperatively.

Methods

The research protocol was approved by our Institutional Review Board. Subjects included were hospitalized inpatients booked for elective surgery. Exclusion criteria were age <18 years, inability to read or speak English, psychosis, and central neurological impairment. The anaesthestist interviewed the patient the day prior to the schedued surgery. Immediately after completing the patient visit, the anaesthestist was asked to assess both the patient's present level of anxiety and predict the level of anxiety at the time of surgery using a visual analogue scale. The anaesthetist was also asked to indicate the symptoms and signs on which the assessment was based. At the same time the patient's level of anxiety was quantified using both the State-Trait Anxiety Inventory (STAI) and the Multiple Affect Adjective Check List (MAACL). These questionnaires measure both trait anxiety (a personality characteristic) and state anxiety (anxiety level at a given moment) and have been extensively validated. The MAACL also assesses hostility, depression and sensation-seeking. The patient was also instructed to complete a second STAI-state anxiety questionnaire one hour preoperatively or immediately prior to receiving any premedication if this was earlier than one hour.

Data Analysis

The anaesthestists' assessments were correlated with the results of the questionnaire using linear regression techniques. Statisticaly significant differences between the correlation coefficients were determined using Fisher's r-Z transformation and independent t-tests.

Results

Twenty-two anaesthesiologists (12 staff and 10 residents) assessed 95 patients preoperatively. Each anaesthesiologist assessed between two and six different patients. The patients included 41 females and 54 males with ages ranging from 18 to 73 years. There was a significant correlation between the STAI and MAACL anxiety scores the afternoon before surgery ($r = 0.53$, $p < 0.0005$). There was also a highly significant correlation between the STAI scores the afternoon before surgery and those preoperatively ($r = 0.74$, $p < 0.001$).

However, the correlation between the anaesthestists' assessment of the patients' level of anxiety at the afternoon vist or the projected level of anxiety and those of the STAI and MAACL scores were nonsignificant (see Table). There was no difference between staff and resident anaesthestist abilities. A statistically significant correlation was found if the anaesthestists were subdivided according to the signs and symptoms used in their assessments; those who directly asked their patients how anxious they felt had a good correlation with the STAI score.

TABLE Correlation Coefficients

	Test scores	
Anaesthetists' scores	*Afternoon STAI*	*Preoperative STAI*
All	0.28	0.23
Staff	0.33	0.23
Residents	0.23	0.24
Asked	0.66*†	0.44*
Not asked	0.006†	0.11

*Significance of fit $p < 0.05$.
†Difference between $p < 0.006$.

Discussion

This study substantiates the finding that patient anxiety the afternoon before surgery correlates with that immediately preoperatively.[3] However, this study also indicates that anaesthestists as a group are poor at assessing and predicting preoperative anxiety when compared to objective questionnaires completed by the patient. This finding is consistent with previous studies by clinical psychologists.[4] The predictive accuracy was, however, significantly improved when anaesthestists specifically asked patients whether or not they were anxious. As the efficacy of preoperative anxiolytic medication is critically dependent on the assessment of preoperative anxiety, we recommend the more forthright practice of directly asking the patient about their level of anxiety.

References

1 *Norris W, Baird WLM.* Pre-operatively anxiety – a study of the incidence and etiology. Br J Anaesth 1967; 39: 503–9.
2 *Madij TH, Paasuke RT.* Anaesthetic premedication: aims, assessment and methods. Can J Anaesth 1987; 34: 259–73.
3 *Lichtor, JL et al.* Preoperative anxiety: does anxiety level the afternoon before the surgery predict anxiety level just before surgery? Anesthesiology 1987; 67: 595–9.
4 *Wiggins JS.* Personality and Prediction. Principles of Personality Assessment. Pub. Addision-Wesley, 1973.

Failure to predict myocardial ischaemia in patients with coronary artery disease

H. Smith, H.J. Nathan
Department of Anaesthesia, University of Ottawa Heart Institute, Ottawa Civic Hospital, Ottawa, Ontario

Prevention of perioperative myocardial infarction is a challenge faced by all anaesthetists caring for patients with ischaemic heart disease. We use the preoperative assessment to identify patients at risk and attempt to control HR, BP, and PCWP to avoid adverse changes in the myocardial oxygen supply/demand ratio. Patients with severe coronary artery disease were studied to determine if these strategies are effective in preventing perioperative myocardial ischaemia.

Methods

Informed consent was obtained from 100 patients with good LV function scheduled for elective CABG. All patients received a standardized premed and fentanyl-based anaesthetic. Leads II and V_5 were recorded on a Holter monitor applied one hour preop. A pulmonary artery catheter and arterial line were inserted before induction of anaesthesia. Data was collected during six phases: pre-induction, 1 min post-induction, 1 min

post-intubation, pre-incision, 1 min post-incision, and 1 min post-sternotomy. A 30-second Holter strip was taken at each phase and read by a blinded observer. Thirty-eight patients whose ST-segments were isoelectric in both leads on arrival in the OR were included in the study. The control group consisted of 30 patients whose ST-segments remained isoelectric during all six phases. The remaining eight patients who developed ≥1 mm change in their ST-segments in one or both ECG leads became the ischaemic group. ECG's and CK-MB fractions were collected 2 and 24 hours postop. New myocardial infarctions were defined as those with both CKMB > 5 per cent of total CK and new Q waves on the postop ECG.

Results

Preoperative factors assessed (age, previous antianginal therapy, number of diseased vessels, class of angina, and previous MI) were equally represented in the control and ischaemic groups. The ST-segments were elevated in five patients and depressed in three. Most patients developed ST-segment change within 1 min of induction and all had occurred pre-incision. Five of eight showed ST changes in both leads. There were no significant differences in haemodynamic variables between the two groups. The onset of ischaemia was not associated with a clinically significant change in any variable. At ischaemia onset, the mean values ± 1 SD (n = 8) were: HR 62 ± 12, SBP 115 ± 18, PCWP 15 ± 4, CI 2.5 ± 0.8, HR × SBP 7186 ± 1735, MAP/HR 1.4 ± 0.3. No patient fulfilled the criteria (ECG and CK-MB) for new myocardial infarction.

Discussion

In a group of patients at high risk of myocardial ischaemia, we compared those with ECG evidence of ischaemia with those whose ST-segments remained isoelectric. We were unable to find pre- or intraoperative factors that could predict the occurrence of ST-segment changes. None of the patients in the ischaemic group had changes of ≥2 mm. We attempted to improve the accuracy of this technique by including only patients whose ST-segments were isoelectric pre-induction. Although a change of ≥1 mm is accepted as indicating myocardial ischaemia this may require further validation.

In all 38 patients haemodynamic determinants of myocardial oxygen supply and demand remained within an optimal range and yet eight developed significant ST-segment changes. This suggests that increased coronary vascular resistance may have caused myocardial ischaemia in this setting. Further studies will be required to explore this interesting possibility.

A comparative study of patient-controlled epidural analgesia (PCEA) and continuous infusion epidural analgesia (CIEA) during labour

P.Y.H. Yu, D.R. Gambling, C. Cole, G.H. McMorland
Department of Anaesthesia, University of British Columbia and Grace Hospital, Vancouver, British Columbia

Patient-controlled administration (PCA) of analgesics has gained wide application in pain relief with the advent of modern technology allowing a safe delivery system. PCA using intravenous meperidine has been studied during labour.[1-3] Patient-controlled epidural infusions of local anaesthetic for analgesia during labour has not been previously described. This study was designed to compare in a prospecive, randomized, single blinded and placebo-controlled manner the efficacy and safety of PCEA with the established CIEA technique.

Methods

Following approval from the Screening Committee for Research Involving Human Subjects and informed consent, 27 ASA physical status I or II nulliparous parturients in established term labour who requested epidural analgesia were selected. Epidural catheterisation, using a standard technique was performed. Once the patients were comfortable, they were randomized into two groups. Patients in Group A received a continuous epidural infusion of 4 ml·hr^{-1} of 0.125 per cent bupivacaine, with the ability to receive an additional bolus of 4 ml every 20 minutes. Patients in Group B received 12 ml·hr^{-1} of 0.125 per cent bupivacaine through the same PCA system but with the demand button deactivated. Patients in both groups received an initial dose of 8 ml 0.25 per cent bupivacaine and were unaware of their group assignment. All patients received the same instructions and expected to get pain relief after making a demand. Analgesia (using a linear visual analogue scale), height of sensory block, motor block, maternal BP and fetal heart rate were recorded hourly. The total bupivacaine dose, duration and outcome of labour and Apgar scores were recorded. Results were analyzed using Chi square analysis for ordinal data and a two sample t test for interval data.

Our PCA system was a modified IVAC 530 infusion pump designed by the U.B.C. Biomedical Engineering Department with the ability to determine the infusion rate, demand bolus and lock-out time.

Results

The two groups were well matched for age, height, weight, cervical dilatation and duration of labour. The babies' birthweight, Apgar scores and mode of delivery were similar in the two groups. Patients in the PCEA group made more demands per hour as they were required to titrate their analgesia, but overall

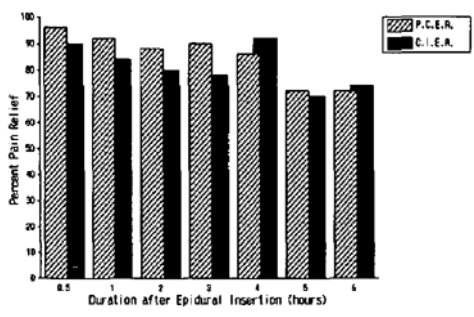


FIGURE 1 Patient analgesia obtained: PCEA vs CIEA (mean).

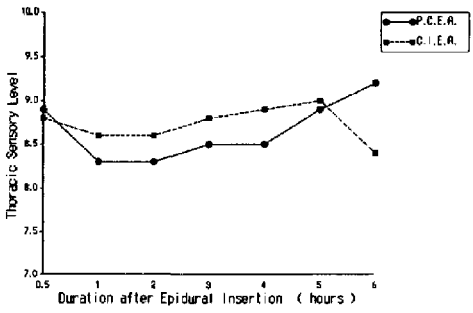


FIGURE 2 Height of sensory blockade (mean)

required less bupivacaine (11.2 mg ± 0.85 vs 15.2 ± 0.5 $mg \cdot hr^{-1}$, $p < 0.01$) during labour than those in the CIEA group. Pain scores prior to epidural insertion were similar in both groups. The mean pain scores obtained were comparable in both groups and the degree of analgesia is depicted in Figure 1. The mean height of sensory blockade obtained by both groups is depicted in Figure 2.

Discussion

PCEA provides analgesia during labour which is comparable to that achieved with CIEA. This is accomplished with a lower overall local anaesthetic requirement. Patients appreciated the ability to control their own level of pain relief and expressed satisfaction using the PCA system.

References

1 *Scott JS.* Obstetric analgesia: a consideration of labour pain and a patient-controlled technique for its relief with meperidine. Am J Obstet Gynecol 1970; 106: 959–78.

2 *Evans JM, MacCarthy J, Rosen M, Hogg MIJ.* Apparatus for patient-controlled administration of intravenous narcotics during labour. Lancet 1976; X: 17–8.

3 *Robinson O, Rosen M, Evans JM, Revill SI, David H, Rees GAD.* Self-Administered intravenous and intramuscular pethidine: controlled trial in labour. Anaesthesia 1980; 35: 763–70.

Haemodynamic and serial CPK-MB responses in CABG patients with positive technetium pyrophospate-SPECT

D.C.H. Cheng, F. Chung, R.J. Burns, C.M. Feindel
Departments of Anaesthesia, Medicine and Surgery, Toronto Western Hospital, University of Toronto, Toronto, Ontario

Reduction of coronary perfusion in response to tracheal intubation in the absence of haemodynamic change was documented by thallium scan in 45 per cent of 22 patients undergoing CABG.[1] Recently, we demonstrated that the presence of prebypass myocardial ischaemia is associated with a 3.8-fold increase in risk of myocardial infarction (MI), as documented by technetium pyrophosphate uptake using single-photon emission computed tomography (TcPPi-SPECT) in patients undergoing elective CABG.[2] In the present study, the significance of haemodynamic and serial cardiospecific creatinine phosphokinase (CPK-MB) responses to perioperative MI was correlated in elective CABG patients with positive TcPPi-SPECT.

Methods

Institutional approval was obtained. Radionuclide angiocardiography and TcPPi-SPECT were performed 24 hr pre-op and 48 hr post-op. A standard high dose fentanyl anaesthetic protocol was used. Complete haemodynamic profiles were recorded (a) during the pre-bypass period at: control (CONT), one minute after induction (IND), intubation (INT), skin incision (INC), sternotomy (STERN) and aortic cannulation (CANNU); (b) during the post-bypass period at one minute after off pump (PUMP), protamine administration (PROTA), pericardial closure (PERIC), sternal wiring (WIRE) and skin closure (CLOSE). Post-op CPK-MB was measured q4h. Analysis between patients with positive and negative TcPPi-SPECT was done by repeated measured ANOVA and t tests with correction of equal variance.

Results

Thirty patients were studied: two patients were excluded due to unstable haemodynamics at 48 hrs post-op for follow-up scan.

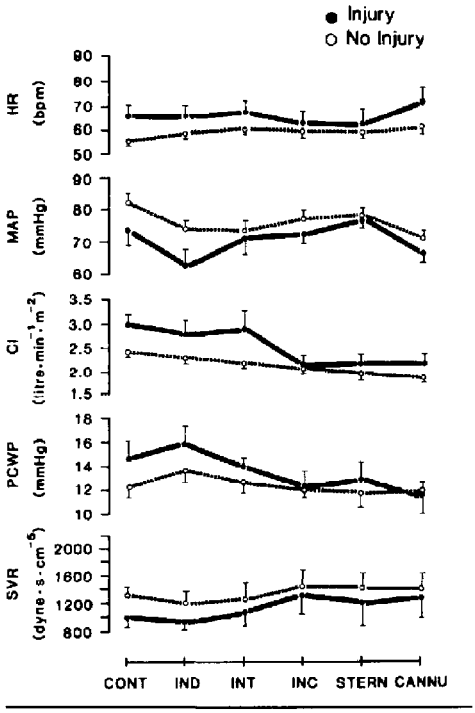


FIGURE 1 Haemodynamics Pre-bypass.

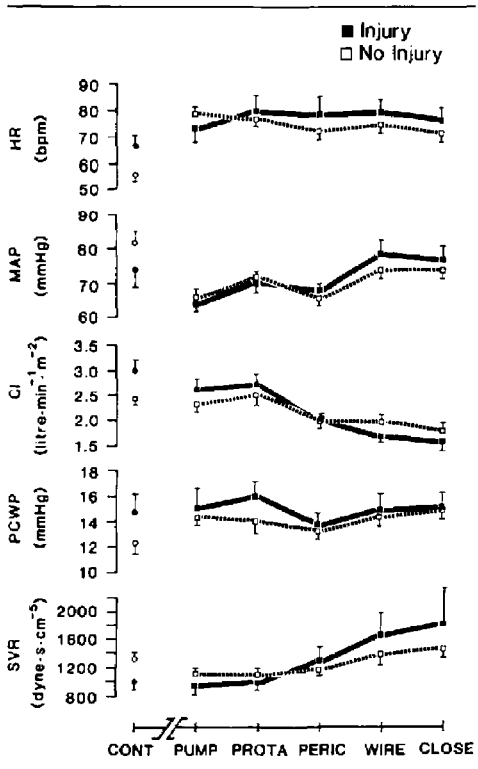


FIGURE 2 Haemodynamics Post-bypass.

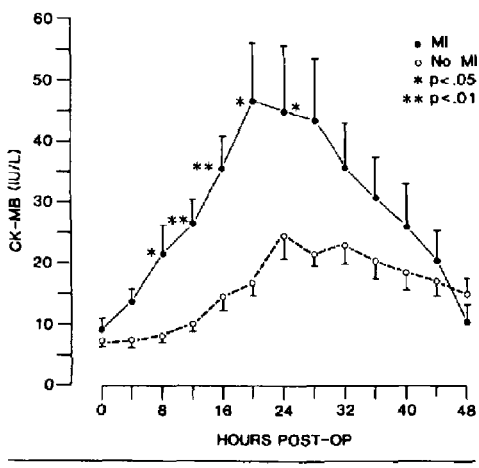


FIGURE 3 CPK-MB Results.

Seven of 28 patients (25 per cent) developed irreversible myocardial necrotic mass of 38.0 gm ± 5.5 (SE) as documented by post-op TcPPi-SPECT. No statistically significant haemodynamic changes were observed between the two groups during pre-bypass (Figure 1) and post-bypass (Figure 2) periods. The intra-op requirement of propranolol, nitroglycerin, nitroprusside and phenylephrine was not different. However, CPK-MB was significantly elevated from 8–24 hrs in the MI group (Figure 3).

Discussion

Scintigraphically, 25 per cent of elective CABG patients developed period MI, which can be optimally diagnosed by CPK-MB at 8–24 hrs post-op. This outcome is not preventable by careful control of haemodynamic indices of myocardial oxygen supply and demand, as shown during the pre-bypass and post-bypass periods.

References

1 *Kleinman B, Henkin RE, Glisson SN et al.* Qualitative evaluation of coronary flow during anesthetic induction using thalium-201 perfusion scans. Anesthesiology 1986; 64: 157–64.

2 *Cheng CDH, Chung F, Burns RJ, Houston P, David T.* Perioperative myocardial ischemia: predictor of myocardial infarction by technetium pyrophosphate-SPECT. Anesthesiology 1987; 67: A594.

Perioperative electrocardiographic changes in a surgical population

C.L. Irish, P.H. Manninen, A.W. Gelb

Department of Anaesthesia, University Hospital, University of Western Ontario, London, Ontario

The electrocardiogram (ECG) is a routine monitor during anaesthesia. Perioperative ECG abnormalities are often detected on surgical patients, yet the frequency, interpretation, and significance of these abnormalities remains unknown. In a previous study of neurosurgical patients 30 per cent developed ECG changes intraoperatively and 53 per cent developed an abnormal postoperative ECG.[1] The purpose of this study was to document the comparative frequency of intraoperative and postoperative ECG abnormalities in a general surgical population, and to determine their significance.

Methods

After Institutional Ethics Committee approval, a standard 12 lead ECG was obtained preoperatively as well as postoperatively within one hour of admission to the recovery room on 72 surgical patients. Intraoperatively, in addition to lead II, a continuous tape (Holter) monitor of modified chest leads V_1 and V_5 was recorded for the duration of anaesthesia on 60 patients. Patients undergoing emergency, neurosurgical, or cardiovascular procedures were excluded from the study. The charts of all patients were examined for ischaemic events in the postoperative period. Chi square analysis or unpaired t test were performed on the data where appropriate.

TABLE New postoperative ST–T wave changes (n = total within each group)

Overall incidence (n = 72)	19%
Anaesthetic technique	
Regional (n = 14)	14%
General (n = 58)	21%
ASA physical status classification	
I (n = 34)	12%
II (n = 22)	18%
III (n = 16)	37%
Ischaemic heart disease (n = 11)	27%

Results

The mean age (± SD) was 54 ± 19 years with 34 males and 38 females. Intraoperative Holter monitoring revealed abnormalities in 20 per cent (12/60) of patients. These consisted of nodal rhythm in four, complete heart block in one, multiple PVC's in five, and ST segment changes in two patients. The overall incidence of new postoperative ECG changes was 22 per cent (16/72). One patient had a new 1° AV block, one patient had a new right bundle branch block pattern, while 12 patients had new non-specific ST or T wave abnormalities. The remaining two patients had marked ST segment elevation. The intraoperative changes persisted in only two patients into the recovery room. With respect to age, sex, preoperative ASA classification, incidence of ischaemic heart disease, or anaesthetic technique, no statistically significant difference between groups was found (Table). No patient with perioperative repolarization abnormalities had any suspected myocardial ischaemic event postopertively. Only one documented myocardial ischaemic event occurred which proved to be fatal on the tenth postoperative day in a patient with no perioperative ECG changes.

Discussion

The incidence of new postoperative ECG changes of 22 per cent found in this study compares with the literature.[2] A similar, although unrelated, incidence of intraoperative ECG changes of 20 per cent was found. The perioperative ECG changes are less than those seen in patients undergoing neurosurgical procedures. No subset of patients are immune from these ECG changes. While subclinical myocardial ischaemia may be responsible for some of these changes, the aetiology of many perioperative changes must be considered to be non-ischaemic.

References

1 *Manninen PH, Lam AM, Gelb AW.* Electrocardiographic changes during and after isoflurane-induced hypotension for neurovascular surgery. Can J Anaesth 1987; 34: 549–54.

2 *Breslow MJ, Miller CF, Parker SD, Walman AT, Rogers MC.* Changes in T-wave morphology following anesthesia and surgery. A common recovery room phenomenon. Anesthesiology 1986; 64: 398–402.

The choice of balanced salt solution administered during major vascular surgery: influence on acid–base status

H. Wong, C.H. Kehler

Department of Anesthesia, University of Manitoba, Winnipeg, Manitoba

Because relatively large amounts of balanced salt solution are routinely administered during and after abdominal aortic surgery, the composition of the solution may have a significant effect on metabolic and acid–base balance. The concentration of chloride ion in normal saline (NS) is 154 $mEq \cdot L^{-1}$. Lactated Ringer's solution (RL) has two anions; chloride (109 $mEq \cdot L^{-1}$) and lactate (28 $mEq \cdot L^{-1}$). Administration of large volumes of NS may produce a hyperchloremic metabolic acidosis by acutely reducing the plasma bicarbonate concentration. In contrast, administration of solutions containing organic anions (lactate, citrate) may produce a metabolic alkalosis via hepatic conversion of these ions to bicarbonate.[1,2] The purpose of this study was to compare the effect of NS with RL administration on acid–base balance in patients having abdominal aortic surgery.

Methods

This study received institutional approval and informed consent was obtained from each patient. Randomization was done to assign patients having elective abdominal aortic aneurysm repair or aorto-bifemoral bypass grafting to one of two groups based on the type of intravenous fluid that they were to receive intraoperatively (NS or RL). All other aspects of patient care were unchanged from the routine that is followed for major vascular surgery at our institution and data were collected to compare the relevant uncontrolled variables in the two groups. Arterial blood gas analysis and electrolyte measurements were made at the following time periods: (1) preoperative, (2) preinduction, (3) before aortic cross clamp (AXC), (4) 30 minutes after AXC applied, (5) five minutes after AXC released, (6) 30 minutes after AXC released, (7) following skin closure, (8) 30 min post-ICU admission, (9) at rectal temp = 37 degrees, (10) post-extubation (if applicable), (11) 24 hours postoperative, (12) 48 hours postoperative. Demographic parameters were assessed using Chi square analysis and ANOVA. For purposes of statistical analysis, pH values were converted to hydrogen ion concentration. Parameters of acid–base status and serum electrolytes were compared using Student's t test for unpaired data.

Results

Seventeen patients were randomized to each of the two study groups. Comparison of demographic data revealed no difference between the groups with regard to age, sex, weight, surgeon, surgical procedure or incidence of coexisting medical diseases (cardiopulmonary, renal or diabetes). Comparison of preoperative serum glucose, electrolytes, creatinine, albumin, haemoglobin and volume intake 24 hours prior to surgery also showed no differences between the two groups. Blood loss, volume of blood and crystalloid replacement, urine output, aortic clamp time, lowest nasopharyngeal temperature, PaO_2, $PaCO_2$ and anion gap did not differ between the two groups during the intraoperative period. Significant intra- and postoperative differences were

TABLE Results (Mean ± SEM)

	Time period 1	2	3	4	5	6
Hydrogen ion (nM)						
NS	38.7 ± 1.1	39.2 ± 1.1	39.5 ± 1.1	44.0 ± 1.1 *	50.0 ± 1.1 *	48.0 ± 1.1 *
RL	38.2 ± 1.0	38.1 ± 1.1	37.8 ± 1.1	40.4 ± 1.1	45.6 ± 1.1	42.8 ± 1.1
Bicarbonate (mEq·L^{-1})						
NS	24.3 ± 0.7	24.7 ± 0.6	22.8 ± 0.6	19.2 ± 0.6 *	18.9 ± 0.6 *	19.4 ± 0.6 *
RL	23.6 ± 0.6	25.4 ± 0.6	23.7 ± 0.6	21.2 ± 0.6	22.2 ± 0.6	22.1 ± 0.6
Base excess (mEq·L^{-1})						
NS	1.1 ± 0.5	0.9 ± 0.5	−0.8 ± 0.5 *	−5.1 ± 0.5 *	−6.3 ± 0.5 *	−5.9 ± 0.5 *
RL	0.1 ± 0.5	1.6 ± 0.5	0.5 ± 0.5	−2.2 ±0.5	−2.8 ± 0.5	−2.0 ± 0.5
Chloride (mM)						
NS	101 ± 2.0	104 ± 1.8	109 ± 3.7	112 ± 2.0 *	112 ± 1.9 *	112 ± 2.1 *
RL	100 ± 1.7	103 ± 2.1	105 ± 2.4	105 ± 1.8	105 ± 1.9	105 ± 1.9

	Time period 7	8	9	10	11	12
Hydrogen ion (nM)						
NS	49.7 ± 1.2 *	47.1 ± 1.1 *	45.4 ± 1.1	46.5 ± 1.4 *	42.2 ± 1.1	38.9 ± 1.1
RL	42.5 ± 1.1	41.4 ± 1.1	44.5 ± 1.1	41.7 ± 1.3	41.2 ± 1.2	38.6 ± 1.2
Bicarbonate (mEq·L^{-1})						
NS	18.8 ± 0.7 *	20.6 ± 0.6 *	21.3 ± 0.6 *	22.4 ± 0.7 *	22.8 ± 0.6 *	21.7 ± 0.6 *
RL	21.9 ± 0.6	24.1 ± 0.6	25.2 ± 0.6	25.7 ± 0.7	25.7 ± 0.7	24.5 ± 0.7
Base excess (mEq·L^{-1})						
NS	−6.6 ± 0.6 *	−5.3 ± 0.5 *	−3.8 ± 0.5 *	−3.1 ± 0.6 *	−1.7 ± 0.5 *	−0.7 ± 0.5
RL	−2.0 ± 0.5	−0.9 ± 0.5	0.0 ± 0.5	0.7 ± 0.6	0.8 ± 0.6	0.3 ± 0.6
Chloride (mM)						
NS	112 ± 4.3	111 ± 1.7 *	108 ± 2.1	110 ± 1.7	108 ± 2.0	94 ± 2.2
RL	105 ± 3.0	103 ± 1.7	104 ± 2.4	102 ± 3.0	103 ± 2.8	99 ± 2.1

*Significant difference between the group at the indicated period ($p < 0.05$).
Time periods 1–12 are described in "Methods."

observed in hydrogen ion concentration, bicarbonate concentration, base deficit and chloride concentration as indicated in the Table.

Discussion

As the results indicate, the intraoperative use of normal saline is associated with a hyperchloremic metabolic acidosis which persists into the immediate postoperative period and resolves within the first postoperative day. The use of lactated Ringer's solution, in contrast, maintains intraoperative H^+ concentration and base excess closer to the normal range with a significant difference in bicarbonate concentration between the two groups lasting as long as 48 hours postoperatively. Many factors may disturb acid–base balance during major surgery. Although the intraoperative choice of balanced salt solution is unlikely to upset hydrogen ion homeostasis by itself, the results of this study suggest that lactated Ringer's solution is more likely to maintain normal acid–base balance than normal saline when used during surgery that is associated with the production of a metabolic acidosis.[3]

References

1 *Hartmann AF, Senn MJE.* Studies in the metabolism of sodium r-lactate. I. Response of normal human subjects to the intravenous injection of sodium r-lactate. J. Clin Invest 1932; 11: 327–35.
2 *Litwin MS, Smith LL, Moore FD.* Metabolic alkalosis following massive transfusion. Surgery 1958; 45: 805–13.
3 *Stokke DB et al.* Declamping acidemia in major vascular surgery. Dan Med Bull 1980; 27: 274–7.

Effect of thoracic sympathetic blockade on global and regional haemodynamic changes of acute ischaemia

W.S. Beattie, E.L. Fallen
McMaster University Medical Centre, Hamilton, Ontario

Recently, thoracic epidural anaesthesia has been shown to reduce myocardial infarct size.[1] Thoracic sympathectomy reduces the physiologic correlates of myocardial oxygen consumption after anterior descending coronary artery (LAD) ligation. Other laboratories have shown that the haemodynamic response to ischaemia is governed by the site of ischaemia. That is, LAD occlusion is compensated for with an increase in sympathetic activity which causes maintenance of stroke volume (SV), and peripheral resistance (TPR), and increased cardiac output (Q). This response is not seen in posterior ischaemia due to a vagal afferent mediated withdrawal of sympathetic activity.[2] Haemodynamically, this is manifested as profound decrease in SV, Q, TPR with little change in heart rate (HR). Presumably, the non-ischaemic portions of the ventricle respond by increasing their stroke work. To further assess thoracic sympathectomy as a treatment modality this study was designed to assess the effects of sympathectomy (SPX) on global haemodynamic regional myocardial responses to circumflex coronary artery (CFX) and LAD occlusions.

Methods

Four groups of six anaesthetized, open-chest dogs were utilized. Blood gases were maintained in the physiologic range. BP was measured through the femoral artery, cardiac output measured with a Swan Ganz catheter, pressure was measured on a Miller PC 470 cathether. Regional cardiac dimensions of the non-ischaemic area were measured utilizing an ultrasonic Franklin Dimension gauge (Model 100). Surgical sympathectomy (T_1–T_6) was carried out in 12 dogs. After surgery, dogs were allowed to stabilize; baseline measurements were taken of Q, HR, BP, ventricular pressure and segment length data. Pressure-length loops were displayed on line on an oscilloscope. The work of regions of the heart can be approximated using the combined simultaneous output of the ventricular pressure transducer and the ultrasonic dimension gauges to form the pressure-length loop. The area of the pressure-length loop is an approximation of the stroke work (SSW) of the non-ischaemic segments of the heart. In addition, the length of this segment at end diastole (EDSL) serves a sensitive measure of pre-load. Twelve dogs had CFX ligation (six after sympathectomy) and 12 dogs had LAD ligation (six after sympathectomy). After

TABLE Results (Mean ± SD)

	CFX	*CFX sympathectomy*	*LAD*	*LAD sympathectomy*
Cardiac output ($L \cdot min^{-1}$)	3.10 ± 0.24	3.04 ± 0.24	3.53 ± 0.29	2.81 ± 0.46
Cardiac output with ischaemia ($L \cdot min^{-1}$)	2.43 ± 0.21	2.12 ± 0.3	3.25 ± 0.26	2.11 ± 0.5
% Change	−23.1	−25.5	−8.6*	−25.5
Change in MAP with ischaemia (mmHg)	−10.1 ± 2.4	− 19.4 ± 3.8	−0.4 ± 1.7*	−10.1 ±2.1
Diastolic segment length (mm)	10.97 ± 0.7	8.56 ± 0.4	10.13 ± 0.5	9.7 ± 0.41
Change after ischaemia (mm)	+0.64 ± 0.2	+0.90 ± 0.3	0.08 ± 0.01*	+1.0 ± 0.2
Segment stroke work ($mm^2 \cdot Hg$)	348.4 ± 42.1	239.1 ± 21.4	312.1 ± 32.5	256.7 ± 28.1
Change with ischaemia ($mm^2 \cdot Hg$)	28.6 ± 16.2	31.0 ± 18.0	120.0 ± 18.1*	28.1 ± 17.8

*$p < 0.01$ when compared with the other 3 groups.

occlusion, Q, HR, MAP, ventricular pressure and segment length were recorded and the dogs were then reperfused (two LAD dogs fibrillated; both were non-sympathectomized and could not be resuscitated). The remaining 22 dogs had the experiment repeated. At the cessation of the experiment, the dogs were sacrificed and the hearts excised, the aortic root was perfused at 100 mmHg with India ink while the ligature was in place. The hearts were dissected to determine ischaemic bed size. LAD and CFX were found to perfuse approximately 40 per cent of LV tissue and the ischaemic bed sizes were not significantly different.

Results

See the Table. These studies show that despite similar areas of perfusion (potential infarct size) the haemodynamic effects of ischaemia are different. CFX occlusion results in a significantly greater drop in output than LAD occlusion ($p < 0.001$). SPX does not alter the haemodynamic changes of CFX occlusion. SPX results in a three-fold greater decrease in cardiac output (8.6 to 25.5 per cent; $p < 0.001$) after LAD occlusion. Blood pressure is not as well maintained after sympathectomy in either CFX or LAD occlusion. LAD occlusion causes a 38.5 per cent increase in SSW. This occurs at the same preload and afterload, signifying an increase in contractility. This increase in SSW is significantly greater than seen in the other three groups ($p < 0.01$). Each of the other groups shows a significant increase in SSW of approximately ten per cent ($p < 0.05$). These increases occur at lower afterloads. In the CFX and both sympathectomized groups, there are significant increases in EDSL ($p < 0.01$). There is no significant increase in EDSL for the LAD ligation.

Discussion

Drops in Q after LAD occlusion are attenuated by sympathetic mediated increases in work of the non-ischaemic segments of the heart. No such mechanism was seen after CFX occlusions. After sympathectomy the heart compensates for acute ischaemic-induced loss of output by dilating (Starling mechanism). This is not as powerful a mechanism as the sympathetic mediated compensation. If the results of an open chest dog study can be applied to man, it suggests thoracic epidurals are of limited use in acute treatment of infarction. Sympathetic blockade after CFX infarction would have no effect since they are essentially auto-sympathectomized. In cases where dilation cannot be easily accomplished (large dilated heart; stiff hearts; OR patients with large infarcts) sympathetic blockade may result in a deteriorating haemodynamic picture.

References

1 *Davis RF, DeBoer LWV, Maroko P.* Thoracic epidural anesthesia reduces myocardial infarct size after coronary occlusion in dogs. Anes Analg 1986; 65: 711–7.

2 *Fallen EC, Beattie WS, Tait GA.* The effect of afterload on the cardiodepressor reflex response to coronary artery occlusion in dogs. Can J Physiol Pharmacol, 1985; 63: 1560–4.

Haemolysis during cardiopulmonary bypass

R. Martin, S. McKenty, J.P. Tétrault
Département d'anesthésie, Centre Hospitalier Universitaire de Sherbrooke, Fleurimont, Québec

One of the problems associated with cardiopulmonary bypass (CPB) is haemolysis of red blood cells. The present prospective study was undertaken to verify the influence of the type of priming and of initial acceleration on red blood cell trauma.

Methods

Inclusion criteria

Eighty adult patients undergoing coronary artery bypass grafting (CABG) under CPB were allocated at random to one of the four following groups according to the nature of the priming solution used and to the initial speed of CPB flow:

Group 1: D5LR* 1 min†
Group 2: D5LR 2 min
Group 3: LR‡ 1 min
Group 4: LR 2 min

*= Lactated Ringer's and Dextrose 5% as priming solution (2 litres).
† = 1 minute or 2 minutes: elapsed time from the start of the pump to the full calculated output for the patient according to his body surface ($2.4\ L \cdot m^{-2} \cdot min^{-1}$).
‡ = Lactated Ringer's as priming solution (2 litres).

Exclusion criteria

Patients scheduled for: reoperations (CABG), valvular surgery, emergency CABG, diabetics.

TABLE Plasma haemoglobin (Hb) and blood glucose (mg% ± SD) at the end of CPB

	Plasma Hb	*Blood glucose*
Group 1 D5LR 1 min	507.7 ± 62.2*	543.5 ± 91.9†
Group 2 D5LR 2 min	109.9 ± 63.6	518.7 ± 85.8‡
Group 3 LR 1 min	155.2 ± 46.3	133.2 ± 15.6
Group 4 LR 2 min	122.6 ± 59.4	147.9 ± 29.3

*$p < 0.001$ G1 vs G2, G3, G4 (Student's t test).
†$p < 0.0001$ G1 vs G3, G4 (Student's t test).
‡$p < 0.0001$ G2 vs G3, G4 (Student's t test).

The following data were collected and analysed
Qualitative data: sex, age, number of grafts, intensity of suction (low, medium, high), size of canula.

Quantitative data: CPB duration, mean blood pressure during CPB, mean pump flow, mean O_2 inflow, postoperative CPK-MB, number of intra- and postoperative blood transfusions, plasma osmolarity, plasma haemoglobin and plasma glucose.

The statistics used were: for qualitative data, Chi square test; for quantitative data, analyses of variance and Student's t tests for two by two comparisons (with Bonferroni correction).

Results
No statistically significant differences were found among the four groups for the following data: sex, age, number of grafts, size of cannula, suction flow, CPB duration, mean BP during CPB, mean CPB flow average output, mean O_2 output during CPB, CPK MB postoperatively, number of blood transfusions, plasma osmolarity. However, significant differences among the groups were found for plasma haemoglobin and glucose at the end of CPB (Table). When the D5LR solution was used for priming and this with a short elapsed time to full flow (1 min) (Group 1), there was three to four times more haemolysis when compared with the three other groups studied.

Conclusion
We conclude that there is an interaction between the presence of glucose in the priming solution and the initial acceleration of pump flow. The combination of D5LR with a one-minute time interval to full pump flow leads to a significant degree of haemolysis.

Detection of venous air embolism in dogs by increased end–tidal nitrogen during room air ventilation

G.B. Russell, M.T. Snider, J.L. Loomis, R.B. Richard, T.M. Rutherford
Department of Anesthesia, The Milton S. Hershey Medical Center, The Pennsylvania State University, College of Medicine and the Noll Laboratory for Human Performance Research, Hershey, Pennsylvania

Increased end–tidal nitrogen (ETN_2) is a specific and sensitive sign of venous air embolism (VAE). Because of the relatively small amount of nitrogen that gets into the blood stream during VAE, the increase detected in ETN_2 is usually quite small. The sensitivity required of ETN_2 monitors is in the range of 0.01–0.10 per cent nitrogen. ETN_2 monitoring can be used in the operating room only during ventilation with a non-nitrogen (mass fragments $^{14}N^{14}N$) containing gas. Research done on ETN_2 changes after VAE has been performed after denitrogenation and during 100 per cent oxygen breathing or during ventilation with 100 per cent O_2 and an anesthetic gas. We evaluated a non-radioactive isotope of nitrogen ($^{15}N^{15}N$), in an $80\%^{15}N^{15}N{:}20\%^{16}O_2$ mixture, as a venous embolus during room air ventilation in dogs.

Methods
Twelve mongrel dogs, 12–17 kg in weight, were studied. Each was anesthetized with pentobarbitone (30 $mg \cdot kg^{-1}$) and pancuronium (0.1 $mg \cdot kg^{-1}$), and ventilated by a volume ventilator (15 $mg \cdot kg^{-1}$ tidal volume) through an oral endotracheal tube. A percutaneous femoral arterial line and pulmonary artery catheter were inserted. Ringer's lactate (5 $mg \cdot kg^{-1} \cdot hr^{-1}$) was administered through the sideport of the central venous line. One $mg \cdot kg^{-1}$ bolus injections of "heavy" air (containing $^{15}N^{15}N$) were injected in <5 seconds through the sideport into the superior vena cava. ETN_2 changes were detected and measured by a Perkin-Elmer MGA 1100 mass spectrometer (MS). Exhaled gas was sampled by constant suction through a capillary tube attached to an airway connector. Changes in exhaled $^{15}N^{15}N$ and $^{14}N^{14}N$ were compared prior to, during, and after VAE. Expired gases were also collected in Douglas bags prior to embolization and for two ten-minute periods after VAE. These samples were also analyzed for $^{15}N^{15}N$ and $^{14}N^{14}N$.

Results
After VAE of 1.0 $mg \cdot kg^{-1}$ of the $80\%^{15}N^{15}N{:}20\%^{16}O_2$ mixture, increased $ET^{15}N^{15}N$ was detected in all dogs studied. During this same time period, there was no significant increase in $ET^{14}N^{14}N$ (Figure). The "washout curve" of $^{15}N^{15}N$ was similar in contour and duration to that of $^{14}N^{14}N$ in previous studies after room air emboli during 100 per cent O_2 ventilation. $^{14}N^{14}N$ concentrations were consistent both pre- and post-VAE in the collection samples. $^{15}N^{15}N$ concentrations were equivalent to the room air concentration pre-VAE and significantly

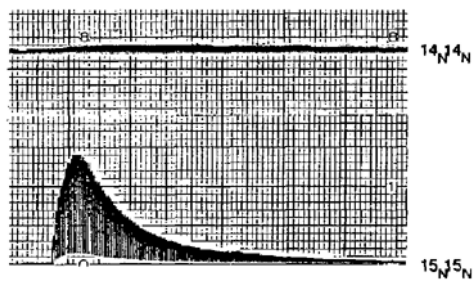


FIGURE Changes in ETN_2 after venous air embolism (VAE) (1 $ml \cdot kg^{-1}$)

TABLE Per cent nitrogen in collected exhaled gas. Mean ETN_2 after VAE 1.0 ml·kg^{-1}

	$^{14}N^{14}N$	$^{15}N^{15}N$
Control	78.427 ± 0.241	0.003 ± 0.002
Post-VAE	78.556 ± 0.186	0.039 ± 0.013

increased post-VAE, but usually for only the initial ten-minute period (Table). PAP increases and BP decreases were consistent with previously reported changes.

Discussion

The use of $^{15}N^{15}N$ as the nitrogen component of an intravenous air bolus allows study of ETN_2 changes after VAE during room air ventilation. This allows ETN_2 changes to be assessed as part of the pathophysiological changes of VAE during room air breathing as well as comparisons of changes during different treatments, i.e., 100 per cent O_2 ventilation. $^{15}N^{15}N$ washout after VAE follows a similar course as $^{14}N^{14}N$ after a room air embolus. This suggests that the solubility and other physical characteristics are the same. Sensitivity of detection on a modified mass spectrometer utilizing the carbon monoxide channel is 0.001 per cent.

End-tidal carbon dioxide measurement in the critically ill neonate: a comparison of two capnometers

M.E. McLeod, B.A.B. McEvedy, H. Kirpalani, G.A. Volgyesi, J. Lerman

Department of Anaesthesia and the Research Institute, The Hospital for Sick Children, University of Toronto, Toronto, Ontario

Non-invasive estimation of arterial CO_2 ($PaCO_2$) in the critically ill neonate can be an advantage both in the neonatal intensive care unit (NICU) and the operating room. Current methods of end-tidal CO_2 ($PaCO_2$) measurement, however, do have limitations in this group of patients. Although transcutaneous estimates may be more accurate, they are less commonly available in the operating room. Two end-tidal CO_2 monitors in routine use are the Puritan-Bennett/Datex (PB/D) capnometer which aspirates gas continuously from the circuit and analyzes it in a remote infrared sensor and the Hewlett-Packard (HP) capnometer which uses an optical infrared sensor for in-line gas analysis. It has been shown that the accuracy of the PB/D can be improved in the small infant by using a catheter to sample from the distal end of the endotracheal tube.[1] This study was undertaken to compare the accuracy of the PB/D capnometer with distal sampling to that of the HP capnometer with its in-line sensor.

Methods

After approval from the Committee on Human Research, 20 simultaneous measurements of $PETCO_2$ and $PaCO_2$ were obtained from 20 intubated neonates (tube size ≧ 3.0 mm) in the NICU. A transcutaneous CO_2 ($PTCO_2$) and O2 (PTO_2) monitor was calibrated and allowed to stablize before the study period to determine the effects of the insertion of the capnometers into the breathing circuit. After the capnometers were calibrated, the neonatal sensor of the HP capnometer was inserted into the breathing circuit. For the PB/D, $PETCO_2$ was sampled through a 19-gauge catheter which was inserted through a modified elbow to the tip of the endotracheal tube. PTO_2 and $PTCO_2$ were recorded before and 15 minutes after insertion of the capnometers. An arterial blood sample was then obtained from an indwelling arterial cannula. Regressions between the two end-tidal measurements and the $PaCO_2$ were determined using least squares linear regression analysis. Transcutaneous values before and after capnometer insertion were compared using paired Student's t tests. Statistical significance of $p < 0.05$ was accepted.

Results

The mean weight of the 20 infants was 2.41 ± 0.64 kg and the mean post-conceptual age was 36 ± 3 weeks. The relationship between $PaCO_2$ and $PETCO_2$ did not differ significantly for the two capnometers and both showed a good correlation with $PaCO_2$. The correlation coefficient, r, for distal $PETCO_2$ (PB/D) versus $PaCO_2$ was 0.81 and for $PETCO_2$ (HP) versus $PaCO_2$ was 0.78. The insertion of the capnometers into the breathing circuit had no effect on PTO_2. The $PTCO_2$, however, increased significantly from 37.8 ± 9.3 to 39.9 ± 9.7 mmHg after insertion ($p < 0.05$). In three infants, clinically significant increases of 6–7 mmHg were observed.

Discussion

The measurement of alveolar CO_2 in infants depends in part on the sample flow rate and response time of the capnometer when a device such as the PB/D is used.[2] The HP, which measures $PETCO_2$ within the circuit has neither of these limitations. However, when a distal technique is used, comparable results can be obtained with the PB/D. It has been shown previously that insertion of the aspirating catheter of the PB/D into the tube and sampling at 150 ml·min^{-1} has no effect on either $PTCO_2$ or $PTCO_2$.[1] The significant increase in $PTCO_2$ seen in this study must therefore be attributed to the neonatal sensor of the HP capnometer as has been reported by others.[3] This suggests that the PB/D capnometer with distal sampling may be more appropriate in the critically ill neonate.

References

1 *McEvedy BAB, McLeod ME, Munini M, Kirpalani H, Lerman J.* A comparison of two non-invasive estimates of $PaCO_2$ in critically ill neonates. Anesthesiology 1987; 67: A485.

2 *Dumpit FM, Brady JP.* A simple technique for measuring alveolar CO_2 in infants. J Appl Physiol 1978; 45: 648.

3 *Epstein MF, Cohen AR, Feldman HA, Raemer DB.* Estimation of $PaCO_2$ by two noninvasive methods in the critically ill newborn infant. J Pediatrics 1985; 106: 282.

Vd/Vt, Qs/Qt and the end-tidal to arterial PCO_2 gradient

F.A. Burrows
Department of Anaesthesia and the Research Institute, The Hospital for Sick Children, University of Toronto, Toronto, Ontario

The end-tidal to arterial PCO_2 gradient (ΔPCO_2) results from two factors; the dead space to tidal volume ratio (Vd/Vt) and the venous admixture (Qs/Qt). An increase in either increases the ΔPCO_2. Children with congenital heart disease (CHD) have abnormalities of both Vd/Vt and Qs/Qt which result in an increased ΔPCO_2.[1] The relationship of the ΔPCO_2 to the Vd/Vt and Qs/Qt has not been investigated. The purpose of this study was to examine the relationship of the Vd/Vt and Qs/Qt to the ΔPCO_2 in children with CHD.

Methods

After approval from the Committee on Human Research 41 patients with cyanotic or acyanotic CHD scheduled for palliative or corrective cardiac surgery were studied. The induction of general anaesthesia was followed by controlled ventilation with an Air Shields Ventimeter® and Mapleson D breathing circuit. Ventilatory parameters and fresh gas flows were adjusted to provide a $PaCO_2$ in the clinical range (30–35 mmHg). Gas flows and expired volumes were measured using a Pitot tube.[2] Distal end-tidal PCO_2 ($PETCO_2$) was measured continuously by infrared analysis. Arterial and mixed venous blood for analysis was drawn simultaneously with the recording of the $PetCO_2$. Expired gas was collected in a Douglas bag to calculate CO_2 production. The study was performed after sternotomy with the patient in the supine position at 37° C rectal temperature. The patient was ventilated for five minutes undisturbed prior to the study to obtain steady state conditions. $PetCO_2$ was corrected for barometric pressure and water vapour pressure. Vd/Vt and Qs/Qt were calculated using standard formulae.[3] The relationship between the $PetCO_2$ and $PaCO_2$; ΔPCO_2 and Vd/Vt; ΔPCO_2 and Qs/Qt; Vd/Vt and Qs/Qt were determined by least squares linear regression. Slopes were compared using Student's t test. Multiple linear regression analysis determined the relationship between ΔPCO_2, Qs/Qt and Vd/Vt. Statistical significance was accepted at $p \leq 0.05$.

Results

Patients varied in weight from 2.93 to 55.4 kg (mean ± SD = 15.29 ± 15.2 kg). $PETCO_2$ underestimated the $PaCO_2$ in all patients studied with a mean (± SD) ΔPCO_2 of 7.46 (± 4.87) mmHg (Figure). The ΔPCO_2 increased as the Vd/Vt and Qs/Qt increased. The coefficient of determination (r^2) between ΔPCO_2 and Vd/Vt = 0.5 and between ΔPCO_2 and Qs/Qt = 0.46. Both correlate directly and linearly with the ΔPCO_2. The linear relationship between Vd/Vt and Qs/Qt is poor (r^2 = 0.32). Multiple linear regression describes the relationship as: $\Delta PCO_2 = 1.55 + 10.91\,(Qs/Qt) + 13.38\,(Vd/Vt)$ ($r^2 = 0.69$).

Discussion

The $PETCO_2$ underestimates the $PaCO_2$ in children with CHD and the ΔPCO_2 increases as the Vd/Vt and Qs/Qt increase. The effect of an increase in Vd/Vt on the ΔPCO_2 has been described, but the ΔPCO_2 is affected independently by both the Vd/Vt and the Qs/Qt. The Vd/Vt is the primary determinant of the ΔPCO_2 but the Qs/Qt becomes increasingly important as the magnitude of the Qs/Qt increases.

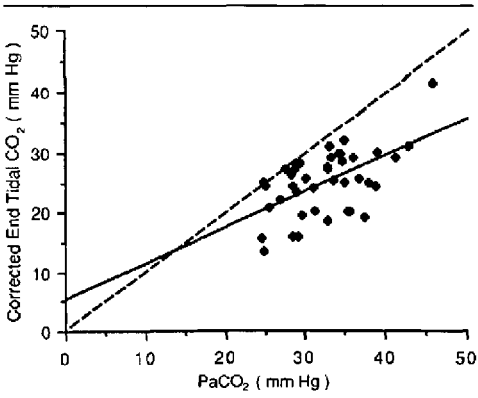


FIGURE The regression line for the correlation between $PETCO_2$ and $PaCO_2$ is represented by the continuous line (——) and is described by the equation: $y = 5.50 + - 0.60x$ ($r^2 = 0.32$) THe line of identity is represented by the line (---).

References

1 *Lindhal SGE, Yates AP, Hatch DJ.* Relationship between invasive and noninvasive measurements of gas exchange in anesthetized infants and children. Anesthesiology 1987; 66: 168–75.
2 *Wolf AR, Volgyesi GA.* A modified Pitot tube for the accurate measurement of tidal volume in children. Anesthesiology 1987; 67: 775–8.
3 *Nunn JF.* Applied Respiratory Physiology. Second edition. London, Butterworths, 1978, pp. 274–309, pp. 227–32.

Oxygen saturation (SaO_2) is unreliable for the early detection of oesophageal intubation in rodents

H. Vaghadia, L.C. Jenkins, R.W. Ford
Department of Anesthesiology, Vancouver General Hospital and Shaughnessy Hospital, The University of British Columbia, Vancouver, British Columbia

Pulse oximetry has been suggested as a monitoring tool that may facilitate early detection of oesophageal intubation.[1] However, no data are available on the changes in oxygen saturation that may be expected immediately after oesophageal intubation. We attempted to determine whether there is significant oxygen desaturation after one minute of oesophageal ventilation in the rodent model.

Methods

Twenty female Wistar rats (mean weight 257 g; range 226–286)

TABLE Arterial Blood Gas results (Mean + SD)

	During induction	*Tracheal ventilation*	*Oesophageal ventilation*
pH	7.31 + 0.09	7.52 + 0.07	7.39 + 0.04
$PaCO_2$ (mmHg)	52.6 + 13.4	28.2 + 4.1	43.1 + 5.8
PaO_2 (mmHg)	108.3 + 45.6	147.8 + 54.5	44.3 + 21.5
SaO_2	95.3 + 44.4	98.5 + 2.5	67.8 + 27.7

each with a chronically implanted carotid artery cannula were studied. Each animal was anaesthetized with four per cent halotane in oxygen and N_2O (FIO_2 = 0.5). Ten minutes after induction, simultaneous intubation of the trachea and oesophagus was achieved with 14-gauge Jelco cannulae. The tracheal cannula was first connected to a Harvard rodent ventilator (rate 100/min; tidal volume 4.5 ml) and the animal was ventilated with 1.5 per cent halothane in an oxygen/nitrous oxide mixture (FIO_2 = 0.5). After one minute of tracheal ventilation, arterial blood gases (ABGs) were sampled and the ventilator was then connected to the oesophageal cannula. ABGs were then taken after 60 seconds of oesophageal ventilation. Finally, the tracheal cannula was reconnected to the ventilator and the animal was ventilated with 100 per cent oxygen until awake. Four animals died during the study.

Results

The data are summarized in the Table and Figure. As shown in the Figure, 7 of 16 animals (43 per cent) failed to demonstrate an SaO_2 below 85 per cent after one minute of oesophageal ventilation.

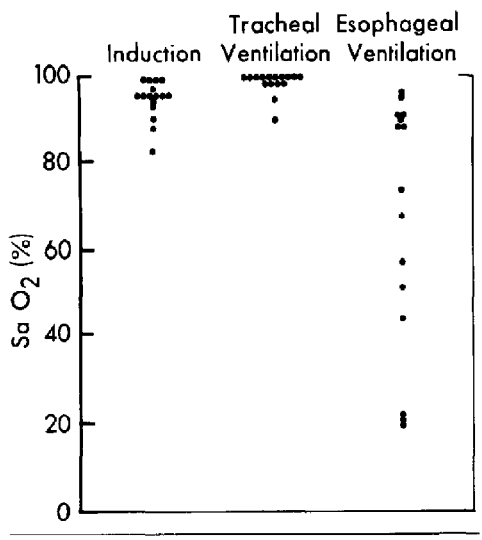


FIGURE Oxygen saturation. Measurements were made one minute after oesophageal intubation.

Discussion

This study demonstrates that reliable changes in SaO_2 do not occur after one minute of oesophageal ventilation. This would suggest that pulse oximetry would be of limited use for the *early* detection of oesophageal intubation.

Reference

1 *Vejima T.* Esophageal intubation. Anesth Analg 1987; 66: 481.

Systolic arterial pressure determination by a new pulse monitor technique

D.T. Wong, R.D. Valley, G.A. Volgyesi, B. Bissonnette, J. Lerman

Department of Anaesthesia and the Research Institute, The Hospital for Sick Children and the University of Toronto, Toronto, Ontario

The doppler ultrasound technique has been widely used in paediatrics to accurately estimate systolic arterial blood pressure (SBP). However, the doppler technique can be affected by electrical interference e.g., electrocautery, sensor probe displacement and movement artifacts. In order to minimize the problems associated with the doppler ultrasound, a new pulse monitor (Mr Pulse) was recently developed. The purpose of this study was to compare the accuracy of SBP measurements obtained with Mr Pulse to a standard doppler technique.

Methods

This new pulse monitor is based on the principle of a finger plethysmograph which transforms a change in volume into a change in pressure. The sensor consists of a small air-filled chamber that is taped to the pulp of a distal phalanx (Figure 1). An increase in the pulp volume with each arterial pulsation decreases the volume of the sensor. This change in volume is transmitted as a pressure wave along a narrow plastic tubing to the pressure transducer in the monitor. There, the pressure wave is transformed into an audible signal.

Eleven ASA physical status I or II patients aged 18 days to 13 years undergoing general anaesthesia were studied. Standard aneroid sphygmomanometers with appropriately sized cuffs and

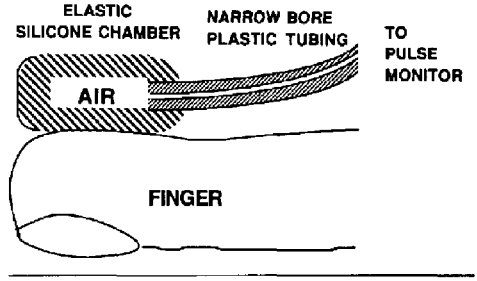


FIGURE 1 Diagram of the Mr Pulse sensor.

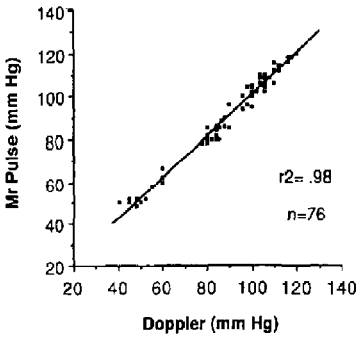


FIGURE 2 Systolic arterial pressure: Mr Pulse vs Doppler.

Parks Electronics Model 811 Ultrasound Doppler units were used. The Mr Pulse sensor was applied to the distal phalanx with tape and the doppler sensor was applied over the ipsilateral radial artery. To obtain a SBP measurement, the blood pressure cuff was inflated until the pulse and doppler signals were eliminated. The cuff was then deflated at a rate of 2 mmHg/sec. The pressures at which the Mr Pulse and doppler signals reappeared were recorded. SBP values obtained with both techniques were compared using least squares linear regression analysis and the coefficient of determination (r^2).

Results

Seventy-six paired systolic arterial blood pressure (SBP) measurements were obtained in 11 patients. The mean SBP reading was 92.0 mmHg for the Mr Pulse group and 90.6 mmHg for the doppler group. There was a high index of correlation of SBP between the Mr Pulse and the doppler technique ($r^2 = 0.98$, Figure 2).

Discussion

Although invasive SBP measurements by peripheral arterial cannulation is considered a standard for estimating the "true SBP," many have acknowledged its inaccuracies and limitations. First, the invasive nature and associated complications limit its use.[1] Secondly, invasive SBP measurements are subject to the errors of zeroing and calibration as well as underdamping and overdamping of the transducer system.[2] Doppler ultrasound is considered an accurate noninvasive technique to estimate SBP. The doppler estimated SBP has shown a close correlation to that obtained by arterial cannulation.[3]

Mr Pulse is as accurate as the doppler technique in estimating SBP. In comparison to the doppler ultrasound, Mr Pulse has the following advantages: the sensor is quick and easy to apply; the sensor position is not critical; it is not subject to electrical interference, e.g., electrocautery; there is no electrical hazard and it is relatively inexpensive.

References

1 *Slogoff S, Keats AS, Arlund C.* On the safety of radial artery cannulation. Anesthesiology 1983; 59: 42.

2 *Gardner RM.* Direct blood pressure measurement–dynamic response requirements. Anesthesiology 1981; 54: 227.

3 *Reder RF, Dimich I, Cohen ML, Steinfeld L.* Evaluating indirect blood pressure measurement techniques. A comparison of three systems in infants and children. Pediatrics 1978; 62: 326.

Non-invasive haemodynamic monitoring during Caesarean section in patients with pregnancy-induced hypertension

M.L. Flanagan, J. Fuller, P.L. Ross, D.R. Gambling
Division of Obstetrical Anaesthesia, University of British Columbia and Grace Hospital, Vancouver, British Columbia

Pregnancy-induced hypertension (PIH) occurs in five to ten per cent of all pregnancies and is a major cause of maternal morbidity and mortality. Comparison of the haemodynamic changes in these patients versus normal patients during Caesarean section has been limited by the risks associated with use of the pulmonary artery catheter for thermodilution measurements. Impedance cardiography is a non-invasive method to monitor cardiac output and has been shown to correlate well with the thermodilution[1] and dye dilution[2] techniques during Caesarean section. The purpose of the present study was to compare the haemodynamic changes occurring during Caesarean section between pregnancy-induced hypertensive and normal patients using a non-invasive technique.

Methods

Following approval by the University of B.C. Screening Committee for Research involving human subjects, informed consent was obtained from ten normal elective and ten pregnancy-induced hypertensive patients undergoing Caesarean section. The pregnancy-induced hypertensive patients had been treated with magnesium sulphate prior to Caesarean section. After the patients had received an intravenous infusion of at least 1.0 L of lactated Ringer's solution lumbar epidural anaesthesia was administered using carbonated lidocaine ($17.3\ mg \cdot ml^{-1}$) in sufficient volume to achieve an analgesic level of T_4. Multiple determinations of the cardiac output were made using a transthoracic impedance method prior to administration of lidocaine (in the supine and left lateral positions) at the beginning of surgery, delivery and 5, 10, 15, 30, 60, 90 and 120 minutes after delivery. Maternal heart rates and blood pressures were also monitored. Statistical analysis was done by the t test for unpaired data. A p value < 0.05 was considered significant.

Results

The two groups of patients were similar with respect to age, height, weight and gestation. The cardiac output in both groups rose slightly with delivery and decreased until 60 minutes after delivery. The patients with pregnancy-induced hypertension had significantly lower cardiac outputs than normals until 60 minutes post partum ($p < 0.05$) (Figure). This was associated with a higher mean blood pressure and higher systemic vascular resistance than normal patients. There were no significant differences in heart rate between the two groups.

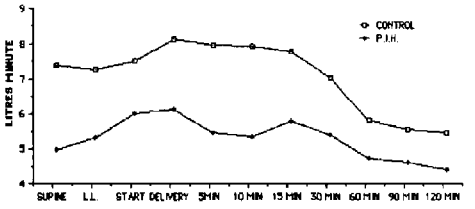


FIGURE Mean cardiac output.

Discussion

Case reports[3] of severe PIH patients who warranted pulmonary artery catheterization have suggested that these patients have a hyperdynamic circulation. In our study, the patients had not received vasodilator therapy and hence they had high systemic vascular resistances and low cardiac outputs just prior to delivery and up until 60 minutes post partum. This finding lends support to the mechanism of PIH being related to release of a renin-like substance.

References

1 *Secher NJ et al.* Measurements of cardiac stroke volume in various body positions in pregnancy and during Caesarean section. A comparison between thermodilution and impedance cardiography. Scan J Clin Lab Invest 1979; 39: 569–76.

2 *Milsom I et al.* Measurement of cardiac stroke volume during Caesarean section: a comparison between impedance cardiography and the dye dilution technique. Acta Anaesthesiol Scand 1983; 27: 421–26.

3 *Rolbin SH et al.* Haemodynamic monitoring in the management of severe pre-eclampsia and eclampsia. Can Anaesth Soc J 1981; 28: 363–69.

Flammability of nasopharyngeal airways, oesophageal stethoscopes, nasogastric tubes, and feeding tubes in oxygen- and nitrous oxide-enriched atmospheres

G.L. Wolf, J.I. Simpson

State University of New York/Health Sciences Center at Brooklyn, Department of Anesthesiology, Brooklyn, New York

Operating room fires continue to represent a hazard to anaesthetized patients, in spite of the rare use of flammable anaesthetic agents. This is related, in part, to the introduction of new potential fuels and ignition sources into an oxygen/nitrous oxide-enriched atmosphere. In order to define the risk, we have examined the flammability of various plastic and rubber materials placed into the upper airway of anaesthetized patients.

Since our report of $Oxidant_{O_2}$ and $Oxidant_{N_2O}$ Indices of Flammability of endotracheal tubes,[1] we have examined the flammability indices of nasopharyngeal airways, oesophageal stethoscopes, nasogastric tubes, and feeding tubes in oxygen- and nitrous oxide-enriched atmospheres.

The Index of Flammability is defined as the minimum fraction of oxidant (oxygen and/or nitrous oxide) in diluent gas (nitrogen) that will support candle-like flame of a material in a test chamber.

TABLE I $Oxidant_{O_2}$ indices of flammability

	$Oxidant_{O_2}$ index (±SD)	*99% Confidence interval*
Oesophageal stethoscopes	0.218 ± (0.011)	0.020
Salem sump nasogastric tubes	0.229 ± (0.006)	0.011
Enteric feeding tubes	0.192 ± (0)	*NV
Plastic nasopharyngeal airways	0.196 ± (0.006)	0.011
Rubber nasopharyngeal airways	0.172 ± (0)	*NV

*NV = no variability to third decimal with n = 5.

TABLE II $Oxidant_{N_2O}$ indices of flammability

	$Oxidant_{N_2O}$ index (±SD)	*99% Confidence interval*
Oesophageal stethoscopes	0.430 ± (0.007)	0.013
Salem sump nasogastric tubes	0.430 ± (0.003)	0.006
Enteric feeding tubes	0.375 ± (0)	*NV
Plastic nasopharyngeal airways	0.415 ± (0.005)	0.010
Rubber nasopharyngeal airways	0.366 ± (0.004)	0.077

*NV = no variability to third decimal with n = 5.

$Oxidant_{O_2}$ Indices of Flammability are listed in Table I. $Oxidant_{N_2O}$ Indices of Flammability are listed in Table II.

All tested materials are flammable in the range of oxygen concentrations common to the clinical setting. We therefore recommended avoidance of the use of these potentially flammable materials in close proximity to an ignition source (laser, high-frequency electrosurgery, high-frequency electrocautery).

Reference

1 *Wolf GL, Simpson JI.* Flammability of endotracheal tubes in oxygen and nitrous oxide enriched atmosphere. Anesthesiology 1987; 67: 236–9.

Interscalene brachial plexus blockade and chronic renal failure – a pharmacokinetic study

R. McEllistrem, J. Schell, K. O'Malley, A.J. Cunningham

Department of Anaesthesia, Royal College of Surgeons in Ireland, Dublin, Ireland

Interscalene brachial plexus block is an ideal anaesthetic technique for arterio-venous (AV) fistula creation in patients with end-stage renal disease. The hazards of general anaesthesia in this high-risk patient population are avoided while upper limb vasodilation, secondary to sympathetic blockade, may facilitate a successful surgical outcome. Previous studies have reported either no difference in duration of anaesthesia following brachial

plexus block between patients with normal and impaired renal function[1] or a significant reduction in the duration of anaesthesia in patients with chronic renal failure.[2]

The objectives of this study were to compare the latency of onset and duration of anaesthesia in patients with normal and impaired renal function following lidocaine interscalene brachial plexus block and to compare the systemic absorption of the drug injected in both patient populations.

Methods

Following ethics committee approval and informed consent 16 patients (ASA physical status I–III) (5 M/11 F) undergoing interscalene brachial plexus block to facilitate upper limb surgery were prospectively studied. Group 1 (n-8) patients had normal renal function while Group 2 (n-8) patients were undergoing A-V fistula creation prior to haemodialysis for chronic renal failure management.

All patients received diazepam 0.15 $mg \cdot kg^{-1}$ two hours before surgery. Following isolation of the brachial plexus using a peripheral nerve stimulator, 0.25 $ml \cdot cm^{-1}$ height lidocaine one per cent solution was injected over a 30-second period. Latency and duration of anaesthesia were determined by pin-prick. The latency time was assessed from commencement of injection of lidocaine to the development of pin-prick analgesia. The duration of analgesia was recorded as time from development of analgesia at the surgical site until the first awareness of pain. Monitoring techniques included automated blood pressure recordings and continuous electrocardiographic display.

Ten ml venous blood aliquots were withdrawn from the contralateral ante cubital fossa at 0, five, ten, 15, 20, 30 and 60 minutes following local anaesthetic administration. Following centrifugation the supernatant plasma was stored at −20° C prior to lidocaine assay by gas chromatography.

The data are expressed as mean ± standard deviation (SD). Statistical analysis between and within the two groups included unpaired Student's t test and repeated multivariate analysis of variance (MANOVA), as appropriate. $p < 0.05$ was considered significant.

Results

Significant biochemical, acid-base and haematological abnormalities were noted in Group 2 patients (Table). The latency of onset and duration of anaesthesia were similar in the two groups studied. Systemic absorption of lidocaine was more rapid in chronic renal failure patients ten mintues following brachial plexus blockade (Figure). C_{max} lidocaine levels were similar in the two patient populations.

TABLE Patient demographics and laboratory assessment – data expressed as Mean ± SD (*p < 0.05)

	Group 1 Normal renal function		*Group 2 Chronic renal failure*
n	8		8
M/F	2/6		3/5
Age (y)	33.5 ± 18.0		47.4 ± 17.7
Hct.	0.36 ± 0.3	*	0.27 ± 0.05
Serum albumin	40.0 ± 3.6	*	37.0 ± 2.3
Serum bicarbonate	22.4 ± 1.2	*	19.9 ± 3.2
Latency of onset (min)	8 ± 2		10 ± 3
Duration of action (min)	46 ± 17		59 ± 19

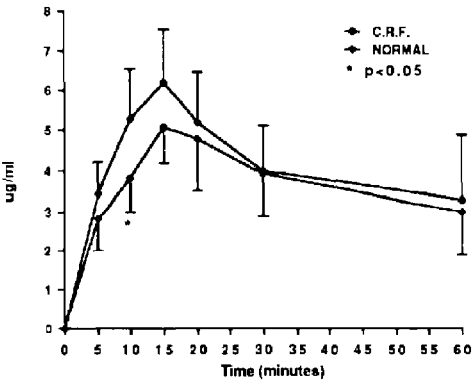


FIGURE Mean plasma lidocaine levels ± S.D.

Discussion

The plasma lidocaine concentration recorded following perineural injection was determined by the extent of systemic absorption, distribution, drug metabolism and excretion. Increased vascularity of the injection site and local tissue pH differences may account for the rapid systemic lidocaine absorption in chronic renal failure patients. Despite the high C_{max} levels recorded in both groups, no patient developed systemic toxicity. One per cent lidocaine solution may be safely administered to patients with normal and impaired renal function to provide effective brachial plexus blockade for short surgical procedures.

References

1 *Beauregard L, Martin R, Tetrault JP.* Brachial plexus block and chronic renal failure. Can J Anaesth 1987; 34: S118.

2 *Bromage PR, Gertel M.* Brachial plexus anesthesia in chronic renal failure. Anesthesiology 1972; 36: 488–93.

Alkalinization of mepivacaine for axillary brachial plexus block

S. Berman, R.W. Rosenquist, B.T. Finucane
Grady Memorial Hospital and Emory University, Atlanta, Georgia

Reduced anaesthetic latency following brachial plexus block has long been desired to facilitate its use in the busy clinical setting. Several investigators proposed and studied either carbonated or alkalinized solutions of local anaesthetic to reduce latency.[1,2]

Studies which employ the subclavian perivascular approach to the brachial plexus demonstrated decreased onset time to motor and sensory block. The purpose of this study was to determine if alkalinization of a one per cent mepivacaine and 1:200,000 epinephrine solution would decrease the onset time of motor and sensory loss following axillary brachial plexus block. In addition, plasma mepivacaine levels were measured to determine if alkalinization caused a change in absorption kinetics of mepivacaine from the axillary space.

Methods

Institutional approval and informed consent were obtained from 20 ASA physical status class I–III patients scheduled for axillary plexus blocks. They were randomly assigned to one of two groups: the bicarbonate group, or the control group.

The Pharmacy prepared the anaesthetic solutions in double-blind fashion. The solutions were identical except for the addition of bicarbonate to one. Fresh epinephrine was added to each solution just prior to administration such that the final mixture contained 10 $mg \cdot ml^{-1}$ of mepivacaine and 1:200,000 epinephrine.

Prior to performing the axillary block, a 20-gauge 2" catheter was inserted into the radial artery of the contralateral arm for sampling.

Axillary brachial plexus blocks were performed using a catheter technique. An 18-gauge catheter over an atraumatic 20-gauge needle was inserted into the axillary sheath. The position was confirmed with a nerve stimulator, the needle removed, and the cathether taped securely in place. Seven $mg \cdot kg^{-1}$ of the local anaesthetic solution then was injected over a two-minute period. Digital pressure was applied to the brachial plexus sheath immediately distal to the injection site to facilitate proximal spread.

The loss of motor function was documented by testing each of five nerves: (1) radial nerve – extending distal phalanx of thumb, (2) median nerve – adduction of the thumb, (3) ulnar nerve – abduction and abduction of the fingers, (4) musculocutaneous nerve – flexion of the forearm, (5) axillary nerve – adduction of the arm. Each nerve scored 0 if normal strength was present, 1 if paresis was present and 2 if paralysis was present. The measurements were made at 5, 10, 20, 30, 40, 50 and 60 minutes.

The loss of sensory function was documented by testing each of five nerves to pinprick: (1) radial nerve – dorsum of the hand at the base of the index finger, (2) median nerve – palmar base of the index finger, (3) ulnar nerve – palmar base of the little finger, (4) musculocutaneous nerve – over the site of the radial artery, (5) axillary nerve – skin over the lower half of the deltoid. Each nerve was scored 0 if normal, 1 if reduced sensation was present, and 2 if sensation was absent. The measurements were made at 5, 10, 20, 30, 40, 50 and 60 minutes.

Plasma mepivacaine levels were obtained before injection and at 5, 10, 15, 20, 25, 30, 35, 40, 50, 60, 70, 80, 90, 100, and 120 minutes and analyzed by gas chromatography.

Results

Axillary brachial plexus blocks succeeded in 8/9 cases in the control group and 10/11 in the bicarbonate group. The failure in the control group was the result of insufficient time between placement of the block and the start of surgery. The block was complete when the patient awoke from general anaesthesia. One patient in the bicarbonate group complained of ringing in the ears after receiving an initial dose. The catheter likely was intravenous and the patient was withdrawn from the study.

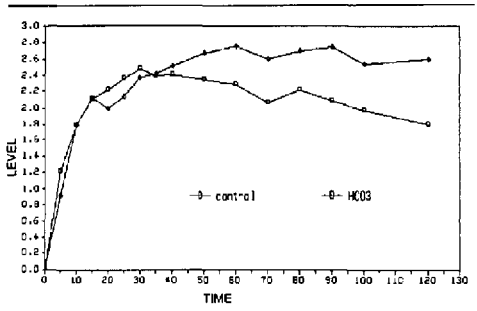


FIGURE Plasma mepivacaine levels ($\mu g \cdot ml^{-1}$).

The results of both motor and sensory scores were analyzed using the Wilcoxon sign rank test. There were no significant differences between the two groups with regard to latency of motor or sensory loss.

Mepivacaine levels were analyzed using analysis of variance with repeated measures. Plasma mepivacaine levels did not differ significantly when comparing the plain group and the alkalinized group.

Discussion

Local anaesthetic activity probably depends on nerve membrane penetration by the non-ionized local anaesthetic. The percentage of the non-ionized form can be increased by raising the pH of the solution. Hilgier published the only study investigating alkalinization of local anaesthetic for brachial plexus blockade. Using a subclavian perivascular model, he concluded that alkalinization of bupivacaine resulted in more rapid onset of sensory analgesia.[1] However, the methods and endpoints in this study were defined poorly and the effect of alkalinization on motor block or plasma local anaesthetic levels were not addressed.

Plasma levels of mepivacaine did not differ significantly when comparing the two groups and the levels remained well below the toxic range. This agrees with DiFazio's findings using plain and alkalinized lidocaine solutions in the epidural space.[3]

In this double-blind study, onset time for motor or sensory block using an axillary brachial plexus model was not influenced by alkalinization of mepivacaine-epinephrine solution. Our data suggest that, despite the theoretical considerations, alkalinization of mepivacaine produces no clinical advantage in axillary brachial plexus block. Further, the time required to add another drug and the dilution of the local anaesthetic may represent disadvantages.

References

1 *Hilgier M.* Alkalinization of bupivacaine for brachial plexus blocks. Regional Anesthesia, April–June, 1985; 58–61.

2 *Sukhani R, Winnie A.* Clinical pharmacokinetics of carbonated local anesthetics I: subclavian perivascular brachial block model. Anesth Analg 1987; 66: 739–45.
3 *DiFazio CA et al.* Comparison of pH-adjusted solutions for epidural anesthesia. Anesth Analg 1986; 760–4.

Evaluation of EMLA in providing split thickness skin graft donor site analgesia

B. Bucyk, H.L. Carle, D. LeGatt, B. Finegan
Departments of Anaesthesia and Laboratory Medicine, University of Alberta Hospitals, Edmonton, Alberta

An eutectic mixture of lidocaine base and prilocaine base (EMLA) has been shown to penetrate intact skin and abolish the pain response to pinprick and intravenous cannulation.[1] The objectives of this study were (1) to evaluate the efficacy of EMLA in providing analgesia for split thickness skin graft donor sites and (2) assess any untoward effects of EMLA when used in combination with a 1.5 per cent CO_2 lidocaine-induced brachial plexus block.

Methods

This study was approved by the hospital ethics committee and informed consent was obtained. Ten ASA physical status I and II patients (age 16–67) scheduled for upper limb skin grafting were studied. After premediation of morphine 0.15 $mg \cdot kg^{-1}$, 60 g of EMLA was applied to the lower limb donor site (surface area 200 cm^2). Thirty minutes prior to surgery a supraclavicular brachial plexus block using 6 $mg \cdot kg^{-1}$ of 1.5 per cent CO_2 lidocaine with epinephrine 1/400,000 was established. Heart rate, blood pressure and serum lidocaine levels were measured. Lidocaine concentrations in serum were determined using an automated fluorescence polarizaton immuno-assay technique (TDx-Analyzer, Abbott Laboratories). Prior to graft harvesting the donor site was sterilized, inspected for adverse skin reaction and tested for pinprick sensation. At the start of skin harvesting, using a Padgett electric dermatome (setting 12/1,000 inch), patients rated sensation as none, pressure, slight pain or severe pain.

Results

One patient withdrew from the study. No significant changes in blood pressure from control values were seen; however, a significant increase in heart rate ($p < 0.05$) from control was noted at the time of establishing the supraclavicular brachial block. No patients developed symptoms or signs of lidocaine toxicity and the highest serum lidocaine concentration measured was 3.8 $\mu g \cdot ml^{-1}$. All donor sites exhibited a mild pallor, but none of the patients complained of skin irritation. Five patients were sensitive to touch immediately prior to graft harvesting, but only one patient experienced pain at the donor site during skin harvesting. This pain was at the margins of the graft area. All patients had good graft survival and donor site healing on follow-up.

Discussion

From these results it appears that EMLA when combined with a brachial plexus block is an acceptable alternative to general anaesthesia for split thickness skin grafting from lower to upper extremity. It is associated with minimal haemodynamic disturbance, absence of toxic side effects and good operating conditions. Although prilocaine has the potential to create methemoglobinaemia, its serum concentration after application of EMLA is negligible.[2] Proper application of the EMLA cream over the entire donor site is important and appropriate patient selection essential. This technique or modifications of it may be especially useful in patients with unstable circulatory and respiratory systems.

References

1 *Duhlwitz A.* Pain relief in vein puncture. Application time and effectiveness of lidocaine-prilocaine-cream. Anaesthetist 1985; 34: 355–8.
2 *Holst A.* An anaesthetic lidocaine/prilocaine cream (EMLA) for epicutaneous application tested for cutting split skin grafts. Scand J Plast Reconstr Surg 1985; 19: 201–9.

Plasma lidocaine levels following transtracheal injection during topical anaesthesia of the upper airway

P.E. Bigeleisen, J.Q. Schisler, B.T. Finucane
Department of Anesthesiology, Emory University and Grady Memorial Hospital, Atlanta, Georgia

The fiberoptic bronchoscope has become an indispensible tool when dealing with difficult airways. The technique of fiberoptic intubation is greatly facilitated by intravenous sedation as well as topical application of local anaesthetic and nerve blocks of the airway. The latter frequently includes a superior laryngeal nerve block as well as transtracheal injection of local anaesthetic. The purposes of this study were to assess patient comfort during fiberoptic intubation with and without transtracheal injection of lidocaine, and to define more accurately the contribution made by transtracheal injection to overall plasma lidocaine levels.

Methods

Twenty patients who predictably would require fiberoptic intubation were included in the study. The study was approved by the Institutional Review Board and informed consent was obtained from all patients. Patients were sedated preoperatively with 10 mg of diazepam PO. Upon arrival in the operating room, routine monitors were attached, an IV infusion started, and a catheter was inserted in the radial artery for sampling purposes. The patient was then further sedated with 5 mg of diazepam and 5 mg of droperidol IV. A fixed quantity of 0.25 per cent phenylephrine was sprayed into each nostril. Lidocaine four per cent (4 $mg \cdot kg^{-1}$) was applied topically to the nasal mucosa and nasopharynx using a hand-operated atomizer. Superior laryngeal nerve blocks were performed using 0.5 $mg \cdot kg^{-1}$ of one per cent lidocaine on each side. The patients were then prospectively and randomly assigned to two groups. The first group received 2 $mg \cdot kg^{-1}$ of four per cent lidocaine as a transtracheal injection (TTI). The second group received an additional equivalent dose of four per cent lidocaine topically to the nasopharynx (NP). Both groups received a total of 7 $mg \cdot kg^{-1}$ of lidocaine. After the

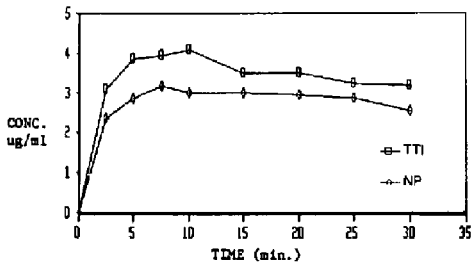


FIGURE Mean plasma lidocaine levels.

last injection of lidocaine, plasma levels were drawn every 2.5 minutes for ten minutes, and evey five minutes thereafter for 20 minutes. Fiberoptic laryngoscopy commenced approximately ten minutes afte the local anaesthetic was administered. After verification of endothracheal tube placement, general anaesthesia was induced with 4 $mg \cdot kg^{-1}$ of thiopentone and maintained with enflurane N_2O, and O_2. Ventilation, vital signs, and patient response were continuously monitored and recorded throughout the procedure. Patient comfort was evaluated retrospectively approximately 24 hours after the procedure. Plasma lidocaine levels were determined using a model 5840 Hewlett Packard gas–liquid chromatograph. Significance between the two groups was determined using a single-tailed Student's test.

Results

Mean plasma lidocaine levels averaged over 30 minutes were significantly higher ($p < 0.01$) in the TTI group than in the NP group. The mean peak lidocaine level in the TTI groups occurred at ten minutes and was 4.06 $\mu g \cdot ml^{-1}$, while the mean peak lidocaine level in the NP group occurred at 7.5 minutes and was 3.16 $\mu g \cdot ml^{-1}$. There was no statistical differnce between mean peak levels. One patient in the TTI group had a plasma lidocaine level greater than 6 $\mu g \cdot ml^{-1}$ (6.04 $\mu g \cdot ml^{-1}$). Similarly, one patient in the NP group had a plasma lidocaine level greater than 6 $\mu g \cdot ml^{-1}$ (6.57 $\mu g \cdot ml^{-1}$). The latter was the highest level achieved in the study. No signs of systemic toxicity occurred in either group.

Patients appeared equally comfortable in both groups. All patients coughed minimally during topical application of anaesthetic to the nasopharynx. All patients but one in the TTI group coughed vigorously during transtracheal injection. Two of these patients also bucked when the endotracheal tube was passed. All patients but two in the NP group coughed or bucked vigorously during laryngoscopy or intubation. One patient in each group was dissatisfied with the technique at postoperative interview. There was no qualitative difference in ease of intubation between the two groups. Mean time to intubation from the onset of laryngoscopy was six minutes in the TTI group, and eight minutes in the NP group. This difference was not statistically significant.

Discussion

Many clinicians are convinced that transtracheal injection of lidocaine is useful in preparing patients for fiberoptic intubation. The results of this study indicate that this is not so. Transtracheal injection does not predictably prevent coughing or bucking during the procedure. It merely alters the time at which it occurs, as demonstrated by the fact that all but one patient in the TTI group coughed during transtracheal injection, and two patients in this group bucked when the endotracheal tube was passed. Mean plasma lidocaine levels were significantly higher in the TTI group, but both groups had mean levels well below the toxic range. Mean peak levels were not statistically different between the two groups, and both groups had individual patients with plasma levels slightly above the level likely to cause mild toxic symptoms. In addition, the transtracheal technique exposes the patient to an additional uncomfortable puncture as well as the danger of trauma to the trachea and allied structures. Based on this study, the risk of systemic toxicity following transtracheal injection of lidocaine is minimal. It is also apparent, however, that there is little reason to routinely perform transthracheal injection of local anaesthetic in preparation for fiberoptic intubation.

MK-801, an NMDA antagonist, reduces volatile anaesthetic MAC and has haemodynamic and EEG effects in rabbits

M.S. Scheller, M.H. Zornow, A. Tateishi, J.C. Drummond, J.E. Fleischer, G.T. Shearman

Merck, Sharp and Dohme Research Laboratory, University of California at San Diego, La Jolla, California

MK-801 is a central nervous system glutamate antagonist that has recently been reported to ameliorate neuronal damage in an animal model of focal cerebral ischaemia.[1] When administered to awake rabbits and cats, MK-801 causes profound and long-lasting sedation. This suggests that MK-801 may have anaesthetic properties. In order to examine this possibility, we determined the effects of a bolus dose of MK-801 on halothane and isoflurane MAC, the electroencephalogram (EEG) and cardiovascular variables in rabbits.

Methods

Ten New Zealand White rabbits (3.0 ± 0.4 kg) were anaesthetized with either haothane or isoflurane in oxygen and orally intubated without the aid of muscle relaxants. Following intubation, the animals were ventilated with either 1.0 per cent halothane (n = 5) or 1.4 per cent isoflurane (n = 5) in oxygen so that end-tidal (ET) CO_2 was normal. A catheter was placed in a marginal ear vein for fluid and drug administration and the base of the tail was shaved. Monitored variables included mean arterial pressue (MAP), heart rate (HR), ET CO_2, ET volatile anesthetic (Puritan Bennett analyzer), temperature (servocontroled to 37°), and the EEG. Following and equilibration period, all variables were recorded (t = 0) and MK-801 (2.5 $mg \cdot kg^{-1}$ dissolved in saline) was infused IV over 5 min. All variables were recorded upon completion of the infusion (t = 5) and at 5

min intervals for 15 min (t = 10, 15, and 20). Once this initial cardiovascular data was obtained, MAC was determined according to the method of Eger *et al.*[2] using the tail clamp technique, beginning at either 1.0 per cent ET halothane or 1.4 per cent isoflurane, concentrations known to be well below established MAC values in the rabbit. Blood samples were obtained at the time of final tail clamping for determination of MK-801 levels. Although halothane and isoflurane MAC for rabbits had been previously determined to be 1.39 ± 0.23 per cent and 2.05 ± 0.19 per cent[3] respectively, halothane and isoflurane MAC were determined in an additional six animals which had not received MK-801 in order to confirm these values. MAP and HR data following MK-801 were compared to pre-MK-801 values with a repeated measures analysis of variance and Dunnett's t tests. MAC of the control animals and MAC of the animals given MK-801 were compared with an unpaired t tests. In all cases, $p < 0.05$ was considered statistically significant.

Results

MAC in the six control rabbits was 1.44 ± 0.10 per cent (mean ± SD) for halothane and 2.12 ± 0.19 per cent for isoflurane. In the animals given MK-801, halothane MAC was significantly reduced to 0.77 ± 0.12 per cent and isoflurane MAC to 0.70 ± 0.19 per cent ($p < 0.05$, Table). MAP and HR decreased significantly at all time points following the start of the MK-801 infusion in the halothane group (Figure 1). In the animals receiving isoflurane, HR was unchanged while MAP was significantly decreased following the administraton of MK-801 (Figure 2). In all halothane anaesthetized animals given MK-801, the predominant EEG frequency became slower and the amplitude increased during the drug infusion. In isoflurane anaesthetized animals, the EEG demonstrated isoelectricity or deep burst suppression. These patterns persisted throughout the experiment except for a high-frequency, low-amplitude activation pattern which always accompanied tail clamping. Plasma MK-801 levels are pending.

TABLE MAC data (mean ± SD)

	Control	*MK-801*
Halothane	1.44 ± 0.10%	0.77 ± 0.12*
Isoflurane	2.12 ± 0.19%	0.70 ± 0.19*

*Significant decrease from control value ($p < 0.05$).

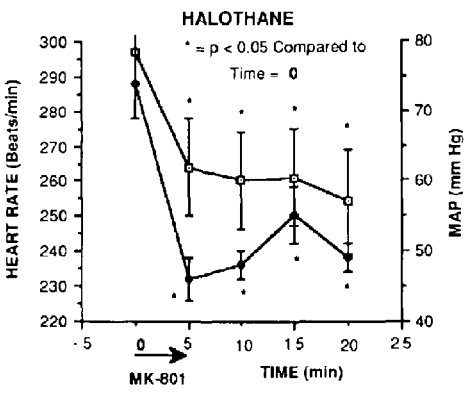


FIGURE 1 Cardiovascular responses to MK-801 in halothane anaesthetized animals (mean ± SEM). -□- Heart rate. -◆- MAP.

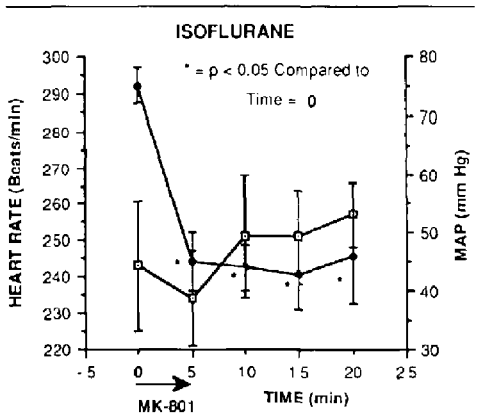


FIGURE 2 Cardiovascular responses to MK-801 in isoflurane anaesthetized animals (mean ± SEM). -□- Heart rate. -◆- MAP.

Discussion

The glutamate antagonist MK-801 has been shown to decrease neuronal injury in an animal model of focal ischaemia.[1] This study demonstrates that MK-801 also possesses significant anaesthetic properties as evidenced by a 57 per cent mean reduction in halothane and isoflurane MAC in the rabbit and the appearance of high amplitude slow waves (halothane) or isoelectricity (isoflurane) in the EEG following its administration. The decreases in mean arterial pressure and heart rate (halothane) seen following the administration of MK-801 may be consistent with its production of a deeper plane of anaesthesia, but could also reflect direct haemodynamic effects of the drug.

References

1 *Oyzurt E, Graham DI, McCulloch J, Woodruff GN.* The NMDA receptor antagonist MK-801 reduces focal ischaemic brain damage in the cat. J Cereb Blood Flow Metab 1987; 7, Suppl 1, S146.

2 *Eger EI, Saidman LJ, Brandstater B.* Minimum alveolar anesthetic concentration: a standard of anesthetic potency. Anesthesiology 1965; 26: 756–63.

3 *Drummond JC.* MAC for halothane, enflurane, and isoflurane in the New Zealand White Rabbit: and a test for the validity of MAC determinations. Anesthesiology 1985; 62: 336–8.

Factors affecting the disappearance of sevoflurane in Baralyme

D.T. Wong, G.A. Volgyesi, H.A. Strong, K. Miyasaka, J. Lerman

Department of Anaesthesia and The Research Institute, The Hospital for Sick Children, University of Toronto, Toronto, Ontario and the National Children's Medical Centre, Tokyo, Japan

Although sevoflurane has several desirable properties, Strum *et al.* raised concerns about its rapid disappearance in soda lime.[1] Therefore, we sought to identify those factors which affect the rate of disappearance of sevoflurane in Baralyme (BL).

Methods

This study was conducted in three parts: (Part 1) To compare the rate of disappearance of sevoflurane in BL and soda lime, 6 μl of liquid sevoflurane and 12 μl of liquid isoflurane were added to four 125 ml flasks containing 20 g of either fresh soda lime, fresh BL, dried exhausted BL or outdated BL. The flasks were sealed and immersed in a 37° C waterbath for three hours. Gas samples (0.5 ml) were withdrawn from the flasks at 5, 15, 30, 60, 120 and 180 minutes and the concentrations of sevoflurane and isoflurane were analyzed by gas chromatography. Assuming a logarithmic decrease in anaesthetic concentrations, the half lives ($t_{\frac{1}{2}}$) of sevoflurane and isoflurane were determined. (Part 2) To determine the effects of water and temperature on the rate of sevoflurane disappearance in BL, 6 μl of liquid sevoflurane and 12 μl of liquid isoflurane were added to eleven 125 ml flasks. Distilled water (0, 0.2, 0.4, 0.6 and 1.0 ml) was added to ten of these flasks (five at 37° C and five at 22° C) each containing 20 g of fresh BL. Liquid sevoflurane and isoflurane were added to one flask containing no BL as a control (37° C). Gas samples were withdrawn and analyzed as described above. (Part 3) To determine the stability of sevoflurane in an alkaline medium, 6 μl sevoflurane and 12 μl isoflurane were added to a 125 ml flask containing 20 ml of 0.1 M NaOH. The flask was immersed in a 37° C waterbath for 24 hours and shaken intermittently. The anaesthetic concentrations were analyzed as described above serially from 5 to 250 minutes.

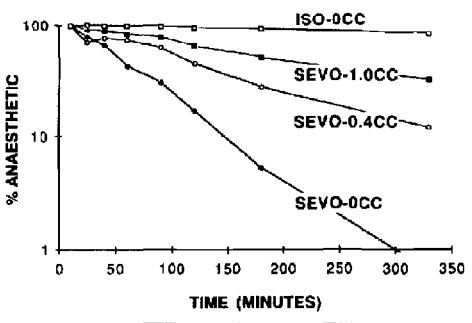


FIGURE 1 Sevoflurane disappearance in Baralyme. Effect of water (37° C).

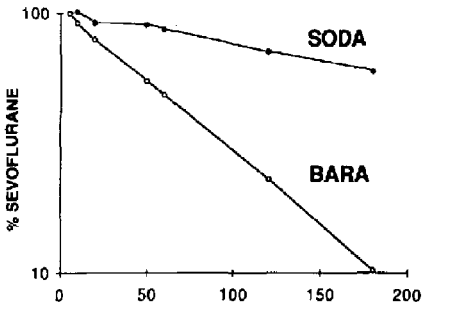


FIGURE 2 Sevoflurane disappearance. Baralyme vs soda lime (37° C).

TABLE Sevoflurane half life

Condition	*Half life (hours)*
Baralyme (37° C)	0.75
Baralyme (22° C)	5.25
Exhausted Baralyme (37° C)	100.0
Control (37° C)	105.0

Results

The effect of water on the rate of disapperance of sevoflurane in BL is shown in Figure 1. The data for 0.2 and 0.6 ml of water are omitted for clarity. The disappearance of sevoflurane in BL ($t_{\frac{1}{2}}$ = 1.0 hour) as compred to soda lime ($t_{\frac{1}{2}}$ = 3.9 hours) is shown in Figure 2. The half life of sevoflurane under different conditions are shown in the Table.

Discussion

We found that the rate of disappearance of sevoflurane in Baralyme (BL) decreased in the presence of water and with exhaustion of the absorber, and increased with increasing temperature. The rate of disappearance of sevoflurane in BL was four times that in soda lime. The rate of disappearance of sevoflurane when mixed with liquid NaOH did not differ significantly from that of the control. The increase in rate of disappearance of sevoflurane with temperature is consistent with the findings of Strum *et al.* There are two possible mechanisms to explain the disappearance of sevoflurane in BL; (1) physical adsorption by a sieve-like mechanism[2] and (2) chemical degradation. Failure of sevoflurane to disappear more rapidly in an alkaline medium does not support the notion of alkaline degradation of sevoflurane. The slower rate of disappearance of sevoflurane in BL in the presence of water and carbon dioxide suggests that this process may be self-limiting in a clinical setting. Further studies are required to determine the clinical significance of these findings.

Supported in part by a grant from the Maruishi Pharmaceutical Inc. Japan.

References

1 *Strum DP, Johnson BH, Eger II EI.* Stability of sevoflurane in soda lime. Anesthesiology 1987; 67: 779.

2 *Grodin WK, Epstein MAF, Epstein RA.* Enflurane and isoflurane adsorption by soda lime. Anesthesiology 1981; 55: A124.

Effects of AAGP, local anaesthetics and pH on the partition coefficients of halothane, enflurane and sevoflurane in blood and buffered saline

L. Sinclair, H.A. Strong, J. Lerman

Departments of Anaesthesia, Clinical Pharmacology and the Research Institute, The Hospital for Sick Children, University of Toronto, Toronto, Ontario

The blood gas partition coefficients of volatile anaesthetics increase with increasing age.[1] The reasons for this have not been fully elucidated although many factors, including the serum concentrations of albumin, cholesterol, and lipoproteins account for much of the difference.

Alpha-1-acid glycoprotein (AAGP) also increases with increasing age[2] but the effect of this protein on the solubility of volatile anaesthetics is unknown. We investigated the effect of physiological concentrations of AAGP (100 mg · dl^{-1}) and albumin (4 g · dl^{-1}) on the solubility of halothane, enflurane and sevoflurane in whole blood and in buffered saline (pH 7.4). Also, the effect of lidocaine and bupivacaine on the solubility of volatile anaesthetics in these solutions was determined. Finally, to determine the effect of pH on solubility, partition coefficients in blood at pH 7.0, 7.4 and 7.8 were measured.

Methods

The partition coefficients of the volatile anaesthetics were determined by the method described by Lerman *et al.*[3] In summary, 5 ml samples of each test solution were added to a 20 ml gas-tight glass syringe and then approximately 15 ml of a mixture of three vapours: 1.0 per cent halothane/1.0 per cent enflurane/0.5 per cent sevoflurane gas were aspirated into each syringe. After the syringes were incubated at 37° C for three hours and shaken intermittently, the concentrations of anaesthetics in the gas phase were determined by gas chromatography. Then 2 ml of each of the equilibrated test solutions were transferred to a 260 ml flask and after incubation at 37° C for one hour, the concentrations of the volatile anaesthetics in the gas phase were determined by gas chromatography.

The liquid/gas partition coefficient of each sample was determined using the equations described previously.[3]

Results and Discussion

The results shown in the Table are the means (± SD) of either two or three separate experiments. The partition coefficients of the anaesthetics in whole blood and buffered saline were similar to those published previously. Physiological concentrations of AAGP did not affect the solubility of the volatile anaesthetics in either of the test solutions. This is in contrast to the significant effect of albumin in buffered saline. Albumin accounts for a large proportion of the solubility of volatile anaesthetics in whole blood.

TABLE Partition coefficients of three volatile anaesthetics (mean ± SD)

	Halothane	*Enflurane*	*Sevoflurane*
Buffered saline (BS)	0.828 ± 0.034	0.783 ± 0.044	0.299 ± 0.017
BS & AAGP (100 mg · dl^{-1})	0.792 ± 0.022	0.765 ± 0.028	0.292 ± 0.006
Whole blood (WB)			
(27 mg · dl^{-1} AAGP)	2.614 ± 0.063	2.167 ± 0.107	0.717 ± 0.043
WB & AAGP (132 mg/dl)	2.524 ± 0.046	2.044 ± 0.052	0.710 ± 0.009
BS	0.894 ± 0.038	0.855 ± 0.124	0.458 ± 0.094
BS & albumin (4 g · dl^{-1})	2.457 ± 0.056	2.024 ± 0.042	0.718 ± 0.035
Blood – pH 7	3.185 ± 0.011	2.537 ± 0.005	0.820 ± 0.021
– pH 7.4	2.845 ± 0.234	2.362 ± 0.088	0.773 ± 0.039
– pH 7.8	2.91 ± 0.052	2.423 ± 0.015	0.764 ± 0.042

The addition of lidocaine (10 μg · ml^{-1}) and bupivacaine (10–50 μg · ml^{-1}) to albumin containing buffered saline and to whole blood respectively had no effect on the volatile anaesthetic partition coefficients. In summary, we found that the solubility of volatile anaesthetics was not affected significantly by AAGP, local anaesthetics or extremes of physiological pH.

References

1 *Lerman J, Gregory GA, Willis MM, Eger EI II.* Anesthesiology 1984; 61: 139–43.

2 *LeDez KM, Swartz J, Strong HA, Burrows FA, Lerman J.* Anesthesiology 1986; 65: A421.

3 *Lerman J, Willis MM, Gregory GA, Eger EI II.* Anesthesiology 1983; 59: 554–8.

The early post-anaesthetic state is primarily light NREM sleep

R.L. Knill, C.A. Moote, M.I. Skinner, E.A. Rose, C. Lim

University of Western Ontario, London, Ontario

During recovery from general anaesthesia and after initial arousal, patients are drowsy and often appear to "sleep." However, the nature of this state of sleep is not known. This state could be important in predispsing to respiratory complications from residual anaesthetic[1] or in delaying "street fitness" in outpatients. The purposes of this study were to (1) determine the specific state of healthy patients during recovery from a standard general anaesthetic and (2) assess surgical and anaesthetic factors which might affect it.

Methods

We studied 10 physical status ASA I patients (6 M and 4 F, age 17–43 yrs, wt 57–76 kg, ht 157–180 cm) following anaesthesia for elective orthopaedic or abdominal surgery. All gave informed consent. Premedication was lorazepam 2 mg SL. Anaesthesia was induced with thiopentone and maintained with N_2O and isoflurane, supplemented with fentanyl. Doses were left to the discretion of the attending anaesthetist. Anaesthesia

TABLE Results (mean ± SE, n = 10)

	Segment (min)		
	0–20	*20–40*	*40–60*
State times (%)			
Sleep	43 ± 10	68 ± 8*	74 ± 7*
– NREM, 1	31 ± 7	42 ± 9	36 ± 10
– NREM, 2	11 ± 8	26 ± 11	38 ± 14*
Wakefulness	57 ± 10	32 ± 9*	26 ± 8*
Awakening (#)	2 ± 1	3 ± 1	3 ± 1
Morphine dose (mg)	3.5 ± 1.0	3.2 ± 0.9	1.2 ± 0.4*†

	Segment (min)		
	60–80	*80–100*	*100–120*
State times (%)			
Sleep	80 ± 5*	78 ± 7*	86 ± 4*
– NREM, 1	35 ± 7	34 ± 9	22 ± 8
– NREM, 2	45 ± 10*	45 ± 12*	64 ± 10*†‡
Wakefulness	20 ± 5*	22 ± 7*	14 ± 4*
Awakenings (#)	2 ± 1	2 ± 1	1 ± 1
Morphine dose (mg)	0.2 ± 0.2*†	0.2 ± 0.2*†	0*†

Different from *0–20, †20–40, ‡40–60 ($p < 0.05$).

times were 90–180 min. Pain during recovery was treated with morphine 1–2.5 mg IV p.r.n. Upon arousal and over the subsequent 2 hrs, the EEG (C4–A1) and electrooculogram were monitored. State was determined in each 30 sec epoch according to standard criteria and analyzed in 20 min segments. Relationships were sought between state times and the following: age, gender, type and duration of surgery, thiopentone dose, N_2O/isoflurane MAC·hrs (estimated) and morphine doses given in recovery.

Results

The dominant state in the monitored period was physiological sleep. The only stages of sleep were light non-rapid-eye-movement (NREM) 1 and 2; deeper NREM and REM sleep were never observed. Sleep was interrupted frequently with brief periods of wakefulness. Awakenings were usually spontaneous. Average sleep time in individual segments correlated directly with the mean cumulative dose of morphine ($r = 0.98$, $p < 0.05$). Overall sleep time, excluding the first 20 min segment, related directly to the induction dose of thiopentone (sleep time range 47–95 per cent, thiopentone dose range 4.4–6.8 mg·kg^{-1}, $r = 0.64$, $p < 0.05$). No other significant relationships were observed.

Discussion

The principal state following arousal from anaesthesia was light and fragmented NREM sleep. This state of sleep appeared to depend upon relief of pain, since the time of sleep in each segment related sensitively to the doses of morphine which had been given and these doses have little intrinsic hypnotic effect. The relative importance of residual thiopentone, N_2O and isoflurane in promoting this sleep is not clear, although the overall amount of sleep after the first 20 min did relate to the thiopentone dose. We conclude that following arousal from a thiopentone/N_2O/isoflurane anaesthetic and pain relief, the state is primarily light NREM sleep. The amount of sleep depends in part on the induction dose of thiopentone.

Reference

1 *Meeke RI, Knill RL, Skinner MI, Rose EA, Soderman RM.* Augmentation of morphine-induced respiratory depression by a subanaesthetic dose of isoflurane. Can J Anaesth 1987; 34: S88.

The effects of halothane on hippocampal theta rhythm in the freely moving rat

W.E. Code, S.H. Roth, B.H. Bland, L. Strunin
Departments of Anaesthesia, Psychology and Pharmacology and Therapeutics, University of Calgary, Calgary, Alberta

Theta rhythm is a characteristic encephalographic (EEG) recording consisting of two subgroups – Type 1 (7–12 Hz) and Type 2 (3–9 Hz).[1] Type 2 theta is cholinergically mediated, confirmed by blockade with atropine sulfate, and is produced by cholinergic agonists and physostigimine. While barbiturates can inhibit theta activity, it has been demonstrated that some anaesthetics such as ethyl ether and urethane can initiate type 2 theta activity. Type 1 theta is associated with voluntary movements and can be abolished with various anaesthetics.

To date, the effects of the currently popular volatile anaesthetics on theta EEG activity have not been studied in detail. Winters[2] proposed an anaesthetic-induced spectrum of excitation and depression in the central nervous system. The state of anaesthesia is therefore likely a net result of a series of different actions involving many sites and mechanisms within the brain. In this study we report the concentration-dependent effects of halothane on internal hippocampal theta EEG activity. Since the hippocampus is involved in memory, awareness, behaviour and sensory input, many features which may be relevant and significant to the state of anaesthesia, it provides an ideal site for the study of the action of anaesthetics. Furthermore, the hippocampus is well characterized morphologically and electrophysiologically. Recent *in vitro* studies have demonstrated that anaesthetics alter the electrical membrane properties of hippocampus neurons. Thus the hippocampus is an appropriate model for an *in vivo* study of anaesthetic action and mechanisms.

Methods

Anaesthetized rats of 250–350 grams are stereotactically implanted with hippocampal electrodes seven to ten days before chronic recordings. During the experiments, the animals are connected via a liquid and electrical swivel commutator while inside a specially designed plexiglass chamber. The chamber permits continuous visual observation of the animals and controlled atmospheric concentrations of halothane. Electrode placement is confirmed histologically following sacrifice.

TABLE The effects of halothane on hippocampal theta EEG activity

	Mean Hz	*Range Hz*
Control movement	6.39 (0.29)*	5.5–7.3
0.5 per cent halothane movement	6.75 (0.58)	6.0–9.3
1.0 per cent halothane immobile	5.62 (0.38)	4.5–7.0
1.5 per cent halothane immobile	4.82 (0.27)	4.0–5.8
2.0 per cent halothane immobile	4.73 (0.68)	3.0–6.0

*Standard deviation of the mean.

Results

It was observed that halothane at concentrations of one to two MAC (minimum anaesthetic concentration) induced a theta EEG rhythm in the absence of any movement. The mean frequencies of the recorded theta hippocampal EEG activity in the absence (control) and presence of halothane are shown in the the Table. In the control situation, theta activity was recorded only with movement. Administration of halothane induced theta activity during immobility and may have enhanced activity (at 0.5 per cent) during movement. This halothane-induced theta activity was blocked with atropine sulfate.

Discussion

These results demonstrate that halothane can induce hippocampal theta rhythm, and suggest that anaesthetics may cause increased neuronal activity rather than depression. These dose-dependent data support the concept of a continuum of anaesthetic-induced states of excitation and depression, as proposed by Winters.[2]

References

1 *Bland BH*. The physiology and pharmacology of hippocampal formation theta rhythms. Progress in Neurobiology. 1986; 26: 1–54.

2 *Winters WD*. A review of the continuum of drug-induced states of excitation and depression. Progress in Drug Research 1982; 26: 225–58.

Evoked potential monitoring during posterior fossa aneurysm surgery: comparison of two modalities

P.H. Manninen, A. Lam, W.E. Nantau
University of Western Ontario, University Hospital, London, Ontario

Evoked potential (EP) monitoring is used during posterior fossa surgery to detect brainstem ischaemia, particularly during the temporary or permanent occlusion of a major feeding artery.[1,2] The purpose of this study was to compare two different EP modalities of monitoring, somatosensory evoked potentials (SSEP) and brainstem auditory evoked potentials (BAEP) during posterior fossa aneurysm surgery.

Methods

After institutional approval, 20 patients undergoing posterior fossa cerebral aneurysm surgery were monitored with SSEP and BAEP. Anaesthesia was induced with thiopentone, fentanyl or sufentanil, lidocaine and succinylcholine. Maintenance of anaesthesia consisted of 50 per cent nitrous oxide/50 per cent oxygen or air/oxygen and isoflurane. Induced hypotension was used in some patients during the dissection of the aneurysm but whenever temporary occlusion of the artery was performed, blood pressure was returned to normal. SSEP were recorded from the somatosensory cortex (C3′ and C4′), and C7 or Erbs point in response to median nerve stimulation with a square wave at 15 mamp. BAEP were recorded from the vertex in reference to the ipsilateal earlobe with monaural alternating clicks using an intensity of 60 DB above threshold delivered at a rate of 21.1 Hz. Bilateral, control SSEP and BAEP were recorded after the induction of anaesthesia and during stable anaesthesia. Throughout periods of dissection, temporary occlusion of the feeding artery and clipping of the aneurysm, both SSEP and BAEP were continuously monitored. SSEP recordings were analyzed using both the amplitude of the cortical peak (N20) and the central conduction time (CCT) (N20-N13). The BAEP were analyzed by measuring the peak V latency and amplitude along with the interpeak latency (V-III). All changes in EP were correlated to postoperative neurological deficits.

Results

Satisfactory recordings were obtained in all 20 patients. The mean age (±SD) was 52 ± 12 years (8 males and 12 females). Seventeen patients presented with a basilar artery aneurysm and three with a vertebral artery aneurysm. Temporary occlusion of the feeding artery was used in 14 patients. Six patients had a permanent occlusion of either the basilar or the vertebral artery. The significant findings are shown in the Table. The SSEP changes considered clinically significant were a decrease in amplitude >50 per cent of N20 and/or an increase in CCT > 1 msec. The significant change in BAEP was an increase in latency >1 msec of peak V. These changes had to be unilateral. Bilateral changes were considered to be due to the effects of anaesthetic drugs or physiological factors. Reversible EP changes occurred during temporary occlusion or retraction and recovered after manipulation, whereas a persistent change remained to the end of the procedure. Of 14 patients with no change in either SSEP or BAEP, one patient had a neurological

TABLE Results

Groups	*Reversible change*	*Persistent change*	*Neuro deficit*
No EP change (n = 14)	—	—	1
SSEP change (n = 4)			
↓ amplitude	0	2	2
↑ CCT	1	0	1
↓ amplitude + ↑ CCT	1	0	1
BAEP change (n = 1)			
↑ latency	0	1	1
SSEP & BAEP change (n = 1)	0	1	1
Total (n = 20)	2	4	7

deficit in the recovery room. This was a short perod of hemiparesis which improved completely within one hour. All EP changes occurring upon retraction of the brainstem (three patients) or during temorary occlusion (three patients) resulted in postoperative neurological deficits. Five patients with deficits (83 per cent) had changes in SSEP while one patient had changes in BAEP only. The duration of temporary occlusion varied from 0.5 to 13 minutes and primarily consisted of multiple occlusions of short duration.

Discussion

Monitoring of both SSEP and BAEP during posterior fossa surgery is feasible and useful especially when temporary or permanent occlusion of the vertebral-basilar arterial system is required. A false negative result may occur if the ischaemic zone is not directly monitored by the neuropathways of the SSEP and BAEP or the event occurs after the cessation of monitoring. The changes that occurred with SSEP monitoring correlated well with postopertive neurological deficits. In only one patient was there a change in BAEP as well as in SSEP. Though the numbers are small in this series, we conclude that SSEP monitoring may be a better indicator of brainstem ischaemia than BAEP during posterior fossa aneurysm surgery.

References

1 *Little JR, Lesser RP, Luders H.* Electrophysiological monitoring during basilar aneurysm operation. Neurosurgery 1987; 20: 421–7.

2 *Lam AM, Keane JF, Manninen PH.* Monitoring of brainstem auditory evoked potentials during basilar artery occlusion in man. Br J Anaesth 1985; 57: 924–8.

Thyroid function and haemodynamic stability after brain death

K.M. Robertson, I.M. Hramiak, A.W. Gelb
Departments of Anesthesiology and Endocrinology, University Hospital, London, Ontario

Hormonal replacement therapy is a new concept in the perioperative management of the heart-beating cadaveric donor. The objective of this study was to examine haemodynamic and endocrine changes in potential organ donors in an attempt to substantiate previous reports of low free T3 and T4 levels associated with haemodynamic instability.

Methods

Thirteen consecutive potential organ donors have been studied. All patients were initially treated with conventional cerebral resuscitative measures. At the time of brain death determination, cardiorespiratory and other essential organ functions were sustained with mechanical support and appropriate measures including intravenous fluid and/or inotropic support. During the interval between declaration of brain death and arrival in the OR for organ procurement, blood samples were taken for the following measurements: free T3, total T3, T4, T-up, FTI,TSH, insulin, blood sugar, cortisol, arterial blood gases, serum electrolytes, CBC and PT/PTT. The haemodynamic profile and the amount of inotropic support at that time were noted.

TABLE Thyroid function after brain death

	Free T3	*Total T3*	*T4*	*FTI*	*T-Up*	*TSH*
Normal	1	3	10	12	10	13
Reduced	12	10	2	1	3	0

Results

There were six men and seven women with a mean age of 35.7 yr. The cause of death was intracranial haemorrhage in nine patients, gunshot wound to the head in two patients, anoxic encephalopathy in one and blocked VP shunt in one. Thyroid function is reported in the Table.

Nine of thirteen donors required inotropic support which was commenced in the ICU and continued intraoperatively to maintain a SBP greater than 100 mmHg or MAP greater than 70 mmHg. Eight of 13 received dopamine at greater than 10 $\mu g \cdot kg^{-1} \cdot min^{-1}$, with or without levophed (two), epinephrine (one), or phenylephrine (two). Eight of 13 were treated with pitressin or DDAVP for diabetes insipidus. Seven of these required inotropic support, while only two of the five without DI required inotropes. No inotropic support was required in the 4/12 with low free T3.

Discussion

Recently it has been proposed that brain death may result in anterior pituitary axis endocrine abnormalities leading to functional instability in many organs from intracellular depletion or abnormal mitochondrial regeneration of ATP. In an experimental study of brain death in baboons, Novitsky *et al.* found a progressive reduction in circulating catecholamines, cortisol, insulin, and thyroid hormones. A similar reduction in free T3 and T4 was found in human organ donors. A requirement for increased mean dosage and duration of inotropic support to maintain adequate arterial blood pressures has been reported in recipients receiving hearts from thyroid hormone (T3 and T4) depleted donors.

Our results substantiate the reduction in free T3, but the reduction in T4 was inconsistent in our patients. A low free T3 did not predict the need for inotropic support. Hormonal therapy may offset the natural course of deterioration of the brain dead patient but the clinical significance of these endocrine changes still needs evaluation.

References

1 *D. Novitzky.* Transplantation 1987; 43: 852.

2 *D. Novitzky.* J Heart Transplantation 1984; 4: 63.

The effect of pentobarbitone anaesthesia on glucose metabolism in traumatized rat brain

M.G. Elphinstone, D.P. Archer, H.M. Pappius
Montréal Neurological Institute, Montréal, Québec

Investigations of the effects of cold injury on cerebral metabolism in rat brain have revealed widespread depression of local

TABLE Cortical LCGU in the lesioned (left) and unlesioned hemisphere (mean ± SD)

	Controls			
	(n = 6)		*p*	
Region	*1 Left*	*2 Right*	*1 vs 2*	*1 vs 3*
Visual	66 ± 16	98 ± 20	0.002	0.02
Auditory	72 ± 21	129 ± 23	0.001	0.05
Parietal	74 ± 15	98 ± 14	0.005	0.001
Sensorimotor	60 ± 14	96 ± 15	0.02	0.001
Frontal	60 ± 15	98 ± 16	0.01	0.001
	Pentobarbitone-treated			
	(n = 6)		*p*	
Region	*3 Left*	*4 Right*	*2 vs 4*	*3 vs 4*
Visual	35 ± 11	43 ± 12	0.001	0.10
Auditory	40 ± 14	50 ± 16	0.001	0.05
Parietal	30 ± 6	41 ± 8	0.001	0.02
Sensorimotor	31 ± 7	50 ± 9	0.001	0.02
Frontal	28 ± 4	39 ± 6	0.001	0.05

cerebral glucose utilization (LCGU) which varies among brain regions.[1] The metabolic depression is most profound in cortical regions ipsilateral to the cold injury, and shows a time variation, being worst three days after the cold lesion is made. Since the effects of barbiturates on cerebral blood flow and intracranial pressure are thought to derive in part from reduction of cerebral metabolism, we have studied the effect of pentobarbital administration on LCGU in cold-injured rat brain using quantitative radioautographic techniques.

Methods

Under general anaesthesia with halothane (two per cent) in oxygen, standardized superficial freezing lesions were made in the left parietal region of male Sprague-Dawley rats (250–300 g) as previously described.[1] Three days later (^{14}C) deoxyglucose (DG) studies[2] were performed on the fasted rats. Vascular catheters were inserted and a plaster cast (from the waist down) was loosely applied under halothane anaesthesia. The animals were then allowed to recover for 3 hr. Blood pressure, rectal temperature, haematocrit and blood gases were monitored in each animal; temperature was maintained with a heating lamp. All animals had freezing lesions. Control animals were conscious during the DG determination while the treated animals received intermittant boluses of intravenous pentobarbitone sufficient to abolish the response to a tail clamp. Treated animals maintained spontaneous ventilation during the DG study. The LCGU experiment was begun by intravenous injection of a bolus of 30 Ci of (^{14}C) DG over 30 sec. Timed arterial sampling of blood radioactivity and glucose levels was performed until decapitation 45 minutes after the start of the experiment. Serial coronal radioautographs were prepared from forzen sections of the brain. Densitometry was performed on 30 specific brain structures in each animal and LCGU calculations performed using the equations of Sokoloff.[2] Cortical LCGU values were analyzed using analysis of variance; individual group differences were isolated by t test. Statistical significance was defined as $p < 0.05$.

Results

As expected, mean values of blood pressure and pH were lower (13.3 vs 17.7 kPa; 7.39 vs 7.41) and $PaCO_2$ higher (6.1 vs 5.3 kPa) in the treated group than in the controls. Temperature and haematocrit were not different in the two groups. The Table summarizes the mean cortical LCGU results, which demonstrate that pentobarbital significantly reduced cortical LCGU in both the lesioned and the unlesioned hemisphere.

Discussion

Pentobarbitone reduced LCGU in cortical regions of both the injured and uninjured hemispheres. The reduction of LCGU was less in absolute terms in the cortical regions ipsilateral to the lesion than on the contralateral side and was proportional to the control LCGU value ($p < 0.001$, calculation not shown).

References

1 *Pappius HM.* Local cerebral glucose utilization in thermally traumatized rat brain Ann Neurol 1981; 9: 484–91.

2 *Sokiloff L, Reivich M, Kennedy C et al.* The (^{14}C)-deoxyglucose method for measurement of local cerebral glucose utilization: theory, procedure and normal values in the conscious and anesthetized albino rat. J Neurochem 1977; 29: 13–26.

Does increased FiO_2 alter NADH redox state and protect brain cells from ICP changes in rabbits?

B. Bissonnette, P. Bickler, G.A. Gregory, J.W. Severinghaus
Department of Anesthesia and the Cardiovascular Research Institute, University of California, San Francisco, San Francisco, California

Elevated intracranial pressure (ICP) is an important pathophysiologic feature of head injury, post-ischaemic reperfusion, and a variety of disease states including cerebral oedema, infection and haemorrhage. Reduced blood flow to the brain and brain stem and imbalance between oxygen supply and demand are viewed as key mechanisms of injury from elevated ICP. Despite the importance of these mechanisms, there is little information on the relationship of intracranial pressure to brain oxygenation. Therefore, we designed this study to examine the effect of progressive changes in ICP on cerebrocortical oxygenation and cerebral blood volume, and also to determine if increased inspired oxygen concentration protects brain cells against these intracranial pressure changes.

Methods

The study was approved by the Animal Care Committee. We continuously recorded systemic arterial pressure, central venous pressure (CVP) and ICP in six male rabbits, weighing 2.8–3.5 kg, who were anaesthetized with urethane and paralyzed with pancuronium. Body temperature was maintained at 39°C by a servocontrolled heat lamp. All animals were mechanically

ventilated to maintain normocarbia. FiO_2 was varied between 0.21 and 1.0. End–tidal gas samples were analyzed by mass spectrometer. The head was fixed in a stereostatic apparatus and two burr holes were drilled. The dura mater was left intact. ICP was measured from an epidural bolt and also from a 22-gauge short lumbar puncture needle positioned in the cisterna magna. The 22-gauge needle was connected to a Y-connector to allow injection of artificial cerebrospinal fluid to increase ICP. The ICP bolt was sealed around the burr hole to avoid any pressure leak. Cerebrocortical NADH fluorescence and ultraviolet reflectance were measured by a microfluororeflectometer;[1,2] the NAD/NADH redox state and relative cerebrocortical blood volume were determined from these measurements. NADH fluorescence measurement *in vivo* is based on the fact that only the reduced nicotine-amide-adenoside dinucleotide fluoresce when it is illuminated at 366 nm; therefore, oxygen decreases fluorescence because it oxidizes NADH. Fluorescence during 100 per cent O_2 and normocarbia was used to determine the baseline NADH; then any change in fluorescence represented change in relative NADH. Cerebrocortical oxygenation was estimated by the measured change in relative NADH. Relative vascular volume (i.e., capillary blood volume) was estimated by the measured change in reflected light (the sum of reflected and scattered light) at 366 nm. The data were analyzed by multiple analysis of variance and a $p < 0.05$ was considered statistically significant.

Results

In all six animals, NADH increased when ICP was greater than 18 ± 2.2 cm H_2O. Above this threshold, the change in NADH was inversely proportional to the change in FiO_2 over the range from 0.21 to 0.5 ($p < 0.05$). However, raising the FiO_2 above 0.5 gave little additional benefit in cerebrocortical oxgenation (i.e., cerebrocortical oxygenation at FiO_2 at 1.0 was not statistically better than at 0.5) (Figure). Capillary blood volume, which is proportionate to cerebrocortical blood volume increased as ICP increased until the ICP exceeded 30 ± 1.8 cm H_2O and then capillary blood volume decreased. This suggests that either the pressure applied by the tip of the fluorometer probe was great enough to mechanically decrease capillary blood flow at the highest ICP pressures, or the obstruction to blood flow produced by increased ICP was not only applied to the venous circulation, but also to the arterial supply.

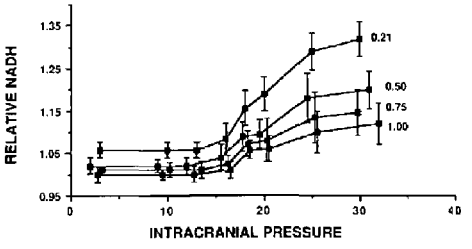


FIGURE Relative NADH at FiO_2 0.21 to 1.0.

Discussion

These results indicate that high ICP definitely decreases cellular oxygen supply. Moreover, another indication of this decrease in cellular oxygenation is the measured increase in cerebrocortical blood volume; this increase in blood volume is acutely determined by an increase in cerebrocortical blood flow in response to hypoxia. Others have shown that this increase in cerebrocortical blood flow produces improved cellular oxygenation and that this is a cellular defense mechanism. In summary, we showed that with elevated ICP: (1) Increased oxygenation of blood gives greater cellular protection against changes in ICP and (2) little additional benefit to cerebrocortical oxygenation is achieved by increasing FiO_2 above 0.5. This should be an important consideration in neurological intensive care units where it has been common practice to increase the FiO_2 to any patient with high ICP even though oxygen toxicity may occur. Our data support that giving an FiO_2 of greater than 0.5 will not improve cerebrocortical oxygenation and may, in fact, damage other organs.

References

1 *Chance B, Legasllais V.* A spectrofluorometer for recording of intracellular oxidation-reduction states. IEEE Trans Biomed Eng, April 1963.

2 *Jobsis FF, O'Connor MJ, Vitale A, Vreman H.* Intracellular redox changes in functioning cerebral cortex. J Neurophysiol 1971; 34: 735–49.

Ergonomic approach to treatment of patient's perioperative stress

R. Spintge, R. Droh

Anaesthesiology, Sportverletzte Hellersen, Lüdenscheid, Germany

The patient in anaesthesia suffers from a certain psychic and somatic distress while waiting for treatment and during treatment. This distress is independent of the actual disease. It always means significant irritation of the whole organism. The main reasons are *anxiety* and *pain*. Some of the most important pathophysiological implications are:

- arrythmias, angina pectoris, hypertension, hyperventilation and asthma
- lowered pain tolerance and general hyperaesthesis
- increased muscle tonus and excitement
- increased plasma levels of catecholamines, steroid hormones, endogenous opioids
- impaired subjective feeling with inadequate defence reactions and reduced compliance

The above lead to an increased demand for anxiolytic and analgesic drugs.[1] It has become clear that pharmaceutical and psychological means alone are not sufficient to prevent these undesired reactions. Psychologically the patient is in a situation of regression. This regression needs some form of a non-verbal communication.[2] We examined whether an ergonomic design of the patient's environment could be of benefit.

Methods

During the past 12 years our research team conducted several clinically controlled randomized studies of the effects of acoustical ergonomic measures in the patient's perioperative environment. About 8,000 patients were studied and the clinical follow-up of 62,000 patients was recorded and evaluated. Age ranged from 10 to 95 years; 60 per cent male, music groups and control groups equally randomized in socioeconomic parameters. Five categories of music could be chosen (frequency of choice by 62,000 patients in per cent in brackets): popular music (44 per cent), soft popular music (six per cent), classics (15 per cent), military music (ten per cent), actual pop hits (25 per cent). Upon entering the operation area the patients received earphones with the chosen music. Earphones were kept on until sleep in general anaesthesia or until the end of operation in regional anaesthesia. Pre- and postoperative questionnaires, interviews, state-trait-anxiety-inventory (STAI), mean arterial blood pressure (MABP), heart rate, plasma levels of stress hormones (cortisol, ACTH, prolactin, beta endorphin, HGH, antidiuretic hormone ADH, adrenaline, noradrenaline), were obtained perioperatively (Figures 1, 2).

Results

In all studies the music groups showed significantly better outcome in psychological, behavioral and physiological parameters (for details see references 3–7).

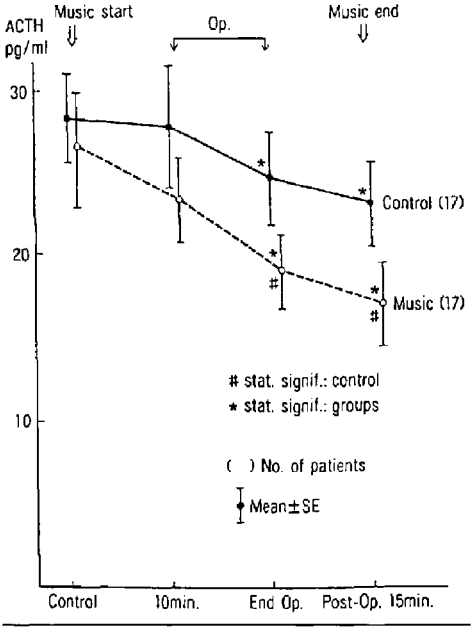


FIGURE 1 Effect of anxiolytic music on plasma ACTH levels in dental procedure.

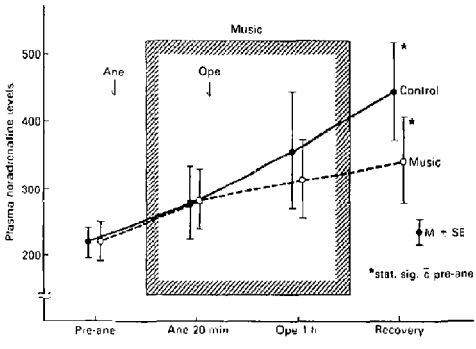


FIGURE 2 Effect of music on plasma levels of noradrenaline.

TABLE Physiological effects of anxiolytic music

System	*Reaction*
Circulation	decrease of heart-rate and blood pressure, anti-arrhythmic effect
Respiration	decrease of respiratory minute volume and O_2 consumption
Metabolism	decreased release of: cathecholamines, ACTH, cortisol, prolactin, β-endorphin

Figure 1 shows plasma levels of adrenocorticotrophic hormone (ACTH) during dental treatment with and without anxiolytic music.[4] Figure 2 shows plasma levels of noradrenaline during orthopaedic surgery with epidural anaesthesia and anxiolytic music.[5] The Table summarizes the psychophysiological results of all studies.

Discussion

When using psychophysiological measures in medicine, one has to follow the same standards as for clinical drug evaluation and monitoring. Thus, standardized and reproducible ways of application, of control of effects and of side-effects were used. Following such a strict regime acoustic ergonomic measures like the application of anxiolytic music via headphones were shown to create valid and beneficial stress-reduction in patients receiving anaesthetics.

References

1 *Tolksdorf W.* Der perioperative Stress. Springer Berlin-Heidelberg-New York, 1986.
2 *Spintge R.* Psychologische and psychotherapeutische Maßnahmen zur Bekämpfung perioperativer Angst. Medical Doctoral Dissertation University of Bonn 1982.
3 *Kamin A. Kamin HP, Spintge R, Droh R.* Endokrinologische Wirksamkeit anxiolytischer Musik und psychologischer Operationsvorbereitung. *In*: (7), pp. 157–168, 1987.
4 *Oyama T, Hatano K, Sato Y, Kudo M, Spintge R, Droh R.* Endocrine effect of anxiolytic music in dental patients. *In*: (7), pp. 223–226, 1987.

5 *Tanioka F, Takazawa T, Kamata S, Kudo M, Matsuki A, Oyama T.* Hormonal effect of anxiolytic music in patients during surgical operations under epidural anaesthesia. *In*: (7), pp. 199–204, 1987.
6 *Halpaap BB.* Musik als Anxiolytikum in der Geburtshilfe. Medical Doctoral Dissertation Free University of Berlin 1988.
7 *Spintge R, Droh R.* Music in Medicine. Springer Berlin-Heidelberg-New York 1987.

Postoperative recovery after cholecystectomy

F. Chung, A. Chung, A.E. Taylor, R.M. Stone, H.M.R. Meier, E. Lautenschlaeger, C. Seyone
Departments of Anaesthesia, Surgery, Psychology and Psychiatry, Toronto Western Hospital, University of Toronto, Toronto, Ontario

Postoperative confusional states and delay in recovery could lead to increased morbidity, mortality and health care costs. There is minimal work in the literature assessing the recovery of mental function after surgery.[1,2] No impairment of mental ability two weeks after general surgery has been reported.[1] However, mental deterioration has also been observed a few weeks later.[2] The purpose of this study is designed to find out the incidence of cognitive impairment after cholecystectomy and its correlation with age.

Methods

All patients undergoing elective cholecystectomy who gave informed consent were included. They were divided into two groups, ≥60 years old, and <60 years old. Exclusion criteria were senile dementia, psychiatric history, neuromuscular incoordination, aphasia and visual impairment. Mental function tests were done preoperatively, first day, second day, third day, and one month later. The tests used were the Symbol Digit Modalities test, The Trail Making test, Digit Span and Mini-Mental State test. Group differences of the mental function were studied on a univariate basis using Student's t test. A repeated measures analysis of variance was carried out between the two age groups to assess the significance of group difference, and change over time. Contrast was specified to test differences at each postoperative time relative to preoperative.

Results

Demographic data is shown in the Table. The mean age of patients ≥60 was 67.4 ± 1.9, and the younger group, 40.3 ± 2.8. There was no difference in sex and duration of surgery. The

TABLE Demographic data

	Δ60 (n = 9)	*<60 (n − 12)*
Age (mean ± SE)	67.4 ± 1.9	40.3 ± 2.8
Sex	2 M: 7 F	6 M: 6 F
ASA class	I II III	I II III*
	2 6 1	9 3 0
Duration of surgery	111.7 ± 10.0	109.6 ± 8.4

*p < 0.05.

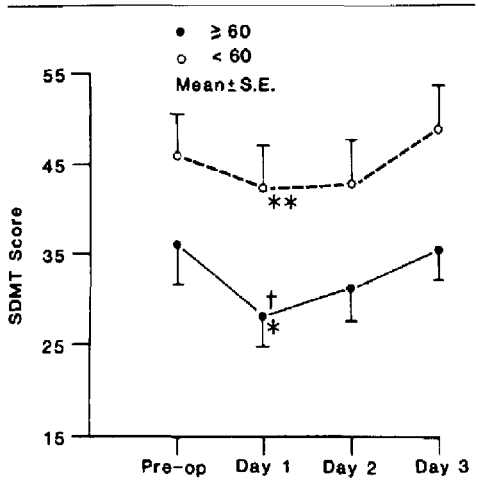


FIGURE 1 Symbol Digit Modalities test. *p < 0.005. **p < 0.03 within group. †p < 0.02 between groups.

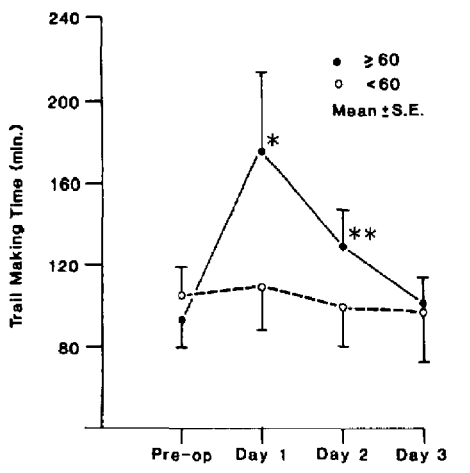


FIGURE 2 Trail Making test. *p < 0.04. **p < 0.02 within group.

Symbol Digit Modalities tested the ability of the patient to compare as many of the 110 items as possible in 90 seconds. There was a significant difference in the score between day one and preoperative score in the elderly, p < 0.005, and in the patients <60, p < 0.03. Comparison between the two groups showed there was a more significant decline in the score in the elderly at day one, p < 0.02 (Figure 1). The Trail Making test was scored by noting the time to connect the same number of consecutively numbered and lettered circles alternately on one worksheet. There was a significant prolongation in the time

required to complete the Trail Making test in the elderly at day one, $p < 0.04$ and at day two, $p < 0.02$. Postoperatively, there was no prolongation in <60 group (Figure 2). There was no significant change in the results of the Digit Span and Mini-Mental State tests between groups and within groups.

Discussion

The Symbol Digit Modalities test showed that both the elderly and the younger patients had a statistically significant decrease in score on day one, with a more marked drop in the elderly group. The Trail Making test showed that the elderly had deterioration on day one and day two post-op. The scores on all patients returned to normal on day three.

References

1 *Simpson BR, William M, Scott JF, Crompton Smith A.* The effect of anaesthesia and elective surgery on old people. Lancet 1961; ii 887–93.

2 *Blundell EA.* A psychological study on the effects of surgery on eighty-six elderly patients. Br J Soc Clin Psychology 1967; 6: 297.

Intraoperative mortality in patients with blunt chest trauma

J.H. Devitt, R.F. McLean

Department of Anaesthesia, Sunnybrook Medical Centre, University of Toronto, Toronto, Ontario

Polytraumatized patients requiring emergency surgery are a great challenge to the anaesthetist. Most studies include penetrating and blunt trauma together. No series to date has reviewed blunt trauma in isolation.[1]

Methods

The charts of all blunt polytraumatized patients requiring emergent surgery with severe injuries in two or more body systems admitted to a regional trauma unit between July 1, 1985 and June 30, 1986 were reviewed retrospectively (Injury Severity Score >17).[2] Patients were divided into two groups; Group I polytraumatized with blunt thoracic trauma and Group II polytraumatized without blunt thoracic trauma. Blunt thoracic trauma was defined as any injury described by the Abbreviated Injury Scale of two or greater for the chest region including single rib fractures.[3] Injury severity scores (ISS) were calculated by an individual unaware of the nature of the study. Both groups were compared with respect to mortality, mean ISS and mean age.

Results

One hundred and sixty-seven patients had an ISS of greater than 17. One chart could not be found. Ninety patients (Group I) had significant thoracic trauma requiring an emergent operative procedure, while 76 patients without chest trauma required emergent surgery (Group II). Groups I and II were similar with respect to age but not severity of injury (Table).

There were eight intraoperative deaths in Group I while only one patient in Group II died intraoperatively. All nine patients died of exsanguination. The greater death rate in the Group I patients was statistically significant ($p = 0.031$, 1-tailed Fisher's exact test). The source of haemorrhage in the Group I patients included abdominal cavity (4), intrathoracic inferior vena cava (2), chest wall (1) and head (1). The source of haemorrhage in the Group II patients was the pelvis. Four patients in Group I presented to the operating room with an incidental thoracic aortic intimal tears.

TABLE Mean age and ISS of Groups I and II

	Group I	*Group II*
Age (±SD)	37.6 ± 19.9	32.3 ± 15.9 NS
ISS (±SD)	34.8 ± 10.6	29.4 ± 8.3 $p < 0.001$ t test

Discussion

All intraoperative deaths were due to exsanguination, regardless of the presence or absence of chest injury. However, patients with blunt thoracic trauma had a significantly higher intraoperative death rate. The presence of blunt chest trauma should be a marker of the severity of the injury sustained by the patient.

Four patients with injuries to the thoracic aorta presented to the operating room for urgent surgical procedures other than vascular repair yet none of these patients died or had significant bleeding from their vascular injuries intraoperatively. The thoracic aortic injuries were repaired at a later date. In our series the presence of a real or suspected injury to the thoracic aorta did not contra-indicate emergency surgery to deal with other life or limb threatening injuries.

All polytraumatized patients brought to the operating room on an emergent basis with any evidence of thoracic injury should have adequate venous access for rapid infusion of fluids or blood products. We also advocate the use of an indwelling arterial catheter for ongoing blood pressure measurement, freeing hands for the resuscitation.

References

1 *Rodriques R, Herrin TJ, Hendrickson M.* Cardiac and thoracic vascular injuries: anesthetic considerations. South Med J 1980; 74: 739–41.

2 *Baker SP, O'Neil B, Haddon W et al.* The injury severity score: a method for describing patients with multiple injuries and evaluating emergency care. J Trauma 1974; 14: 187–96.

3 American Medical Association's Committee on the Medical Aspects of Automotive Safety; Rating the Severity of Tissue Damage: The Abbreviated Scale. JAMA 1971; 215: 277.

Prevention of fat and marrow microembolism during cemented arthroplasty

R.J. Byrick, J.B.M. Mullen, R.S. Bell, D. Forbes, J.C. Kay

St. Michael's Hospital, University of Toronto, Toronto, Ontario

Pulmonary microembolism of fat and marrow contents have been demonstrated after insertion of cemented prostheses. Pres-

surization of the reamed contents of the intramedullary canal results in "intravasation" of particulate microemboli. We have assessed the impact of two techniques of intramedullary cavity lavage in the prevention of fat and marrow microembolism in an anaesthetized dog model.

Methods

Twenty-eight mongrel dogs were studied. Arterial blood pressure (BP), pulmonary artery (PA) and left atrial (LA) pressures were monitored continuously. Cardiac output ($\dot{Q}$), intrapulmonary shunt fraction (Qs/Qt), and physiologic deadspace to tidal volume (V_D/V_T) ratio were measured prior to bilateral cemented arthroplasty (BCA) and at timed intervals after BCA.

In one group (NL) (n = 9) two femora were exposed, and reamed prior to control measurements, with no lavage. In the second group (ML) (n = 9), 100–200 ml of saline was flushed by syringe through a 12 cm catheter into the distal intramedullary cavity. In the third group (PL) (n = 10) a commercially available pulsatile lavage technique was used to flush each reamed intramedullary cavity with 1.5 L of sterile saline, combined with intermittant suction of cavity contents. BCA was then performed in the same manner in all three groups.

All animals were killed 90 minutes after BCA. The lungs were excised, fixed in inflation, and three blocks of tissue taken from the mid-sagittal slice of each lung. Morphometric measurements were performed on six samples from each dog, 12 fields per sample, for a total lung area of 72 mm^2 per animal.

Results

No significant changes in BP, LAP or RAP were noted in any group after BCA. Significant increases in PAP were noted in the NL and the ML groups (Table), together with a significant decrease in PaO_2 and increase in Qs/Qt. $\dot{Q}$ decreased significantly only in the NL group.

Histological examination revealed significantly fewer fat microemboli in the PL group (121 ± 52) than in either the NL group (471 ± 224) or the ML group (307 ± 155) per 72 mm^2.

TABLE Cardiorespiratory data (mean + SD) before and 15 minutes post-BCA

	NL		*ML*	
	Control	*Post-BCA*	*Control*	*Post-BCA*
PAP (mmHg)	14.9±3.6	22.4±2.5*	16.0±1.9	23.6±2.8*
PaO_2 (mmHg)	95.0±10.7	78.9±11.2*	93.6±10.9	79.3±13.1*
Qs/Qt	28.2±5.1	33.3±8.1*	22.7±7.1	29.7±8.7*

	PL	
	Control	*Post-BCA*
PAP (mmHg)	17.6±4.2	20.0±4.6
PaO_2 (mmHg)	99.4±10.6	92.6±11.7
Qs/Qt	19.6±6.4	20.5±8.3

*Denotes significant change from control using two-way analysis of variance and Dunnett's multiple range test with significance level of 0.05.

Discussion

In this BCA model, large volume, high-pressure pulsatile lavage significantly reduced physiologic derangements which are characterized by elevated PAP, reduced PaO_2 and elevated Qs/Qt. Low-volume, low-pressure manual lavage did not prevent either physiologic or pathologic evidence of microembolism. These data show that the lavage technique after reaming in this model determines the number of fat and marrow emboli. In this model, pulsatile lavage is much more effective clearing and preventing fat microembolism than the manual technique used. These data also suggest that cardiopulmonary changes after BCA are primarily due to the effects of microembolism and not methylmethacrylate monomer, as the same volume and concentration of cement was used in all three groups.

What is the impact of clinical research on anaesthetic practice?

J. Swartz, D.W. Fear, J. Lerman
Department of Anaesthesia and the Research Institute, The Hospital for Sick Children, University of Toronto, Toronto, Ontario

Many clinical trials are published in the anaesthetic literature with the aim of improving the quality of patient care.[1] We have little information, however, about the impact of these trials on clinical practice. The purpose of this study was two-fold: (1) to examine the impact of a published clinical trial on the day-to-day practice of staff anaesthetists in the department in which the study was performed, and (2) to correlate the above information with an in-house chart review.

Methods

"Effect of droperidol pretreatment on postanaesthetic vomiting in children undergoing strabismus surgery"[2] was selected as the reference publication for the following reasons: (1) the protocol was prospective and randomized, (2) learning a new technical skill was not required, and (3) a definite improvement in patient care was demonstrated.

Part 1

A questionnaire was anonymously completed by the 21 members of the Department of Anaesthesia. The questions were designed to determine the practice of each anaesthetist before and after publication of the reference paper.

Part 2

An in-house chart review of anaesthetic practice during strabismus surgery was performed. The charts of all children undergoing strabismus repair during two periods were reviewed: a four-month period three months before the study, and a four-month period three months after publication of the results.

Statistical significance ($p \leq 0.05$) was determined using Fischer exact test and Chi-square analysis with Yates correction.

TABLE I Questionnaire results

	Before publication	*After publication*
Droperidol used ≥50% cases	5/21 (23%)	16/21 (76%)*
Used recommended dose regimen	2/5 (40%)	9/16 (52%)
Practice changed by study	—	14/21 (67%)

*$p < 0.001$ compared to before publication.

TABLE II Chart review

	Charts	*Droperidol given*	*Recommended dosage (75 μg·kg⁻¹)*	*Average dose range (μg·kg⁻¹)*
Before	87	18 (20%)	11 (61%)	61 (10–75)
After	48	33 (68%)	13 (39%)*	56 (16–115)

*$p < 0.05$ compared to before publication.

Results

Part 1

There was 100 per cent response to the questionnaire (Table I). The reasons given for not using droperidol included delayed extubation, prolonged awakening, and possible ineffectiveness as an antiemetic.

Part 2

The charts of 135 patients were reviewed. Patients were included if they fulfilled the inclusion criteria of the reference publication. The charts were divided into two groups, before and after publication of the study results (Table II). In almost all cases after publication of the study, droperidol was given at induction as recommended.

Discussion

Sixty-seven per cent of anaesthetists agreed that they were favourably influenced by the droperidol study. Results of both questionnaires and chart reviews indicated that the use of droperidol increased significantly after publication of the study; however, approximately 50 per cent of those who changed their practice admitted that they did not follow the recommended dosage regimen. This observation was supported by the chart review (Table II). We conclude that the reference publication did affect clinical practice in this institution. Further studies are needed to determine the relationship between clinical trials and clinical practice in the anaesthetic community.

References

1 *Evans CE, Hayes RB, Gilbert JR et al.* Educational package on hypertension for primary care physicians. Can Med Assoc J 1984; 130: 719–22.

2 *Lerman J, Eustis S, Smith DR.* Effect of droperidol pretreatment on postanaesthetic vomiting in children undergoing strabismus surgery. Anesthesiology 1986; 65: 322–5.

General anaesthesia for Caesarean section – time intervals and maternal haemodynamics

J.F. Boylan, A.J. Cunningham, E. Carton, N.J. McDonald, W.P. Scarlett, J. Moriarty

Beaumont Hospital, Dublin, Ireland

There is still controversy about the role of operative delay in the genesis of neonatal depression following Caesarean section under general anaesthesia. Prolonged induction delivery[1] (IDI) and uterine incision-delivery[2] interval (UDI) have been implicated, and the relevance of IDI recently questioned.[3] We re-examined the association between IDI, UDI and Apgar score in elective and emergency patients. In the former group, maternal haemodynamic changes at induction of anaesthesia were also studied.

Methods

Sixty-four women were studied. Thirty-six were elective patients; the remainder were sectioned for failure to advance in established labour. All pregnancies were singleton, of at least 37 weeks gestation and without evidence of fetal growth retardation or distress. Each patient received preoperative sodium citrate, was pre-oxygenated for three minutes with left lateral tilt, and underwent rapid sequence induction of anaesthesia with thiopentone 4 $mg \cdot kg^{-1}$ and succinylcholine 1 $mg \cdot kg^{-1}$. After tracheal intubation under cricoid pressure, anaesthesia was maintained until delivery with 50 per cent nitrous oxide/oxygen/0.5 per cent halothane and pancuronium 0.07 $mg \cdot kg^{-1}$. One- and five-minute Apgar scores (A1, A5) were assigned by a neonatologist. Time from induction to delivery (IDI) and first uterine incision to delivery (UDI) were noted. Maternal heart rate and systolic/diastolic blood pressure (SBP, DBP) were recorded from ECG and electronic oscillotonometer, respectively, in elective patients. Data were analyzed using Student's t test and Kendall's tau (T) coefficient. P values less than 0.05 were deemed significant.

Results

Apart from maternal age (32.8 ± 6.6 vs 26.8 ± 5.4 yrs) and gestation (39.0 ± 1.3 vs 40.4 ± 1.4 wks) (mean ± SD, $p < 0.01$) demographic data did not differ between elective and emergency patients, respectively. Prolonged IDI and UDI were both associated with lowered A1 in the elective group ($p < 0.05$). In emergency patients, IDI alone was inversely associated with lowered A1 ($p < 0.01$). Elective patients delivered of depressed infants (A1 < 7) had significantly higher post-intubation SBP and DBP (Table).

TABLE Maternal BP post-intubation

	A1 < 7 (n = 26)	*A1 ≥ 7 (n = 9)*
SBP	162.56 ± 19.35*	147.23 ± 15.40
DBP	107.89 ± 12.09†	94.54 ± 12.25

Data are means ± SD; *$p < 0.05$; †$p < 0.01$.

Discussion

Our data confirm the impression that prolonged IDI and UDI are associated with neonatal depression at Caesarean section; the relative contribution of each remains uncertain. However, IDI is the sole prognostic indicator at emergency section. The association between lowered A1 and post-intubation hypertension has not been previously reported in healthy patients, and may reflect an alpha-adrenergically mediated response to stimuli under light anaesthesia. Despite recent advice to the contrary, rapid delivery remains a priority at all Caesarean sections.

References

1 *Datta S, Ostheimer GW, Weiss JB et al.* Neonatal effect of prolonged anesthetic induction for cesarean section. Obstet Gynecol 1981; 58: 331.

2 *Crawford JS, Burton M, Davies P.* Time and lateral tilt at caesarean section. Br J Anaesth 1972; 44: 477.

3 *Zagorzycki MT, Brinkman CR.* The effect of general and epidural anesthesia upon neonatal Apgar scores at repeat cesarean section. Surg Gynecol Obstet 1982; 155: 641.

Caesarean section with epidural carbonated lidocaine and fentanyl

M.J. Tessler, D.R. Biehl, M.A. Naughler
University of Manitoba, St. Boniface General Hospital, Winnipeg, Manitoba

Epidural blockade for Caesarean section requires large volumes of local anaesthetics to induce appropriate sensory blockade. The dose of local anaesthetic used can result in significant hypotension, yet still be inadequate for complete analgesia. Epidural narcotics have been used as adjunct to local anaesthetics to improve analgesia during various surgical procedures.[1] We therefore investigated the effects of adding fentanyl 50 μg to carbonated lidocaine for elective Caesarean section. We assessed maternal analgesia and neonatal well-being.

Methods

Informed consent was obtained from 45 ASA physical status I or II women scheduled for elective Caesarean section. The parturients were randomly divided into two groups. In Group A (23 patients) after a test dose of 2 ml of lidocaine, fentanyl 50 μg (1 ml) was added to the first 10 ml of carbonated lidocaine with epinephrine 1:200,000. This solution was injected into the epidural space via an epidural catheter inserted at L_{2-3} or L_{3-4}. Group B (22 patients) received normal saline (1 ml) added to the carbonated lidocaine administered in identical fashion. The total dose of carbonated lidocaine administered was determined by the clinical judgement of the individual anaesthetist.

After delivery and assessment of the newborn by the attending neonatologist, the neurologic and Adaptive Capacity Scoring System (NACS)[2] was performed at birth and 24 hours by a nurse specifically trained in this technique. A score of greater than 35 was taken as a sign of neonatal wellbeing. Neither the anaesthetist nor the nurse were aware of which group the patient belonged. A blood sample was drawn from the umbilical vein at delivery for analysis of fentanyl concentration.

TABLE Results

	Group A Fentanyl n = 22	*Group B Saline n = 22*	
Intravenous medication			
Ephedrine	8	8	
Fentanyl	0	8	$p < 0.05$
Diazepam	1	2	
Metoclopramide	3	3	
Promethazine	0	2	
Apgar score			
1 min 0–6	2	1	
7–10	20	21	
5 min 0–6	0	0	
7–10	22	22	
Evaluation of anaesthesia			
Patient			
1	0	0	
2	2	2	
3	4	7	
4	13	10	
Anaesthetist			
1	0	0	
2	2	1	
3	5	8	
4	14	10	
NACS at birth			
0–34	12	9	
35–40	10	13	

While the mother was recovering in the post-anaesthesia room, both she and her anaesthetist were asked to assess the anaesthetic for the procedure on a scale of 1 to 4 (1 = poor analgesia, 4 = excellent analgesia).

All drugs administered intraoperatively were recorded. Analysis of differences between groups was by unpaired t test or Chi square where appropriate.

Results

One patient in the fentanyl group was excluded from analysis because of inadequate sensory blockade leading to induction of general anaesthesia. One patient in the saline group also had inadequate anaesthesia but was induced after delivery so is included in the results.

The patients were similar in age, parity, gestational age and previous Caesarean sections. The IV medications, analgesia scores, Apgar and NACS scores are shown in the Table. There was significant difference in intravenous supplementation for analgesia in Group A. There was no significant difference in the Apgar scores, patient or anaesthetist evaluation of the anaesthetic, or NACS scores at birth or on day one. No neonate required naloxone for resuscitation. There was no case of maternal pruritis requiring medication.

Discussion

The significant decrease of intravenous supplementation required in Group A suggests that epidural anaesthesia was improved by the addition of fentanyl to the local anaesthetic. However, neither the patient nor the attending anaesthetist detected any significant overall differences in the quality of the block after the procedure. No adverse effects on the fetus were found. We conclude that fentanyl fentanyl 50 μg offers some benefit when administered in conjunction with carbonated lidocaine in the epidural space for elective Caesarean section.

References

1 *Gafford MP, Bansal P, Lawton C et al.* Surgical analgesia for caesarean delivery with epidural bupivacaine and fentanyl. Anesthesiology 1986; 65: 331–4.

2 *Amiel-Tison C, Barrier G, Shnider SM et al.* A new neurologic and adaptive scoring system for evaluating obstetric medications in full-term newborns. Anesthesiology 1982; 56: 340–50.

A comparison of lidocaine hydrochloride, lidocaine hydrocarbonate and pH-adjusted lidocaine hydrochloride for Caesarean section

D.J. Liepert, M.J. Douglas, G.H. McMorland, D.R. Gambling, J.H.K. Kim, P.L.E. Ross

Department of Anaesthesia, University of British Columbia and Grace Hospital, Vancouver, British Columbia

Local anaesthetics are generally marketed as the acidic salt in order to improve solubility. Raising the pH of the local anaesthetic by the addition of bicarbonate has been shown to shorten the onset time and time to peak effect, and to increase the duration of action and quality of the block.[1] DiFazio *et al.*[2] compared the commercial preparation of plain lidocaine and epinephrine with pH-adjusted commercially prepared lidocaine and epinephrine and confirmed these findings. In this study, two per cent lidocaine hydrochloride, lidocaine hydrocarbonate and pH-adjusted two per cent lidocaine hydrochloride (all with freshly added epinephrine) were compared in a double-blind manner for Caesarean section anaesthesia.

Methods

Following approval from the Clinical Screening Committee for Research involving Human Subjects of the University of British Columbia, 37 patients (ASA physical status I or II), presenting for elective Caesarean section under epidural anaesthesia were enrolled. Informed consent was obtained and then an epidural catheter was placed at either the L_{2-3} or L_{3-4} level. The observer anaesthetist and the patient were blinded as to the local anaesthetic solution used. The patients were randomly assigned to receive either two per cent lidocaine hydrochloride, lidocaine hydrocarbonate or pH-adjusted two per cent lidocaine, all with freshly added 1:400,000 epinephrine. The pH-adjusted solution was made by the addition of 2 ml of 8.4 per cent (wt/vol) sodium bicarbonate to 20 ml of two per cent lidocaine. The local anaesthetic was incrementally administered to achieve a loss of thermosensitivity (ice) to T_3 or T_4. Time zero was taken as the time of administration of the test dose. Measurements recorded were: onset time, time to peak effect, time to loss of temperature sensation to ice at S_2 and duration (regression of the block by two segments). The pH of the solution was measured within ten minutes of the mixing using a digital pH meter. Quality of analgesia was also noted on a scale of one to four, based on the amount of intravenous fentanyl required by the patient.

Data were analyzed for statistical significance using analysis of variance for randomized groups and the Kruskall-Wallis test. Significance was considered if $p < 0.05$.

Results

The groups were similar with respect to age, height, weight, parity and total amount of local anaesthetic administered. The only significant difference was in the pH of the 3 solutions ($p < 0.05$).

Discussion

According to the manufacturer, the pH for two per cent lidocaine can vary between five and seven. The actual pH is usually between 6.6 and 6.9. The pKa of lidocaine is 7.87. DiFazio *et al.*'s study compared commercially prepared lidocaine with epinephrine, which has a lower pH than that found in this study where the epinephrine was feshly added. As the body has to buffer the more acidic local anaesthetic, it is understandable that DiFazio demonstrated a difference, while our study did not. Controversy has existed over the efficacy of lidocaine hydrocarbonate compared to lidocaine hydrochloride.[3] This study has confirmed earlier work which failed to demonstrate a difference between lidocaine hydrochloride and lidocaine hydrocarbonate.

References

1 *McMorland GH et al.* Effect of pH-adjustment of bupivacaine on onset and duration of epidural analgesia in parturients. Can Anaesth Soc J 1986; 33: 537–41.

2 *DiFazio CA et al.* Comparison of pH-adjusted lidocaine solutions for epidural anesthesia. Anesth Analg 1986; 65: 760–4.

3 *Bromage PR, Nickel PM.* Local anesthetics: the carbonation controversy. Published by the University of Colorado Health Sciences Center, Denver, Colorado. 1986.

Metabolic acidaemia and cardiovascular performance in anaesthetized newborn swine

J. Oyston, J. Lerman, G.A. Volgyesi, F.A. Burrows

Department of Anaesthesia and the Research Institute, The Hospital for Sick Children and the University of Toronto, Toronto, Ontario

Both halothane[1] and metabolic acidaemia[2] decrease cardiac output in the newborn. Therefore, we investigated the combined effects of metabolic acidaemia and halothane anaesthesia on the cardiovascular system and baroreflex responses of newborn swine.

Methods

With the approval of the Animal Care Committee, seven fasting unpremedicated Yorkshire swine, ages five to seven days, and weighing 1.5–3.5 kg were studied. Anaesthesia was induced with halothane, nitrous oxide and oxygen. The trachea was intubated and the swine ventilated with oxygen and halothane. The external jugular vein was cannulated under direct vision using a Cook 5 cm double-lumen cannula, and the internal carotid artery using a five french feeding tube. Ventilation was adjusted to obtain a normal arterial pH and pCO_2. The swine were paralyzed with vecuronium and the inspired halothane concentration adjusted to maintain an end–tidal concentration of 0.5 MAC, analyzed using a Beckman LB2 medical gas analyzer. Ten $ml \cdot kg^{-1}$ of 0.2 per cent saline in five per cent dextrose were infused to compensate for the period of fasting.

Cardiovascular mesurements (heart rate (HR), systolic arterial pressure (SAP), central venous pressure (CVP), dP/dt and cardiac index (CI)) were recorded. Cardiac output was measured in duplicate using a dye dilution technique. Baroreceptor responses were measured both after 30 $\mu g \cdot kg^{-1}$ of phenylephrine (pressor) and after an infusion of sodium nitroprusside (depressor) administered in random order ten minutes apart. The R–R interval was correlated to the previous systolic arterial pressure using least squares linear regression.[3] A correlation coefficient ≥ 0.7 indicated a valid response. Acidaemia was induced using an infusion of 2 $ml \cdot kg^{-1}$ of 0.5 M hydrochloric acid over five to ten minutes followed by a slower infusion to maintain the pH below 7.20. Ionized calcium concentration was measured before and after acidaemia. Cardiovascular measurements and baroreceptor responses were repeated. Statistical significance was determined using paired t tests and ANOVA.

Results

The mean arterial pH was lowered from 7.45 to 7.15 ($p < 0.01$). The only statistically significant changes were an increase in dP/dt (Table), and an increase in plasma ionized calcium. There were no significant differences in baroreceptor responses before and after acidaemia (Table).

TABLE Results

	Control	*Acidaemia*
pH	7.45 ± 0.04	7.15 ± 0.06†
Ionized Calcium ($mm \cdot L^{-1}$)	1.3 ± 0.1	1.6 ± 0.1†
HR ($beats \cdot min^{-1}$)	209 ± 24	194 ± 30
SAP (mmHg)	79 ± 7	80 ± 9
CVP (cmH_2O)	3.0 ± 1	3.7 ± 2.0
dP/dt ($mmHg \cdot sec^{-1}$)	1050 ± 65	1305 ± 91*
CI ($L \cdot min^{-1} \cdot kg^{-1}$)	0.3 ± 0.05	0.22 ± 0.05
Pressor response $msec \cdot mmHg^{-1}$	1.1 ± 0.23 (n = 5)	2.7 ± 1.1 (n = 5)
Depressor response $msec \cdot mmHg^{-1}$	3.0 ± 2.2 (n = 7)	2.9 ± 1.9

Data are Mean ± SD, n = number of valid responses.
*Significantly different from control, $p < 0.05$.
†Significantly different from control, $p < 0.01$.

Discussion

This study demonstrates that newborn swine tolerate a severe and acute metabolic acidaemia combined with 0.5 MAC halothane anaesthesia. Supported in part with a grant from Heart and Stroke Foundation of Canada.

References

1 *Gallagher TM, Burrows FA, Lerman J.* Anesth Analg 1987; 66: S64.
2 *Fisher DJ.* Pediatr Res 1986; 20: 756–80.
3 *Smyth HS, Sleight P, Pickering GW.* Circ Res 1969; 24: 109–21.

The use of continuous lumbar epidural fentanyl for post-operative pain relief in thoracotamies

S.D. Bell, R. Berman, L. Ensalada
Thomas Jefferson University Hospital, Philadelphia, Pennsylvania

Although morphine and fentanyl are both effective analgesics when administered epidurally, morphine has several disadvantages which include delayed respiratory depression and a higher incidence of nausea, vomiting, and pruritus.[1] In addition, fentanyl has the advantages of rapid onset of action and short duration of action[2] and is thus well suited for use as a continuous infusion. The effectiveness of fentanyl for postoperative pain relief has been demonstrated using a continuous thoracic technique.[3] Since lumbar epidural catheter insertion is a technique in which most anaesthetists are skilled, we undertook this study to evaluate the effectiveness of continuous postoperative lumbar epidural fentanyl infusion for postoperative analgesia.

Methods

The protocol was approved by our institutional review board and informed consent was obtained from each patient. Twenty patients scheduled for pulmonary lobectomy had a lumbar catheter inserted preoperatively at the L_{3-4} or the L_{4-5} interspace. All fentanyl concentrations were at 10 $\mu g \cdot ml^{-1}$.[3] Upon emergence from anaesthesia, the patient received a bolus dose of 50 μg fentanyl epidurally (which was repeated in 20 minutes if needed) and started on a continuous epidural infusion of 60 $\mu g \cdot hr^{-1}$. The dose was titrated to patient comfort which was determined by the patients being able to cough, deep breathe, and elevate their arms with minimal increases in discomfort compared to their resting state. Arterial blood gases were done every four hours and 20 minutes post any infusion rate change, then shifted to every eight hours when the infusion was constant for six hours. Visual analog pain scale with both resting and movement values (0 – no pain; 10 – maximal pain) was evaluated every eight hours for the first 24–36 hours. Preop FEV_1 were done in unpremedicated patients. Postop FEV_1 were done the morning after surgery. Data were analyzed by paired t test and a $p < 0.05$ was considered significant.

TABLE I Respiratory measurements

Arterial pH	*PCO_2*	*Respiratory rate*	*FEV_1 as a % of preoperative value*
7.37 ± 0.04	42 ± 3.8	16 ± 4.1	69 ± 8.6

TABLE II Infusion rates and pain scores

Infusion rates Fentanyl ($\mu g \cdot hr^{-1}$)	*Visual analogue pain score*	*Visual analogue pain score with movement*
68 ± 18	1.7 ± 0.5*	1.9 ± 0.6*

*$p < 0.05$ compared to post infusion times.

Results

Table I shows respiratory measurements of the patients. Table II shows fentanyl infusion rates with visual analogue pain scores.

Discussion

Continuous lumbar epidural infusion is an effective and safe way to administer fentanyl for postoperative analgesia in thoracotomies. During the continuous infusion the pH remained above 7.35 in all patients except for two transient values of 7.32 and 7.33, which occurred shortly after the initial fentanyl bolus. PCO_2 remained stable during the continuous infusion. FEV_1 values of 69 per cent of preoperative value is much imporved over the 25–45 per cent normally reported.[1,3] Mean visual analogue pain scores were 1.7 ± 0.5 and had minimal increases to 1.9 ± 0.6 with bilateral arm elevation and incentive spirometry demonstrating both static and dynamic analgesia when contrasted to the visual analogue scores of 5.2 ± 1.3 in post-infusion state. Titration of pain management is relatively easy since dosage ranges are narrow and fentanyl's onset of action is rapid.

References

1 *Cousins MJ, Mather LE.* Intrathecal and epidural opioids. Anesthesiology 1984; 61: 276–310.
2 *Rutter DO et al.* Extradural opioids for post operative analgesia. Br J Anaesth 1981; 53: 915–9.
3 *Welchew EA, Thorton JA.* Continuous thoracic epidural fentanyl. Anaesthesia. 1982; 37: 304–16.

Continuous intercostal nerve block for pain relief following thoracotomy – a new approach

V. Chan, F. Chung, D.C.H. Cheng, T.J. Kirby, A. Chung, A. Sandler

Departments of Anaesthesia and Surgery, Toronto Western Hospital, University of Toronto, Toronto, Ontario

Pain associated with respiratory movement can impair lung function following thoracotomy. It limits voluntary ventilation thus predisposing patients to possible complications of atelectasis, pneumonia and hypoxaemia. A "prn" mode of parenteral narcotic administration seldom achieves satisfactory analgesia. Extradural use of narcotic agents carries certain side effects. Intercostal nerve block is effective in relieving pain for thoracotomy but repeated block requiring needle puncture at multiple intercostal levels is necessary.[1] The present study was undertaken to evaluate the use of continuous intercostal nerve block for post-thoracotomy pain relief.

Methods

This study was approved by the Institutional Ethics Committee. Six patients scheduled for thoractomy for lung resection were studied. All patients were monitored with electrocardiogram, oximeter, and radial artery catheter. Diazepam 5–10 mg PO was given as premedication. General anaesthesia was induced with 3–5 $mg \cdot kg^{-1}$ thiopentone and succinylcholine 1 $mg \cdot kg^{-1}$. Patients were intubated with either a double lumen tube or an endotracheal tube and bronchial blocker. Anaesthesia was maintained with nitrous oxide, oxygen, isoflurane and pancuronium. Fentanyl, 2 $\mu g \cdot kg^{-1}$, was given at skin incision and 1 $\mu g \cdot kg^{-1}$ every hour as needed. Before the thoracic cavity was closed at the conclusion of the surgery, two extradural catheters were inserted into the posterior intercostal space. The first catheter was put one space above and the second, one space below the site of incision under direct vision. Each catheter punctured the parietal pleura over the posterior chest wall internally and advanced 3–4 cm either cephalad or caudad. Its position in the posterior intercostal space was secured with suture and its other end was brought out near the incision site. After reversal of muscle relaxant, all patients were extubated at the end of the operation.

Thirty minutes following surgery, the extradural catheter was aspirated to check for blood and air. After a negative aspiration test, 10 ml of 0.5 per cent bupivacaine with 1:200,000 epinephrine was injected into the catheter through a filter, slowly over 5 minutes. The same procedure was repeated in the second catheter. Bupivacaine "top-up" was given every six hours for 24 hours. Intravenous morphine 0.05–0.07 $mg \cdot kg^{-1}$ was given upon request for pain relief. The total amount of intravenous morphine used within 24 hours was documented. Serum bupivacaine levels were measured at pre-top-up, 2.5, 5, 10, 20, and 60 minutes after top-up.

Results

All patients were successfully studied and there were no complications. The data are shown in Table I. The mean 24-hour morphine requirement was 11.17 ± 3.89 mg. One patient required no intravenous morphine supplement and one required

TABLE I Results

	Mean	*SD*
Age	64.33	4.37
Weight (kg)	69.35	5.31
Duration of surgery (hr)	3.17	0.17
24-hour morphine (mg)	11.17	3.89

TABLE II Serum bupivacaine levels ($\mu mol \cdot L^{-1}$, mean ± SE)

	Pre top-up	*2.5 min*	*5 min*	*10 min*	*20 min*	*60 min*
1st intercostal	0	1.0 ± 0.5	0.93 ± 0.5	0.97 ± 0.5	0.97 ± 0.5	0.82 ± 0.4
2nd-top-up	0.56 ± 0.3	1.29 ± 0.4	1.52 ± 0.4	1.61 ± 0.4	1.75 ± 0.5	1.58 ± 0.5
3rd-top-up	1.30 ± 0.4	2.14 ± 0.6	2.12 ± 0.6	2.17 ± 0.6	2.22 ± 0.7	2.26 ± 0.7
4th-top-up	2.0 ± 0.8	3.15 ± 1.1	3.39 ± 1.1	3.46 ± 1.1	3.31 ± 1.1	3.24 ± 1.1

only 2 mg of intravenous morphine. The serum bupivacaine level is shown in Table II, and is below toxic level.

Discussion

Continuous intercostal block by insertion of extradural catheters into the posterior intercostal space is an effective and alternate method of pain relief following thoractotomy.

Reference

1 *Moore DC.* Intercostal nerve block for postoperative somatic pain following surgery of thorax and upper abdomen. Br J Anaesth 1975; 47: 284.

Dipyridamole-thallium scanning in the preopertive assessment of patients undergoing abdominal aortic surgery

R. Grant, C. Morgan, M. Page, D. Malm, V. Huckel, L. Jenkins

Departments of Anaesthesiology and Medicine, Vancouver General Hospital, Vancouver, British Columbia and Department of Anaesthesia, Brompton Chest Hospital, London, England.

Previous studies suggest that patients with clinical coronary artery disease (CAD) are at risk of developing myocardial ischemia following intrarenal aortic cross clamping (XC)[1] and that patients with CAD as demonstrated by dipyridamole-thallium scanning (DTS) are at a 50 per cent risk of cardiac morbidity or mortality when undergoing aortic or lower limb vascular surgery.[2] Studies have also shown that resting LVEF determined by nuclear angiography does not identify a subgroup of patients in whom PCWP and CVP correlate poorly.[3,4] The present study was designed to assess the usefulness of DTS in predicting the intraoperative relationship between CVP and PCWP and overall cardiac performance during aortic surgery.

Methods

Following institutional approval 25 ASA physical status II–IV patients scheduled for elecive aortic aneurysm grafting underwent DTS. Patients with normal scans (Group I) were compared to those with redistribution perfusion defects (Group II). Patients were premedicated with diazepam 0.15 $mg \cdot kg^{-1}$ and morphine 0.15 $mg \cdot kg^{-1}$. Intravenous, radial artery, and pulmonary artery catheters were inserted under local anaesthesia. Anaesthesia was induced with fentanyl 5–6 $\mu g \cdot kg^{-1}$ and thiopentone 1–3 $mg \cdot kg^{-1}$. Muscle relaxaton was achieved with pancuronium 0.1 $mg \cdot kg^{-1}$. Anaesthesia was maintained with N_2O 50–70 per cent in O_2 and isoflurane 0–1.5 per cent end–tidal as well as further increments of fentanyl. Hypertension or excessive increases in filling pressures were treated with nitroglycerin IV. Mannitol 0.2 $g \cdot kg^{-1}$ IV was given before XC.

Measured values included heart rate, BP, CO, CI, CVP, PCWP, and PA pressures. Data were obtained prior to induction (control), two to three minutes prior to XC, 2, 15, 30, and 45 minutes post XC, two minutes after XC release, during surgical closure and one hour after reaching the recovery room. The relationship between CVP and PCWP was assessed with linear regression analysis for each group. The changes in CVP and PCWP two minutes after XC were similarly analyzed. Within each group measured values were compared with control using ANOVA and then with the Neuman Kuels test. Between group comparisons were made with the independent student t test. $P < 0.05$ was considered significant.

Results

There were 11 patients in Group I and nine in Group II (five patients with fixed perfusion defects on DTS were not included in the data analysis). Preinduction PCWP was lower in Group I than in Group II. There were no other significant haemodynamic differences between groups at any time. In Group I the CI was significantly lower than control throughout the XC period; in Group II the CI was significantly lower than control at 45 minutes post XC only. In both groups PCWP was significantly correlated with CVP; for Group I: PCWP = 4.5 + 0.64 (CVP); $r = 0.64$, $p < 0.001$ and for Group II: PCWP = 3.6 + 0.92 (CVP); $r = 0.83$, $p < 0.001$. Analysis of 95 per cent confidence limits showed no difference between these correlations. At the time of XC the observed changes in CVP and PCWP correlated significantly (Figure). For Group I: δ PCWP = −0.52 + 0.69 (δCVP); $r = 0.78$, $p = 0.005$ and for Group II: δPCWP = −0.15 + 0.95 (δCVP); $r = 0.87$, $p = 0.002$. There was no difference between these correlations.

Two patients in Group I and one patient in Group II received nitroglycerin intraoperatively. There were no significant ECG

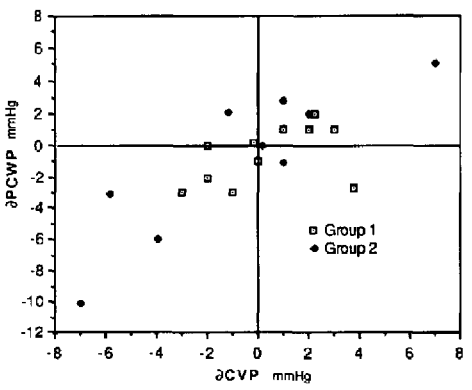


FIGURE δCVP vs δPCWP 2 min after aortic cross damping.

changes documented postoperatively. All patients were discharged from hospital.

Discussion

An abnormal DTS did not identify patients in whom CVP correlated poorly with PCWP. In previous reports a low-resting LVEF similarly did not identify such a patient group.[3,4] In our patients there was a better correlation between CVP and PCWP than in those reports; the reason for this is not apparent. Other studies have involved anaesthetic management that is not appropriate for the patient with CAD[1] or failed to report the anaesthetic technique employed.[2] This may partly explain the better correlation between CVP and PCWP and better patient outcome in our series. An abnormal DTS is not always an indication for coronary angiography prior to aortic surgery.

References

1 *Attia R et al.* Myocradial ischemia due to infrarenal aortic cross clamping during aortic surgery in patients with severe coronary artery disease. Circulation 1976; 53: 961–5.

2 *Boucher C et al.* Determinations of cardiac risk by dipyridamole-thallim scanning before periferal vascular surgery. N Engl J Med 1985; 312: 389–94.

3 *Ansley D et al.* The relationship between central venous pressure and pulmonary capillary wedge pressure during aortic surgery. Can J Anaesth 1987; 34: 594–600.

4 *Gallacher N et al.* The value of nuclear angiography in the preoperative assessment of patients undergoing elective aortic surgery. Can Anaesth Soc J 1984; 31: 523–7.

The haemodynamic effects of epidural anaesthesia for peripheral vascular surgery – systemic effects of epidural epinephrine

F.C. Salevsky, D.G. Whalley, G.R. Robbins

Department of Anaesthesia, Royal Victoria Hospital and McGill University, Montreal, Quebec

The haemodynamic (HD) effects of epidural anaesthesia are dependent on the type and dose of anaesthetic, the addition of epinephrine and the patient's age and medical condition.[1,2] The present study was designed to determine the haemodynamic effects of epidural epinephrine (E), 5 $\mu g \cdot ml^{-1}$, added to bupivicaine (B), 0.75 per cent in elderly patients with cardiac disease undergoing peripheral vascular surgery (PVS).

Methods

After institutional approval and informed patient consent, ten patients requiring PVS were randomly allocated in a double-blind fashion to Group B or Group B + E (Table I). All patients had a history and/or ECG evidence of cardiac disease for which they were being treated (Table II). Patients were premedicated with diazepam 5–10 mg PO together with their usual cardiac medication. On arrival in the operating room, a modified V_5 ECG and a non-invasive blood pressure monitor were applied and a pulmonary artery catheter was inserted. After control measurements were obtained, the patient sat up and 12 ml of test solution (bupivacaine 0.75 per cent ± epinephrine, 5 $\mu g \cdot ml^{-1}$) was injected epidurally over five minutes at $L_{3,4}$. The patient was then placed supine and a urinary catheter inserted. Haemodynamic measurements were obtained at 15 and 45 min after epidural injection. Pulmonary wedge pressure and central venous pressure were maintained at control values by an infusion of lactated Ringer's solution. The maximum cephalad spread of anaesthesia and the time taken to achieve it were measured by pinprick. Data are reported as mean ± standard deviation and were analyzed using a paired Student's t test; $p < 0.05$ was considered to be significant.

TABLE I Demographic data

Group	*B*	*B + E*
Age (yr)	70.0 ± 8.3	71.8 ± 12.3
Ht (cm)	165 ± 16	164 ± 6
Wt (kg)	68.2 ± 5.9	67.2 ± 12.3
BSA (m)	1.75 ± 0.15	1.72 ± 0.14

TABLE II Cardiac medication

	Number of patients
Digoxin	3
Diuretics	4
β-Adrenergic agonists	1
Ca-channel blockers	4

TABLE III Haemodynamic data (*$p < 0.05$ – between groups)

	Group	*MBP (torr)*	*CO* $(L \cdot min^{-1})$	*SVR* $dyne \cdot sec \cdot cm^{-5}$	*HR (bpm)*
Control	B	110.4 ± 13.4	4.0 ± 1.7	2716 ± 2171	70.2 ± 9.5
	B + E	98.2 ± 16.5	4.2 ± 1.5	1876 ± 507	69.2 ± 14.7
15 min	B	109.4 ± 16.5*	4.1 ± 1.5	2243 ± 924*	66.2 ± 9.7
	B + E	77.6 ± 19.3	5.0 ± 1.2	1174 ± 434	67.8 ± 11.0
45 min	B	81.0 ± 17.9	4.1 ± 0.7	1779 ± 483	59.8 ± 7.4
	B + E	87.0 ± 25.1	4.9 ± 1.1	1380 ± 590	63.8 ± 9.7

Results

Haemodynamic data for the demographically similar groups at control, 15 and 45 min are presented in Table III. There were significant differences in mean blood pressure (MBP) and systemic vascular resistance (SVR) between the groups at 15 min, but not at 45 min. No significant difference in heart rate (HR) or cardiac output (CO) was present between the groups at any time. The highest sensory block was achieved within 20 min in all cases and averaged $T_{3,5}$ (Group B, range T_8–T_2) and $T_{4,0}$ (Group B + E, range T_7–T_2). Urine output at 15 and 45 min was 131 ± 122 and 182 ± 150 ml (Group B) and 74.0 ± 76.0 and 196 ± 154 ml (Group B + E). Fluid given at 15 and 45 min was 740 ± 164 and 1340 ± 350 ml (Group B) and 890 ± 394 and 1640 ± 297 ml (Group B + E).

Discussion

The data show a greater decrease in MBP and SVR in Group B + E than in Group B at 15 min. A difference in the level of

sympathetic block between groups is unlikely considering the similarity of sensory block levels. The difference in HD effects could be explained by a variation in the absorption of B between groups. The addition of E to B may slow the absorption of B thus decreasing the plasma concentration of B. A more likely possibility is that the difference in HD between the groups at 15 minutes is due to β-adrenergic stimulation by absorbed epinephrine. The lack of HR response cannot be ascribed to any effect of β-adrenergic blocking medication and likely represents differential end–organ response to epinephrine in these elderly patients. The study shows that in elderly patients with cardiac disease undergoing PVS, epidural epinephrine, 5 $\mu g \cdot ml^{-1}$ added to bupivacaine, 0.75 per cent is associated with a greater decrease in MBP and SVR than epidural bupivacaine, 0.75 per cent.

References

1 *Stanton-Hicks, MD.* Cardiovascular effects of extradural anaesthesia. Br J Anaesth 1975; 47: 253–63.

2 *Whalley DG, Labrecque P, Quance D.* The cardiovascular effects of epidural bupivicaine 0.75 per cent with epinephrine for femoral-popliteal bypass surgery. Can J Anaesth 1987; 34: S100.

Does peri-aortic collateral vascularisation predict the haemodynamic changes following abdominal aortic cross-clamp and release?

A.J. Cunningham, J.F. Boylan, D.P. O'Toole

Department of Anaesthesia, Royal College of Surgeons in Ireland, Dublin, Ireland

Studies comparing the haemodynamic responses to infra-renal aortic cross-clamping and release in patients undergoing abdominal aortic aneurysm (AAA) resection and by-pass grafting for aorto-iliac occlusive disease (AOD) have noted either no differences between the two patient populations[1] or a tendency for less marked haemodynamic changes in the AOD group.[2] The extent of peri-aortic vascularisation has been proposed as a possible explanation for altered haemodynamic responses between AAA and AOD patients.[3] The objectives of this study were to compare the haemodynamic changes following clamping, during the clamp period and following clamp release and to correlate the extent of preoperative angiographic collateralisation with the observed haemodynamic changes in patients with AOD.

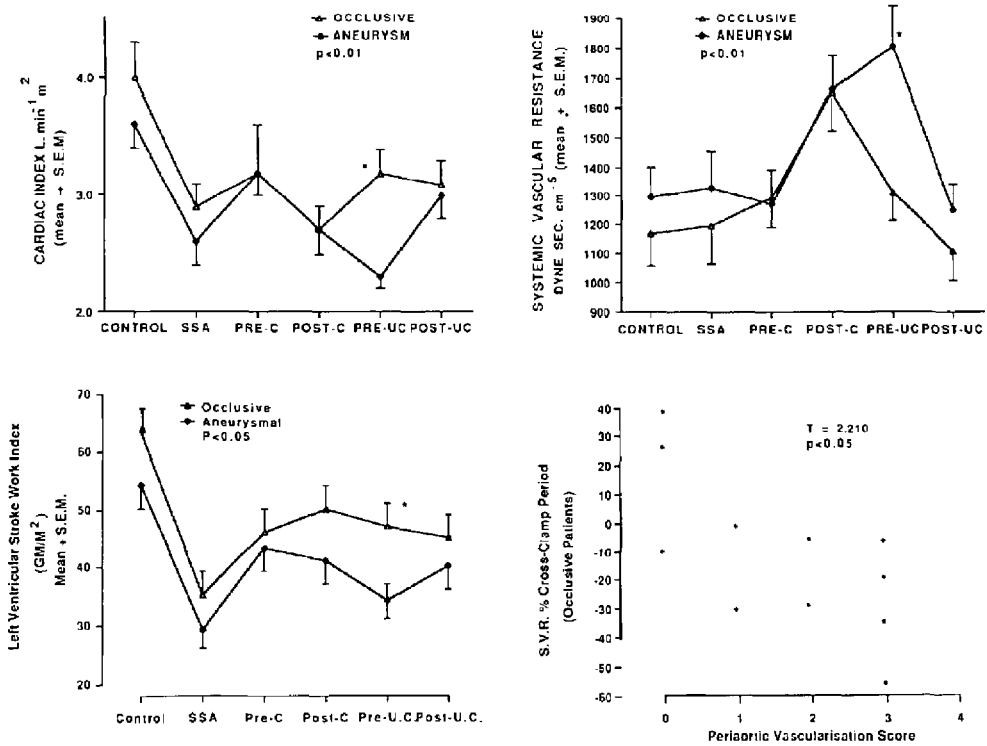


FIGURE Results.

Methods

Following ethical aproval and informed consent 30 ASA physical status II–III patients (18AAA, 12 AOD) were prospectively evaluated with Goldman multivariate risk analysis. LVEF was determined by radionuclide scanning and 2D-echocardiography. AAA patients were evaluated with abdominal ultrasound while AOD patients underwent preoperative aortography. A scoring system was devised to assess the extent of periaortic collateral vascularisation. A standardised anaesthetic technique was employed – fentanyl 3–6 $\mu g \cdot kg^{-1}$ IV followed by induction with thiopentone 2–3 $mg \cdot kg^{-1}$. Pancuronium 0.1 $mg \cdot kg^{-1}$ facilitated tracheal intubation and controlled normocapnic ventilation was commenced with N_2O/O_2 and 0.5–1 per cent isoflurane. Radial and pulmonary artery catheters were inserted under local anaesthesia. Pulmonary artery occlusion pressure was maintained between 10–15 mmHG and hypertension >20 per cent of baseline was controled by nitroglycerin infusion. Measured and derived haemodynamic data were obtained before and after induction, aortic cross-clamping and release. Statistical analysis between and within the two groups included parametric Student's t testing, repeated multivariate analysis of variance and Spearman rank correlation coefficient testing. Data was expressed as mean ± SEM; $p < 0.05$ was considered significant.

Results

The AOD patients were younger, required less perioperative fluids and had a longer aortic cross-clamp time. Aortic cross-clamp was associated with similar reductions in CI and elevation of SVR in both groups. During the cross-clamp period, LVSWI and CI decreased while SVR increased in the AAA group, while the AOD group showed an improved CI, stable LVSWI and a reduced SVR, which correlated with the extent of peri-aortic vascularisation seen on preoperative aortography (Figure).

Discussion

The extent of collateral circulatory changes associated with AOD influences the magnitude of CI and SVR changes during the cross-clamp period. Chronic collateral circulation associated with AOD may permit continuous pelvic and lower extremity perfusion during aortic cross-clamping. Preoperative information concerning the extent of peri-aortic collateralisation may influence the choice of anaesthetic management and monitoring techniques.

References

1 *Dunn E, Prager RL, Fry W, Kirsu MM.* The effect of abdominal aortic cross-clamping on myocardial function. J Surg Res 1977; 22: 463–6.

2 *Damask MC, Weissman C, Rodriguez et al.* Abdominal aortic cross-clamping. Metabolic and hemodynamic consequences. Arch Surg 1984; 119: 1330–7.

3 *Johnston WE, Balestrieri FJ, Plonk G et al.* The influence of periaortic vessels on the intraoperative effects of acute aortic occlusion in patients with aorto-occlusive disease or abdominal aortic aneurysm. Anesthesiology 1987; 66: 386–9.

Anaesthesia experience with interventional cardiology

S. Malviya, F.A. Burrows, L. Benson, A.E. Johnston

Departments of Anaesthesia, Cardiology and the Research Institute, The Hospital for Sick Children, University of Toronto, Toronto, Ontario

In recent years interventional cardiology has replaced thoracic and cardiac surgery in the treatment of certain cardiac abnormalities in children. In our institution we provide anaesthetic support for the following procedures: dilation of recoarctation of the aorta (CoArc); patent ductus arteriosus occlusion (PDA); aortic valve dilation (AVD); dilation of pulmonary artery stenosis (PAS); and dilation of pulmonary valve stenosis (PVS).[1] It is important to establish a steady haemodynamic state to determine the pressures and oxygen saturations in the various cardiac chambers since management decisions are made on the basis of estimates of intracardiac shunts, vascular resistances and systemic and pulmonary flows in these patients. In this paper we review the anaesthetic and non-anaesthetic experience since the inception of interventional cardiology at our institution.

Methods

With approval from the Committee on Human Research we reviewed the charts of all patients who had undergone interventional cardiological procedures from January 1984 to July 1987. Patients were divided into four groups depending upon the anaesthetic technique administered: ketamine by infusion (K); inhalational anaesthesia with controlled ventilation (I); narcotic anaesthesia (N) and intravenous or intramuscular sedation with or without anaesthesia standby (S). The charts were reviewed for anaesthesia/sedation or procedural related complications. Intergroup comparisons were analyzed using Chi-square analysis.

Results

One hundred and seventy-six patients underwent a total of 184 procedures. The numbers in each group were: K (n = 90), I (n = 42), N (n = 26) and S (n = 26). A total of 112 complications were identified for an incidence of 60.9 per cent. Thirty-five complications (31.2 per cent) were anaesthesia related, one complication (0.9 per cent) was related to sedation and 76 complications (67.9 per cent) were related to the procedure. Vascular thrombosis or injury was the most common complication occuring in 33 procedures. Of these, seven resolved with heparin therapy, ten required fibrinolytic therapy and 16 required surgical repair. Cardiovascular instability occurred during 28 procedures. In 19 cases it was transient related to procedural manipulations, procedural-induced arrhythmias and in one case myocardial depression from halothane. In four cases it resulted from acute aortic insufficiency following aortic valve dilation (AVD). Three of these patients required surgical repair of the valve with one death. There were four cases of cardiac perforation, two of which required urgent thoracotomy. There was one cardiac arrest from catheter-induced ventricular fibrillation. Respiratory complications occurred in 26 procedures and were most common in patients anaesthetized with ketamine ($p < 0.05$) (Table). Nineteen of these patients developed CO_2

TABLE Respiratory complications

	Ketamine	*Sedation*	*Inhalation*	*Narcotic*
Obstruction	7/90	0/26	0/42	0/26
Hypercarbia	13/90	1/26	1/42	4/26

retention and seven developed airway obstruction which required intubation in five. All seven patients with airway obstruction and 13 of 19 patients with CO_2 retention were anaesthetized with K. Respiratory complications (Table) were most common in patients in Group K and least common in patients in Group I or Group S ($p < 0.05$).

Discussion

Interventional cardiological procedures are associated with complications that require the immediate availability of an operating room and cardiopulmonary bypass. Anaesthesia with ketamine and spontaneous respiration is preferred by the cardiologists because the cardiovascular data obtained is readily comparable to previous or future studies carried out with sedation. Our experience indicates that ketamine is associated with a higher incidence of respiratory complications than the other techniques. We recommend careful monitoring by personnel experienced in airway management in patients undergoing interventional cardiac catheterization under ketamine anaesthesia or heavy sedation.

Reference

1 *Benson LN, Smallhorn JS, Freedom RM, Truster GA, Rowe RD.* Pulmonary valve morphology after balloon dilatation of pulmonary valve stenosis. Cath Cardiovas Diag 1985; 11: 161–6.

Comparison of SNP and AMP during low dopamine infusion in the dog

B.A. Finegan, A.S. Clanachan
Departments of Anaesthesia and Pharmacology, University of Alberta Hospital, Edmonton, Alberta

The vasodilators sodium nitroprusside (SNP) and glyceryl trinitrate (GTN) are frequently used in combination with dopamine (DA) to decrease afterload and enhance cardiac output. Adenosine (AR), an endogenous vasoactive nucleoside, may also be useful in combination with DA. It possesses direct vasodilator activity and its anti-adrenergic effects reduce baroreceptor-reflex-induced tachycardia and renin secretion. AR has been advocated for the management of supraventricular tachycardia[1] and for the induction of deliberate hypotension.[2] The haemodynamic effects of adenosine 5'-monophosphate (AMP), a soluble AR pro-drug, were compared with SNP during infusion in narcotic/barbiturate anaesthetized dogs.

Methods

Following premedication with morphine (10 mg), anaesthesia was induced with fentanyl (30 $\mu g \cdot kg^{-1}$) and pentobarbitone (30 $mg \cdot kg^{-1}$) and maintained with an infusion of fentanyl (20 $\mu g \cdot kg^{-1} \cdot hr^{-1}$), pentobarbitone (3 $mg \cdot kg^{-1} \cdot hr^{-1}$) and pancuronium (100 $\mu g \cdot kg^{-1} \cdot hr^{-1}$). Animals were ventilated mechanically to maintain normoxia and normocapnia by a Harvard respirator. ECG, pulmonary artery, systemic arterial and central venous pressures, and cardiac output were measured. A-V nodal conduction time was determined from high-speed recordings of the ECG during pacing with a fluroscopically positioned atrial bipolar pacing electrode at 3 Hz. After 45 min stabilization, control measurements were obtained and a DA infusion (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) was initiated. Ten min after the start of the infusion AMP or SNP (randomized order) were infused at a rate sufficient to lower and maintain mean arterial pressure (MAP) at 80 mmHg for ten minutes.

TABLE Results: Effects of AMP or SNP infusion during dopamine administration (5 $\mu g \cdot kg^{-1} \cdot min^{-1}$)

	DA (n = 8)	*DA + AMP (n = 8)*	*DA (n = 8)*	*DA + SNP (n = 8)*
Dose ($mg \cdot kg^{-1} \cdot min^{-1}$)	—	1.1 ± 0.3	—	0.014 ± 0.005
BP – mean	104 ± 3	80 ± 0	106 ± 4	80 ± 1
HR ($beat \cdot min^{-1}$)	89 ± 8	121 ± 8	97 ± 9	159 ± 17
PA – systolic	20 ± 1	23 ± 2	20 ± 1	16 ± 1
– diastolic	5 ± 1	7 ± 1	5 ± 1	5 ± 1
CO ($L \cdot min^{-1}$)	2.0 ± 0.2	3.2 ± 0.3	2.2 ± 0.2	2.4 ± 0.2
SV (ml)	23.9 ± 3.3	27.3 ± 3.4	24.9 ± 3.0	17.5 ± 3.5
SVR ($dyne \cdot sec \cdot cm^{-5}$)	4581 ± 627	2172 ± 246	3864 ± 400	2824 ± 265
PVR ($dyne \cdot sec \cdot cm^{-5}$)	223 ± 37	165 ± 24	203 ± 20	152 ± 19
A-V interval (msec)	135 ± 6	119 ± 8	135 ± 6	118 ± 8

Results and Discussion

Both SNP (14 ± 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and AMP (1.1 ± 0.3 $mg \cdot kg^{-1} \cdot min^{-1}$) induced a rapid fall in MAP to the target value. Maintenance of this pressure was easier with AMP.

Cardiac output was higher during DA-AMP administration than with either DA alone ($p < 0.01$) or DA-SNP combination ($p < 0.01$) (Table). This difference in cardiac output between the combinations was probably due to the marked reflex tachycardia during DA-SNP infusion that caused a significant decrease ($p < 0.05$) in stroke volume. Potential AMP-induced A-V nodal depression was not observed; A-V nodal conduction time decreased ($p < 0.05$) during both DA-AMP and DA-SNP administraiton, probably due to reflex elevation of sympathetic tone. DA-AMP has advantages over DA-SNP and this combination warrants further investigation.

References

1 *Dimarco JP, Sellers TD, Berne RM et al.* Adenosine: electrophysiologic effects and therapeutic use for terminating paroxysmal supraventricular tachycardia. Circulation 1983; 68: 1254–63.
2 *Fredholm BB, Sollevi A.* Cardiovascular effects of adenosine. Clin Physiol 1986; 6: 1–21.

The effect of age on the duration of block produced by bolus injection of atracurium

W.S. Beattie, D.N. Buckley, C. Deguzman, J.R. Hewson, J.B. Forrest
McMaster University Medical Centre, Hamilton, Ontario

We are aware of one study that assesses the effect of age on the action of atracurium.[1] D'Hollander *et al.* utilized continuous infusion and concluded no effect of age. Continuous infusion

pharmacokinetics are different and may alter the resonse to atracurium. The purpose of our study was to assess the effect of age on the duration of block after a single bolus of 0.5 $mg \cdot kg^{-1}$ of atracurium.

Methods

The study was approved by the Research Committee of our institution. Patients were ASA physical status I or II with normal serum electrolytes, and Hgb, undergoing procedures with minimal blood loss. Patients taking antibiotics or drugs known to prolong neuromuscular blockage were specifically excluded from the study. Patients between the ages of 16 and 84 received either a balanced or a nitrous narcotic technique. All patients were premedicated with mepenidine 1.0 $mg \cdot kg^{-1}$ and dimenhydrinate 1.0 $mg \cdot kg^{-1}$ one hour preoperatively. The balanced technique consisted of fentanyl 5.0 $\mu g \cdot kg^{-1}$; a sleep dose of thiopentone, N_2O/O_2; FiO_2, 30–40 per cent and enflurane 0.5–1.5 per cent. The nitrous narcotic group received a sleep dose of thiopentone and 10.0 $\mu g \cdot kg^{-1}$ of fentanyl, N_2O/O_2; FiO_2, 30–40 per cent.

Neuromuscular function was assessed with a Puritan Bennet NMT 221. After calibration, atracurium 0.5 $mg \cdot kg^{-1}$ was administered over one minute. We did not observe flushing or evidence of histamine release. All patients were monitored for changes in heart rate, blood pressure, during induction every 30 seconds and every five minutes thereafter. A continuous record of NMT was obtained which allowed for determination of total duration of block; defined as the time from injection to 100 per cent return train-of-four: speed of onset; time from injection to 100 per cent blockage: time of absolute blockade; time in minutes from 100 per cent blockade to one per cent recovery, and recovery time (one per cent return T_1 to 100 per cent recovery T_4). Analysis was by means of linear regression. Unpaired t tests were utilized to compare one anaesthetic technique to the other.

Results

The regression equation for the duration of block with the nitrous narcotic technique was Dur (min) = 28.3 + 0.4 (age) $r = 0.82$ $p < 0.001$. The balanced technique relationship is shifted upward and the slope is increased where Dur (min) = 49.4 + 0.6 (age) $r = 0.96$ $p < 0.001$. The Table compares technique and effects of age on the onset, absolute block and recovery of block. Onset was inversely related to age; however, the anaesthetic technique did not alter or shift this relationship. Absolute block was directly related to age and the addition of enflurane to the anaesthetic shifted the relationship upward and increased the slope.

TABLE Results

	Narcotic/nitrous $n = 21$	*Narcotic/ethrane* $n = 17$
Onset (time from 100% to 0%)	9.4–0.07 (age) $r = 0.81$ $p < 0.001$	9.6–0.09 (age) $r = 0.82$ $p < 0.01$
Absolute block 0% to 1% recovery	−5.6 + 0.4 (age) $r = 0.86$ $p < 0.01$	−12 + 0.5 (age) $r = 0.84$ $p < 0.01$
Recovery 1% T_1 to 100% T_4	21–34 min	27–40 min

Discussion

The results show total duration of block and absolute duration of block are directly related to age. Enflurane appears to potentiate the block with the greatest effect in the elderly. There was no problem with reversal of neural blockade. No adverse events related to the use of atracurium in the elderly were seen. The effect of age on duration of neural block is small but may be potentiated by inhalational agents. D'Hollander *et al.* controlled PCO_2 while our study did not. A possible explanation for the divergent findings of our studies could be that our elderly patients were hyperventilated with resultant alkalosis, which secondarily prolongs the effect of atracurium. In a separate group of elderly patients with PCO_2 controlled at 40 mmHg the duration of block was 59.8 ± 3.6 minutes. This time varied 3.4 ± 0.6 minutes from the time predicted by the above regression equation. Moreover, when PCO_2 was adjusted to 35 mmHg, no prolongation of block occurred in these eight patients.

Reference

1 *d'Hollander AA, Luycky C, Barvais L, DeVille A.* Clinical evaluation of atracurium besylate requirement for a stable muscle relaxation during surgery. Lack of age related effects. Anesthesiology 1983; 59: 237–40

Monitoring of neuromuscular function from the facial muscle and hypothenar muscle – an electromyographical evaluation

M.D. Sharpe, A.M. Lam, R. Merchant, P.H. Manninen
Department of Anaesthesia, University Hospital, University of Western Ontario, London, Ontario

Accurate assessment of residual neuromuscular blockade following a surgical procedure necessitating the use of muscle relaxants is of critical importance to ensure adequate postoperative respiratory function. Intraoperative train-of-four (TOF) stimulation of the facial muscle groups has been used as a monitor of neuromuscular blockade due to the proximity and convenience to the anaesthetist. However, limited assessment has been made of the correlation between facial muscle and hypothenar muscle blockade. The purpose of this study was therefore to compare the evoked electromyogram of the orbicularis oris and hypothenar muscles in healthy patients following administration of atracurium during general anaesthesia.

Method

Following institutional approval, seven healthy patients, aged 17–51, were studied. General anaesthesia was induced with thiopentone (4–5 $mg \cdot kg^{-1}$), fentanyl (3 $\mu g \cdot kg^{-1}$), lidocaine (1 $mg \cdot kg^{-1}$) and tracheal intubation facilitated with succinylcholine (0.5–0.75 $mg \cdot kg^{-1}$). Maintenance anaesthesia consisted of oxygen/nitrous oxide (30/70 per cent), and isoflurane

(0.75–1.0 per cent). Positive pressure ventilation was used to maintain normocapnia. Thirty minutes following the administration of succinylcholine and after full recovery of the electromyogram (EMG), control EMG's were recorded as follows: using two DATEX NMT-221 monitors (Puritan-Bennett), EMG's were recorded simultaneously with surface electrodes over the hypothenar muscles and orbicularis oris muscles in response to TOF supramaximal stimuli delivered to the ulnar and facial nerves respectively at a frequency of 2 Hz. Atracurium (0.6–1.0 $mg \cdot kg^{-1}$) was administered (time zero) to abolish T4. Spontaneous recovery of the neuromuscular function was then allowed to occur and EMG with display of T4/T1 ratios were continuously recorded. When the T4/T1 ratio of the orbicularis oris muscle reached 0.7, the simultaneous T4/T1 ratio of the hypothenar muscle was also noted. The recovery time (from time zero with T4/T1 of 0 to a T4/T1 of 0.7) was measured in both groups. Residual neuromuscular blockade was then completely antagonized using edrophonium (0.5 $mg \cdot kg^{-1}$) and atropine (7 $\mu g \cdot kg^{-1}$) and the EMG recording discontinued. For statistical analysis, comparison of the recovery time was made using a paired Student's t test, and the comparison between T4/T1 of the hypothenar muscle and the orbicularis oris muscle when the latter was 0.7 was made using the Wilcoxon ranked-sum test. Results are given as mean ± SD.

Results

The level of maxium blockade was similar for both muscle groups in each patient. However, the spontaneous recovery of the neuromuscular blockade began in the orbicularis oris muscle prior to the hypothenar muscle in all cases. The time required for the oribularis oris muscle to recover to a T4/T1 ratio of 0.7 was significantly less compared to the hypothenar muscles (31.8 ± 3.1 min vs 44.9 ± 7.5 min, $p < 0.05$). Similarly, when the orbicularis muscle T4/T1 ratio = 0.7, the mean T4/T1 ratio of the hypothenar muscles was significantly less (0.29 ± 0.16, $p < 0.05$).

Discussion

Previous reports have shown that a T4/T1 ratio exceeding 0.7 measured in the hypothenar muscles is indicative of adequate respiratory function.[1,2] This study compared the orbicularis oris with the hypothenar muscle groups to assess the utility of the former muscle group as a monitoring site for determination of adequate recovery. Our results suggest that at the time when the T4/T1 ratio = 0.7 in the orbicularis muscle, the T4/T1 ratio in the hypothenar muscle was significantly less and was at a lvel where respiratory impairment may exist. Our findings are consistent with Caffrey *et al.*'s study[3] comparing orbicularis oculi muscles with hypothenar muscles, and confirm that (1) recovery of neuromuscular blockade in the facial muscle groups precede the hypothenar muscles, (2) the TOF stimulation of the facial muscle group may not be an accurate monitor to determine the return of adequate respiratory function.

References

1 *Ali HH, Wilson RS, Savarese JJ et al.* The effect of tubocurarine on indirectly elicited TOF response and respiratory measurements in humans. Br J Anaesth 1975; 47: 570.

2 *Sharpe MD, Lam AM, Nicholas JF et al.* Correlation between integrated evoked EMG and respiratory function following atracurium administration. Can J Anaesth 1987; 34: S77.

3 *Caffrey RR, Warren ML, Becker Jr.* Neuromuscular blockade monitoring comparing the orbicularis oculi and adductor pollicis muscles. Anesthesiology. 1986; 65: 95.

The influence of resting muscle tone on systemic oxygen consumption during hypothermic cardiopulmonary bypass

C.L. Irish, J.M. Murkin, A. Cleland, J.L. MacDonald, R. Mayer

Department of Anaesthesia and Clinical Perfusion Services, University Hospital, University of Western Ontario, London, Ontario

During cardiopulmonary bypass (CPB), systemic oxygenation is provided by the pump oxygenator. The performance characteristics of membrane oxygenators indicate a maximal oxygen transfer rate of 55 $ml \cdot min^{-1}L^{-1}$ at a perfusion flow rate of 5 $L \cdot min^{-1}$ and 2:1 gas to blood ratio (Bentley BCM-7). Patients with a very large body surface area (BSA) may thus be at risk of incurring an oxygen debt during CPB.[1] This study was designed to investigate the influence of resting muscle tone on systemic oxygen consumption during hypothermia.

Methods

After institutional review board approval, five patients undergoing cardiac surgery utilizing hypothermic cardiopulmonary bypass (CPB) were enrolled in the study. After premedication with lorazepam 0.06 $mg \cdot kg^{-1}$ PO and morphine 0.10 $mg \cdot kg^{-1}$ IM, patients were anaesthetized with sufentanil 15 $\mu g \cdot kg^{-1}$ and intubated after administration of d-tubocurarine 3 mg and succinylcholine 1.5 $mg \cdot kg^{-1}$. Thereafter patients were maintained on air/oxygen with increments of sufentanil as needed for haemodynamic stability without administration of additional muscle relaxants or inhalational agents. Neuromuscular function was assessed using an integrated evoked electromyograph (Datex NMT-221) with a supramaximal train-of-four (T4) stimulus (squarewave pulse 2 msec duration). Once a stable rectal temperature (RT) was obtained during hypothermic CPB, pump flow rate and RT were recorded, and arterial and mixed venous blood was obtained for blood gas analysis and haemoglobin concentration. Either pancuronium 0.15 $mg \cdot kg^{-1}$ ($n = 3$) or succinylcholine 1.5 $mg \cdot kg^{-1}$ ($n = 2$) was then administered and repeat blood samples were drawn following complete suppression of T4. Data were analyzed using a paired t test with $p < 0.05$ required for significance.

Results

The patients, four of whom were male, were 44 ± 9 yr old, with calculated BSA of 1.86 ± 0.12 m^2, perfused at an average indexed flow rate of 2.4 ± 0.2 $L \cdot min^{-1} \cdot m^{-2}$, and at an average rectal temperature of 29.7 ± 0.4°C – none of which differed significantly during the two measurements made at hypothermia. Initial systemic oxygen consumption indexed to BSA

($\dot{V}O_2I$) was 53 ± 4 ml · min^{-1} · m^{-2}, which was significantly reduced to 46 ± 2 ml · min^{-1} · m^{-2} after administration of muscle relaxants and loss of T4 ($p < 0.05$). The latter result is similar to that previously reported.[2]

Discusson

This study demonstrates that in the unconscious, unmoving patient during hypothermia, resting muscle tone makes a significant contribution to $\dot{V}O_2I$. These results are in contradistinction to those reported in normothermic sedated infants[3] and may represent either methodological differences or the influence of hypothermia on resting muscle tone. As we have shown, profound neuromuscular blockade is an important adjunct during CPB, reducing VO_2I approximately 15 per cent.

References

1 *Hill GH, MacManus Q, Lefrak EA.* Cardiopulmonary bypass in the massive patient. Proceed Am Acad Cardiovasc Perf 1984; 5: 173–6.

2 *Murkin JM, Farrar JK, Tweed WA, Guiraudon G, McKenzie FN.* Cerebral versus systemic oxygen consumption during high-dose narcotic anaesthesia for cardiac surgery. Can Anaesth Soc J 1985; 32: S63–4.

3 *Palmisano BW, Fisher DM, Willis MM, Gregory GA.* Effect of paralysis on oxygen consumption in infants after cardiac surgery. Anesthesiology 1983; 59: A140.

Bradycardia after sufentanil and vecuronium

J.A.R. Dobson, J.M. Davies, G.H. Hodgson

Foothills Hospital, University of Calgary, Calgary, Alberta and Plains Health Center, University of Saskatchewan, Regina, Saskatchewan

Case reports document bradycardia and asystole in patients receiving sufentanil and vecuronium. This study was designed to measure the incidence and severity of bradycardia after this combination of drugs and to assess the effect on heart rate of priming with three non-depolarizing muscle relaxants: vecuronium, pancuronium and gallamine.

Methods

One hundred and nineteen unpremedicated ASA physical status I or II patients undergoing elective surgery were studied. Of these, 107 were randomly assigned to one of three groups and received either vecuronium 20 μg · kg^{-1} (Group I), pancuronium 20 μg · kg^{-1} (Group II) or gallamine 200 μg · kg^{-1} (Group III). Those with a pre-induction heart rate less than 61 (n = 12) were allocated to a separate group and received glycopyrrolate 0.2 mg IV before a priming dose of vecuronium. After priming, anaesthesia was induced with sufentanil (100 μg · kg^{-1} · hr^{-1} of surgery) over 1–2 minutes, thiopentone as required and vecuronium (100 μg · kg^{-1}, and maintained with nitrous oxide 60 per cent in oxygen and isoflurane as necessary. Atropine for bradycardia was given when deemed clinically necessary. Data were analyzed by unpaired t tests and a p value < 0.05 considered significant.

TABLE I Heart rate response (mean beats per minute ± 2 SEM)

Group	Baseline	Induction		Maintenance
		Minimum	Maximum	minimum
1 (Vec)	82 ± 6.4	58 ± 4.2*	86 ± 7.2	55 ± 3.2
2 (Panc)	81 ± 4.7	65 ± 3.8	92 ± 5.8	55 ± 3.0
3 (Gall)	76 ± 3.1	67 ± 3.6	88 ± 4.6	58 ± 3.5

* = $p < 0.05$.

TABLE II Atropine requirements

Group	n	Atropine given	% Needing atropine
1 (Vec)	35	7	20.0*
2 (Panc)	36	2	5.6
3 (Gall)	36	1	2.8

* = $p < 0.05$.

Results

Patients in the three groups were comparable with respect to age, sex, weight, haemoglobin, resting heart rate and blood pressure. Heart rates slowed during the induction period in all groups, but minimum heart rates in Group I were significantly slower than in the other groups (Table I). Heart rates were stable during maintenance. The clinical need for atropine was significantly greater in Group I (Table II). In the 12 patients with a pre-induction bradycardia, prophylaxis with glycopyrrolate did not prevent the further need for atropine in seven patients.

Discussion

Our results suggest that clinically significant bradycardia is common, and occasionally severe, in healthy patients receiving sufentanil and vecuronium. However, priming with pancuronium or gallamine greatly decreases the need for atropine, without producing tachycardia. Patients with a pre-induction bradycardia may not be suitable for this combination of agents.

References

1 *May JR.* Vecuronium and bradycardia. Anaesthesia 1985; 40: 710.

2 *Starr NJ, Sethna DH, Estafanous FG.* Bradycardia and asytole following the rapid administration of sufentanil with vecuronium. Anesthesiology 1987; 64: 521–3.

The use of lidocaine for femoral block in suspected MH patients

J. Tough, C.B. Cattran, K. Nolan, G. Allen

Department of Anaesthesia, Ottawa Civic Hospital and University of Ottawa, Ottawa, Ontario

The amide-linked local anaesthetic agents have been implicated as potential triggering agents in malignant hyperthermia susceptible (MHS) patients on mainly theoretical grounds.[1,2] A number of clinical trials have been performed using amide-

TABLE Demographic variables (mean ± SD)

	Number	*Age (yr)*	*Weight (kg)*	*Sex*
Cases	17	34.82 ± 3.13	69.29 ± 2.92	F53%
Controls	6	29.33 ± 1.65	73.12 ± 7.76	F83%

linked local anaesthetics in MHS patients with no adverse reactions.[3] This study was undertaken to show that there are no subtle or subclinical biochemical changes during the use of these drugs in MHS patients.

Methods

Twenty-three MHS patients undergoing diagnostic muscle biopsy (vastus lateralis) were enrolled in the study after giving informed consent. All patients were admitted to hospital and underwent preoperative evaluation which included a history and physical examination, CBC, urinalysis, CPK, and ECG. Premedication consisted of diazepam 0.15 mg · kg^{-1} PO or lorazepam 2–4 mg PO, and morphine 0.15 mg · kg^{-1} IM.

On arrival in the operating room, a large bore intravenous was started. Patients were monitored by ECG, BP cuff, radial arterial line, pulse oximeter, and axilliary temperature probe. All equipment for the treatment of an MH crisis was always readily available.

A femoral and lateral femoral cutaneous nerve block were performed using one per cent lidocaine 5 mg · kg^{-1}. Arterial blood gases (ABG), venous lactate, and CPK levels were drawn prior to induction of regional anaesthesia and every 30 minutes thereafter during surgery. Upon completion of surgery, the patients were transferred to the recovery room and vital signs (PR, RR, core temperature) were monitored for four hours. ABG, CPK, and lactate levels were drawn every 60 minutes.

To examine the effect of potential MH over time, a repeated measures analysis of variance (ANOVA) was carried out. Significant time effects were subsequently analysed using the Tukey multiple range test.[4] Statistical differences are all at $p < 0.05$ unless otherwise stated.

Results

Seventeen of the 23 patients had a positive muscle biopsy. None of the patients developed clinical signs of an MH reaction.

The Table shows the demographic variables. Analysis indicated no significant differences between cases and controls in age or weight.

The results of the analysis of variance indicated no significant differences between the cases and controls for any of the variables we examined. However, significant time differences were noted for all variables except HCO_3^-.

Discussion

Our data provide further evidence that the use of amide type local anaesthetics is safe in patients with MHS. The statistical analysis revealed no significant differences between the MHS and control groups in any of the clinical or biochemical variables. There were, however, significant time-related differences. Note that these time-related changes occurred in a similar fashion and degree in both the MHS and control groups and, therefore, are not indicative of an MH reaction. Although the number of patients in this study is small, it supports other similar studies and provides further evidence that amides may be safely used in MHS patients.

References

1 *Wingard DW, Bobko S.* Failure of lidocaine to trigger porcine malignant hyperthermia. Anesth Analg 1979; 58: 99–103.
2 *Britt BA.* Malignant hyperthermia. A pharmacogenetic disease of skeletal and cardiac muscle. N Engl J Med 1974; 1140–1.
3 *Berkowitz A, Rosenberg H.* Femoral block with mepivacaine for muscle biopsy in malignant hyperthermia patients. Anesthesiology 1985; 62: 651–2.
4 *Kramer, CY.* Extension of the multiple range tests to group means with unequal number of replications. Biometrics 1956; 12: 307–10.

Phrenic nerve stimulation increases blood flow and decreases succinylcholine onset time in the dog diaphragm

R.F. Erian, F. Donati, D.R. Bevan
Departments of Anaesthesia, Royal Victoria Hospital and McGill University, Montreal, Quebec

An increase in muscular activity is accompanied by an increase in muscle blood flow,[1] suggesting that a train-of-four (TOF) or tetanic stimulation would have similar effects. As a result, they may effect neuromuscular blockers onset time by promoting delivery of the drug to muscle.[2] The purpose of this study, therefore, was to determine if stimulation of the diaphragm prior to administration of a muscle relaxant is accompanied by a more rapid and pronounced effect.

Methods

Four anaesthetized mechanically ventilated dogs were studied. Blood flow to the diaphragm was measured using electromagnetic flow probes in the left phrenic and internal mammary arteries. Diaphragmatic activity was measured using transdiaphragmatic pressure (Pdi). Time of onset was measured from injection to the time a specific percentage of twitch depression was attained. Blood flow, time of onset and Pdi were examined under three conditions: rest, train-of-four, and tetanic stimulation. Each animal was started randomly with one of the three conditions and followed successively with the other two, with time allowed between runs for Pdi to return to control. Succinylcholine (50 μg · kg^{-1}, IV) was administered alternately at rest, after a 2-min period of TOF, or a 2-min period of tetanic stimulation. Results are expressed as mean ± SEM, with comparisons made using a paired Student's t test. A p value < 0.05 was considered statistically significant.

Results

At constant blood pressure and acid base balance, phrenic artery blood flow increased to 141 ± 8 per cent of control during TOF and to 211 ± 14 per cent of control during tetanus ($p < 0.01$). Internal mammary blood flow increased to 120 ± 5 per cent and 128 ± 8 per cent of control during TOF and tetanus, respectively ($p < 0.05$). The time to 50 per cent twitch depression (T.5)

decreased from 68.9 ± 6.4 seconds at rest to 31.3 ± 5.1 seconds during TOF ($p < 0.01$) and 25.8 ± 3 seconds during tetanus ($p < 0.01$). Maximal twitch depression produced by succinylcholine was 32.1 ± 5.4, 16.2 ± 1.0 and 9.0 ± 1.5 per cent of control during rest, TOF and tetanus, respectively ($p < 0.01$).

Discussion

This study showed that an increase in diaphragmatic activity was accompanied by a significant decrease in the time of onset and increase in the intensity of twitch depression. Furthermore, this increase in diaphragmatic activity was associated with a significant increase in blood flow proportional to the degree of activity. Although an increase in muscle activity with tetanic stimulation further increased blood flow and depth of twitch depression, the time to 50 per cent twitch depression (T.5) was not statistically different from that of TOF stimulation. These results indicate that drug delivery is inevitably increased from TOF to tetanic stimulation, but the lack of a statistically significant change in T.5 is due to the relative insensitivity of our technique. This becomes apparent when one takes into account: the increased circulation time/onset time ratio, the short onset times of succinylcholine and the further reduction in onset times caused by an increased blood flow as compared with resting. As a clinical application of these results, one can speculate that the lower sensitivity of the diaphragm to muscle relaxants can be compensated for by increasing diaphragmatic activity through hyperventilation or spontaneous breathing immediately prior to administration of the muscle relaxant. This is especially important if one wants to obtain maximal diaphragmatic paralysis as rapidly as possible without increasing the dose of muscle relaxant.

References

1 *Anrep GV, Blalock A, Samaan A.* The effect of muscular contraction upon blood flow in the skeletal muscle. Proc R Soc Lond Biol Sci 1934; 1214: 223–4.

2 *Goat VA, Yeung ML, Feldman SA.* The effect of blood flow upon the activity of gallamine triethiodide. Br J Anaesth 1976; 48: 69–73.

Mouth opening changes following muscle relaxation in an animal

A.F.L. VanDerSpek, P.I. Reynolds, J.A. Ashton-Miller, C.J. Stohler

University of Michigan, Ann Arbor, Michigan.

In a study in humans, a reduction of mouth opening, induced by a constant force, during halothane anaesthesia was associated with the administration of succinylcholine, but not with vecuronium.[1] This reduction occurred during limb muscle flaccidity and loss of neurally evoked muscle responses. The purpose of this study was to determine whether this mouth opening could be observed in other animals.

Methods

This study was approved by the University of Michigan committee on animal use. Anaesthesia was induced in six healthy, adult cats with the inhalation of halothane with oxygen in an induction chamber. Food or water were not withheld; no antibiotics were administered. When the cats lost their whisker reflex and response to paw pinch, they were removed from the induction chamber and placed in the dorsal recumbent position. Under direct laryngoscopy, the vocal folds were sprayed with benzocaine and the trachea was intubated. Ventilation was controlled: electrocardiograph leads, temperature probe, end-tidal anaesthetic and carbon dioxide monitors were placed and their values were recorded. An incision was performed in the inguinal region and the femoral artery and vein were cannulated. The ischiatic nerve was stimulted percutaneously with needle electrodes by a Grass S88 stimulator via a stimulus isolation unit with supramaximal, square wave stimuli of 0.2 millisecond duration, at a frequency of 1 Hz while flexion of the paw (twitch) was observed. Arterial blood pressure and twitch were recorded. At stable end-tidal anaesthetic concentration (2.5 per cent), the head was fixed in a "sniffing" position with sand bags and restraints. Mouth opening was accomplished by means of a constant force (0.835 Newton) spring which was connected to the mandibular canines. The opening force was directed at a 90° angle to the mandibular plane in the mid-sagittal plane. The resulting separation of the maxillary and mandibular incisors was measured in mm in the sagittal plane. After baseline measurements were taken, the muscle relaxants were injected intravenously. Succinylcholine ($0.3\ mg \cdot kg^{-1}$) or vecuronium ($0.1\ mg \cdot kg^{-1}$) was randomly assigned, each to three cats. Fasciculations, loss and return of the first and full twitch were noted. Mouth opening was measured at 30 sec intervals for up to 10 min, followed by measurements at 1 min interval for 20 min. Absolute and relative mouth opening before and after muscle relaxant administration were compared by Mann-Whitney U analysis.

Results

There was no statistical difference between the two groups with respect to weight. Mouth opening reduced significantly ($p < 0.05$) in the cats receiving succinylcholine; those receiving

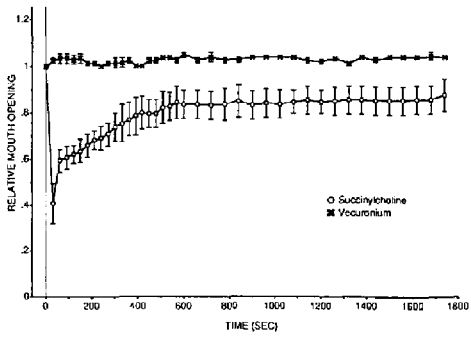


FIGURE Changes in relative mouth opening after muscle relaxation during halothane anaesthesia in six cats.

vecuronium did not. Mouth opening reduction was present after fasciculations, during limb muscle flaccidity and after recovery from succinylcholine-induced relaxation. Mean (±SE) relative changes from baseline mouth opening (1.0) of three cats for each relaxant have been graphed in the Figure.

Discussion

The reductions of mouth opening observed in this study are comparable to those observed in children.

Reference

1 *Van Der Spek AFL, Fang WB, Ashton-Miller JA, Stohler CS, Carlson DS, Schork Ma.* The effects of succinylcholine on mouth opening. Anesthesiology 1987; 67: 459–65.

Differential effects of pancuronium on masseter and adductor pollicis muscles in humans

C.E. Smith, F. Donati, D.R. Bevan
Departments of Anaesthesia, Royal Victoria Hospital and McGill University, Montréal, Québec

The muscles of the upper airway must be relaxed to obtain adequate intubating conditions, and must recover completely to avoid upper airway obstruction. The masseter, which closes the jaw, probably plays an important role in keeping airway patency. This study was designed to investigate the potency of pancuronium at the masseter and adductor pollicis muscles.

Methods

After institutional approval and informed consent, ten adult patients scheduled for elective surgical procedures were studied. Anaesthesia was induced with thiopentone 3-5 $mg \cdot kg^{-1}$ and enflurane 1.5–2.5 per cent end-tidal, and maintained with N_2O 66 per cent in O_2 and fentanyl 1–2 $\mu g \cdot kg^{-1}$. After tracheal intubation, enflurane was discontinued, and ventilation was controlled to maintain an end-tidal CO_2 of 30–35 mmHg. Supramaximal train-of-four stimulation was applied to the ulnar nerve at the elbow and to the nerve to the masseter, which originates from the mandibular branch of the trigeminal nerve below the zygomatic arch, anterior to the mandibular condyles. A force transducer system was attached to both an oral airway and a metal frame fixed to the operating table 10 cm caudad to the chin. Cumulative dose–response curves for first twitch height were obtained with pancuronium, 0.02 $mg \cdot kg^{-1}$, followed by 0.01 $mg \cdot kg^{-1}$ increments. Results were expressed as mean values ± SEM, and comparisons were made using the paired Student's t test.

Results

The end-tidal enflurane concentration was less than 0.3 per cent in all patients during the study period. Control tensions were 473 ± 75 g and 660 ± 118 g at the masseter and adductor pollicis, respectively. The masseter was more sensitive to pancuronium than the adductor pollicis (Figure). The ED50s were 0.025 ± 0.001 and 0.028 ± 0.001 $mg \cdot kg^{-1}$ for the masseter and adductor pollicis respectively ($p < 0.05$). Corresponding figures

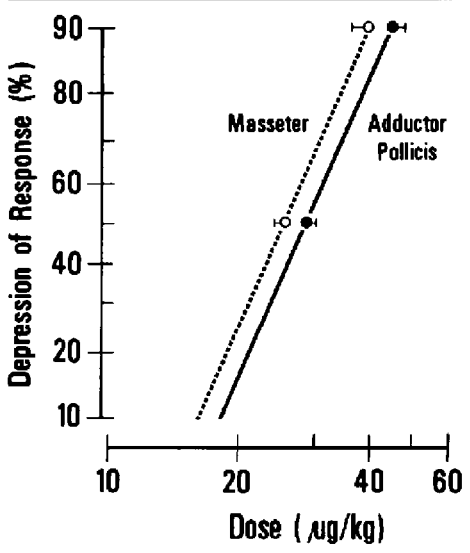


FIGURE First twitch depression versus pancuronium dose at the masseter and the adductor pollicis muscles. Bars represent SEM for ED50 and ED90.

for the ED90s were 0.038 ± 0.004 and 0.043 ± 0.002 $mg \cdot kg^{-1}$, respectively ($p < 0.05$). The time from injection of the initial dose to maximum masseter blockade was 3.2 ± 0.2 min compared with 3.8 ± 0.2 min at the adductor pollicis ($p < 0.01$). After the incremental doses, time to maximum blockade was 1.8 ± 0.1 and 2.6 ± 0.1 min, respectively ($p < 0.01$). A similar degree of train-of-four fade was observed both in muscles at comparable first twitch blockade.

Discussion

The present study demonstrated that, compared with the adductor pollicis, the masseter muscle displays increased sensitivity and faster onset of blockade after injection of pancuronium. These findings support the observation that upper airway muscles are relatively sensitive to non-depolarizing relaxants.[1] Thus, clinically used doses of pancuronium are expected to provide excellent jaw relaxation, but may be associated with post-operative upper airway obstruction.

Reference

1 *Gal TJ, Goldberg SK.* Diaphragmatic function in healthy subjects during partial curarization. J Appl Physiol 1980; 48: 921–6.

Postoperative curarization – a comparison between atracurium, vecuronium and pancuronium

D.R. Bevan, C.E. Smith, F. Donati
Departments of Anaesthesia, Royal Victoria Hospital and McGill University, Montréal, Québec

In Denmark[1] and Australia[2] train-of-four ratios of less than 70 per cent occurred in 42 and 21 per cent of patients on arrival in the PAR. These results were obtained before the introduction of atracurium and vecuronium; neuromuscular monitoring was not used during anaesthesia and muscle relaxants were used in larger doses. The present study was designed to determine the incidence of residual curarization in a Canadian environment.

Methods

One hundred and fifty unselected adult patients were studied as part of an anaesthetic audit. On the days chosen for study all patients were included who had received pancuronium, atracurium or vecuronium and who were expected to resume spontaneous breathing after surgery. The choice of premedication, anaesthetic, muscle relaxant and reversal agent was made by the anaesthetist who was unaware that the patient would be assessed.

Upon arrival in the PAR the force of contraction of the adductor pollicis muscle was recorded after train-of-four (TOF) stimulation of the ulnar nerve every 12 sec. Clinical assessment (head lift, hand grip, tongue protrusion, eye opening) was made in patients who were awake and cooperative. Responses were recorded as "weak" or "normal."

Results

Patients receiving pancuronium had significantly longer duration of anaesthesia, lower TOF and greater incidence of TOF < 70 per cent than patients receiving atracurium or vecuronium (Table). Of the 24 patients demonstrating TOF of < 70 per cent, neuromuscular monitoring had been used in 23, and the weakness was not related to choice of anaesthetic (enflurane or isoflurane) or reversal agent (edrophonium or neostigmine). Six of the 18 awake patients who had TOF of < 70 per cent showed a weak response to at least one clinical test.

Discussion

Impaired neuromuscular activity after anaesthesia was found in 36 per cent of patients who had received pancuronium but the incidence was reduced when the relaxant was atracurium (4 per cent) or vecuronium (9 per cent). The results suggest that use of intermediate relaxants is accompanied by improved recovery of neuromuscular function. Careful objective measurement of neuromuscular function correlated poorly with simple clinical tests and clinical monitoring during anaesthesia was a poor predictor of recovery from paralysis.

TABLE Results

	n	*Dose* $mg \cdot kg^{-1} \cdot hr^{-1}$	*Duration of block (min)*	*TOF (%)*	*TOF <70% (n)*
Pancuronium	47	0.03 ± 0.002	146.9 ± 10.7	74.4 ± 2.7	17
Atracurium	46	0.37 ± 0.03	101.0 ± 9.1	93.2 ± 1.1	2
Vecuronium	57	0.06 ± 0.004	106.3 ± 7.5	89.1 ± 1.9	5

References

1 *Viby-Morgensen J, Jorgensen BC, Ording H.* Residual curarization in the recovery room. Anesthesiology 1979; 50: 539–41.
2 *Beemer GH, Rosenthal P.* Postoperative neuromuscular function. Anaesth Intensive Care 1986; 14: 41–5.

Role of circulation time in the modelling of the onset of atracurium neuromuscular blockade

F. Donati, D.R. Bevan
Departments of Anaesthesia, Royal Victoria Hospital and McGill University, Montréal, Québec

The onset time of neuromuscular blockade is usually not less than 1–2 min, even with large doses of relaxant.[1] This suggests that circulation time is important. This theoretical and experimental study was designed to test the hypothesis that incorporating circulation time into a pharmacokinetic analysis with an "effect" compartment[2] might improve predictions of onset times.

Methods

The study was approved by the Hospital Ethics Committee. After giving informed consent, 63 adults scheduled for elective surgery were given atracurium, 0.07, 0.1, 0.13, 0.21, 0.42, or 0.6 $mg \cdot kg^{-1}$, during stable thiopentone-N_2O-halothane anaesthesia. Evoked force of contraction of the adductor pollicis muscle was recorded with train-of-four stimulation of the ulnar nerve every 12 sec. Time from injection of the drug to 5 per cent, 95 per cent and maximum first twitch depression were measured, and dose–response relationships obtained. Calculations of predicted onset profiles were based on atracurium pharmacokinetic data.[3] The relationship between concentration and effect was derived assuming that access of the drug to the effect compartment was not possible until after a certain circulation time, which was varied between 0 and 1 min. Comparisons were made between predicted and measured values.

Results

The ED50 and ED95 were 0.105 and 0.232 $mg \cdot kg^{-1}$ respectively. For doses of 0.07–0.13 $mg \cdot kg^{-1}$, measured time to maximum block was 6.6 ± 0.3 min (SEM), compared with predicted values of 6.1–7.5 min, depending on circulation time. Time to 5 and 95 per cent blockade decreased markedly with increasing dose (Figure). With 0.6 $mg \cdot kg^{-1}$, these were 0.74 ± 0.05 and 2.2 ± 0.3 min respectively, much longer than the predicted values of 0.15 and 0.9 min, respectively, obtained with a circulation time of zero. The best agreement between predicted and measured values was obtained with calculations including a circulation time of 36 seconds (Figure).

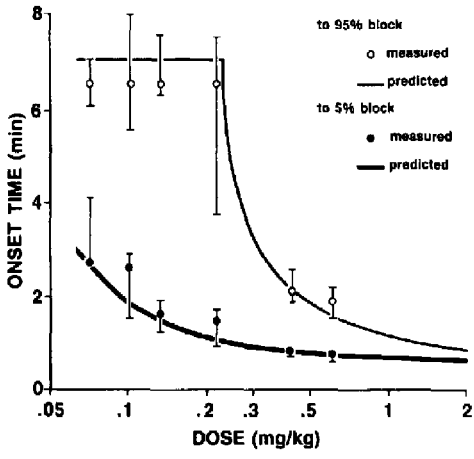


FIGURE Time to 5 and 95 per cent (or to maximum if 95 per cent not attained) blockade versus atracurium dose. Median ± quartiles are shown. Lines represent predicted values for a circulation time of 36 seconds.

Discussion

The introduction of a circulation time accounts for the following observations: (1) time to 95 per cent blockade decreases markedly in the range one to three times ED95, and (2) beyond three times ED95, onset time is reduced little by increasing the dose because circulation time is the major factor affecting onset of neuromuscular blockade. This explains why doses as large as eight times ED95 have failed to produce onset times which are much shorter than with three times ED95.[3]

References

1 *Casson WR, Jones RM.* Vecuronium induced neuromuscular blockade. The effect of increasing dose on speed of onset. Anaesthesia 1986; 41: 354–7.

2 *Sheiner LB, Stanski DR, Vozeh S, Miller RD, Ham J.* Simultaneous modeling of pharmacokinetics and pharmacodynamics: application to d-tubocurarine. Clin Pharmacol Ther 1979; 25: 358–71.

3 *Weatherley BC, Williams SG, Neill EAM.* Pharmacokinetics, pharmacodynamics and dose-response relationships of atracurium administered i.v. Br J Anaesth 1983; 55: 39S–45S.

Atracurium-vecuronium combinations: lack of potentiating effects

F. Donati, J.M. Katz, C.E. Smith, D.R. Bevan
Departments of Anaesthesia, Royal Victoria Hospital and McGill University, Montréal, Québec

The potentiation of metocurine and pancuronium neuromuscular blockade may be useful because of avoidance of cardiovascular side-effects.[1] In the case of atracurium and vecuronium, a potentiating combination would be useful to reduce cost. This study was designed to determine the potency of atracurium and vecuronium, when given separately, or in combination.

TABLE Potency of atracurium, vecuronium, and combination (expressed as $\mu g \cdot kg^{-1}$ ± standard error of estimate for the mean). The combination is expressed in vecuronium equivalents. The ratio refers to the actual potency of the combination relative to that of the expected potency in the absence of any potentiation.

Agent	*ED50*	*ED90*	*ED95*
Atracurium	116 ± 6	175 ± 9	201 ± 10
Vecuronium	23 ± 1	34 ± 2	39 ± 2
Combination	20 ± 3	38 ± 5	47 ± 6
Ratio	1.15	0.89	0.83

Methods

The protocol was approved by the Hospital Ethics Committee. Fifty-four adult patients, ASA physical status I or II, who were free of neuromuscular, hepatic or renal disease, were given a thiopentone-fentanyl-nitrous oxide anaesthetic. The force of contraction of the adductor pollicis muscle was recorded following train-of-four stimulation of the ulnar nerve every 12 sec. Thirty-six patients were given either atracurium, 0.075, 0.1 or 0.15 $mg \cdot kg^{-1}$, or vecuronium, 0.015, 0.02, or 0.03 $mg \cdot kg^{-1}$, on a random basis. Linear regression of the logit transformation of maximum first twitch depression versus the logarithm of the dose was calculated to determine equipotent doses. Then, 0.035, 0.05 or 0.065 $mg \cdot kg^{-1}$ atracurium plus an equipotent dose of vecuronium was administered to 18 patients. The potency of the mixture was expressed as "vecuronium equivalents," defined as twice the dose of vecuronium in the combination, and compared with the calculated potency assuming no potentiation. Results are expressed as means ± standard error of estimate for the mean. Student's t test was applied between predicted and measured values.

Results

The ED50, ED90, and ED95 of atracurium and vecuronium are shown in the Table. Vecuronium was found to be five times as potent as atracurium. The potency of the combination was between 0.83 to 1.15 times that of either drug administered separately (Table). The difference was not statistically significant ($p > 0.05$).

Discussion

The hypothesis that atracurium and vecuronium potentiate each other is not supported by this study. The mechanism for the potentiation between certain relaxants is uncertain. However, it was expected that atracurium and vecuronium, whose chemical structures are analogous to those of metocurine and pancuronium respectively, might have retained their mutually potentiating properties. Therefore the results suggest that potentiation of relaxants is affected by minor changes in their structure. It also appears that mixtures of atracurium and vecuronium have few practical applications.

Reference

1 *Lebowitz PW, Ramsey FM, Savarese JJ, Ali HH.* Potentiation of neuromuscular blockade in man produced by combinations of pancuronium and metocurine or pancuronium and d-tubocurarine. Anesth Analg 1980; 59: 604–9.

Regional cerebral blood flow following haemorrhagic hypotension superimposed on deep isoflurane anaesthesia: comparison of techniques to support blood pressure

T.S. Ruta, W.A.C. Mutch

Department of Anesthesia, University of Manitoba, Winnipeg, Manitoba

Controlled hypotension with deep isoflurane is frequently employed during cerebral aneurysm surgery. Little information is available on how haemorrhagic hyotension superimposed on deep isoflurane anaesthesia affects regional cerebral blood flow (rCBF). Such a situation may occur clinically with rupture of a cerebral aneurysm. This study was undertaken to investigate the optimal method of preserving rCBF following haemorrhage. A comparison of prehaemorrhagic rCBF values was made with those determined after restoration of mean blood pressure (BP) to controlled hypotensive levels with either (1) phenylephrine, (2) reinfusion of blood ± normal saline (NS) or (3) decreased inspired isoflurane concentration.

Methods

Twenty-one New Zealand white rabbits weighing 2–2.5 kg were induced with five per cent isoflurane O_2, paralyzed with succinycholine, intubated and ventilated. End-tidal CO_2 was continuously monitored to maintain $PaCO_2$ between 35–40 mmHg. Maintenance anaesthesia was one per cent isoflurane with pancuronium for paralysis. Catheters were inserted into two femoral arteries (FA), one femoral vein and the left atrium (LA). Intracranial pressure (ICP) was measured from the cisterna magna. Four measurements of rCBF were determined by the radioactive microsphere technique.[1] LA injection of 500,000 15 μm diameter microspheres were made with ^{141}Ce, ^{51}Cr, ^{85}Sr, and ^{46}Sc. A reference blood sample was withdrawn from the FA. Brain and blood reference counts were measured by gamma counter and converted to rCBF by computer program. Three groups of seven animals were studied. Measurements of rCBF were made after the BP had stabilized for a period ≥15 min. Huemodynamic manipulations and experimental techniques were identical for the first three flows of each group. Flow 1: one per cent inspired isoflurane. Flow 2: increased inspired isoflurane concentration ≥2 per cent for controlled hypotension to a mean BP ~ 50 mmHg. Flow 3: haemorrhagic hypotension by bleeding from the FA line to a mean BP ~ 30 mmHg. Flow 4: BP returned to a mean BP ≅ 50 mmHg by one of three techniques. Group I: Phenylephrine infusion. Group II: Reinfusion of autologous blood ± NS. Group III: Decreasing the inspired isoflurane concentration. Within group comparisons were by ANOVA for repeated measures (RMANOVA), between group comparison by ANOVA with Duncan's test applied post-hoc; $p < 0.05$ was considered statistically significant.

TABLE Results (Mean ± SEM)

	BP (mmHg)	*ICP (mmHg)*	*$PaCO_2$ (mmHg)*	*rCBF (ml·gm^{-1}·min^{-1})*	
				LPF	*LCF*
Group I (phenylephrine) n = 7					
Flow 1	80.0 ± 4.5†	4.2 ± 0.9	37.4 ± 0.4	0.86 ± 0.11	1.13 ± 0.16*
2	48.7 ± 1.4	4.7 ± 0.7	37.6 ± 0.6	0.73 ± 0.11	1.61 ± 0.26
3	28.4 ± 0.9†	3.1 ± 0.2†	37.1 ± 0.5	0.52 ± 0.04†	1.10 ± 0.13*
4	50.0 ± 1.2	3.6 ± 0.7†	36.1 ± 0.5	0.84 ± 0.09	1.80 ± 0.28
Group II (reinfusion) n = 7					
Flow 1	87.6 ± 3.6†	3.8 ± 0.7*	35.4 ± 0.4	0.90 ± 0.08	0.98 ± 0.09†
2	49.0 ± 1.2	4.5 ± 0.7	37.0 ± 0.4	0.71 ± 0.07	1.73 ± 0.19
3	31.3 ± 1.2†	2.7 ± 0.6†	37.2 ± 0.8	0.59 ± 0.07	1.19 ± 0.12†
4	48.8 ± 1.6	4.2 ± 0.4	37.7 ± 0.8	0.70 ± 0.08	1.47 ± 0.19
Group III (decreased isoflurane) n = 7					
Flow 1	78.9 ± 4.3†	3.2 ± 1.2	36.9 ± 0.6	0.96 ± 0.10	1.05 ± 0.09†
2	49.3 ± 0.7	3.4 ± 1.1	36.9 ± 0.2	0.81 ± 0.09	1.40 ± 0.12
3	31.9 ± 1.0†	1.2 ± 0.3†	38.4 ± 0.9	0.62 ± 0.05†	1.28 ± 0.13
4	47.9 ± 1.0	2.2 ± 0.9	36.6 ± 1.1	0.75 ± 0.09	1.09 ± 0.09*

*$p < 0.05$ within group comparisons vs Flow 2 RMANOVA.
†$p < 0.01$ within group comparisons vs Flow 2 RMANOVA.

Results

Summarized in Table. Comparisons were made of representative brain regions between flow 2 (baseline controlled hypotension, mean BP ~ 50) and flow 4 (BP supported, mean BP ~ 50). Comparisons of flow 2 vs flow 4 revealed no significant difference within or between groups for left parietal flow (LPF). LPF was significantly lower for groups I and III during flow 3. Left cerebellar flow (LCF) was significantly lower in Group II vs. Groups I and III for flow 4. However, LCF4 for Group III data was identical to LCF1.

Discussion

The results demonstrate all three methods of supporting blood pressure were equally effective in preserving LPF following haemorrhage. Decreased isoflurane concentration did not restore cerebellar flow to baseline-controlled hypotensive levels as well as phenylephrine infusion or reinfusion of blood ± NS. However, LCF4 was identical to LCF1 for this group suggesting the difference between groups is of limited clinical significance. All three techniques for support of BP may be clinically useful in the face of haemorrhage during controlled hypotension with isoflurane.

Reference

1 *Heymann MA, Payne BD, Hoffman JIE, Rudolph AM.* Blood flow measurements with radionuclide-labeled particles. Prog Cardiovasc Dis 1977; 20: 55–79.

The effect of hypotension and hypotensive technique on cerebral ischaemia and the dimension of the ischaemic penumbra

J.C. Drummond, D.J. Cole, H.M. Shapiro, M.H. Zornow
Departments of Anesthesiology of the Veterans' Administration Medical Center, San Diego, The University of California, San Diego and Loma Linda University Medical Center, Loma Linda, California

Temporary cerebral vascular occlusion may occur either electively or in urgent circumstances during intracranial vascular procedures. On occasion, induced hypotension is employed simultaneously. It is accepted that the combination should result in a larger area of ischaemia. However, the phenomenon has not been examined systematically nor has the influence of the specific hypotensive techniques been evaluated. The present study examined the extent of ischaemia and the dimension of the ischaemic penumbra followng middle cerebral artery occlusion (MCAO) during normotension and during three hypotensive regimens.

Methods

Preparation

Male Sprague-Dawley rats (weight 350–400 g) were prepared as follows. Anaesthesia was induced with three per cent isoflurane (ISO) in oxygen. After intubation, anaesthesia was maintained with 1.2 MAC ISO (1.87 per cent) in 40 per cent O_2/balance nitrogen. Femoral vessels were cannulated for blood pressure recording, isotope administration and blood collection, and the left MCA was exposed.[1] Rats were then serially assigned to one of four groups: *(1) Normotensive control* (n = 6) 1.2 MAC ISO (which yielded a mean arterial pressure (MAP) of approximately 110 mmHg); *(2) Nitroprusside* (NTP) (n = 5). During 1.2 MAC ISO anaesthesia MAP was lowered to 40–50 mmHg by infusion of NTP; *(3) Isoflurane* (n = 6). End-tidal ISO was increased as required to achieve a MAP of 40–50 mmHg; and, *(4) Hypovolemia* (n = 6). During 1.2 MAC ISO anaesthesia, aliquots of blood were withdrawn to achieve a MAP of 40–50 mmHg. In all groups, hypotension was achieved gradually and was maintained for five minutes prior to MCAO, which was performed according to Bederson *et al.*[2] using microbipolar coagulation during continuous saline irrigation.

Blood flow determination

Eight minutes after MCAO, local cerebral blood flow (l-CBF) was measured autoradiographically using ^{14}C-ido-antipyrine and the method of Sakurada *et al.*[3] Analysis was performed with a Drexel/DUMAS image processing system which had been modified to measure the percentage area of individual hemisphere sections falling within specified CBF ranges. The measured ranges were: 0–6, 6–15 and 15–23 ml · 100 g^{-1} · min^{-1}. Measurements were made in three standard coronal sections: at the level of the anterior end of the corpus callosum and 1.8 mm anterior and posterior to it. The values obtained were averaged and the data are expresed as the percentage of the cross-sectional area falling within each flow range. The flow range areas were compared using an analysis of variance. $P < 0.05$ was deemed significant.

TABLE I Physiologic parameters (±SD) at the time of CBF determination

	MAP (mmHg)	*pH*	*$PaCO_2$ (mmHg)*	*Hct (%)*	*ET ISO (%)*
Control	109 ± 13	7.40 ± 0.02	38 ± 2	45 ± 2	1.89 ± 0.08
Nitroprusside	45 ± 3	7.46 ± 0.21	40 ± 3	43 ± 2	1.95 ± 0.09
Isoflurane	44 ± 2	7.40 ± 0.04	38 ± 2	45 ± 1	3.56 ± 0.41
Hypovolaemia	43 ± 2	7.39 ± 0.01	37 ± 1	41 ± 2	1.91 ± 0.12

TABLE II Per cent (±SD) of hemisphere within CBF ranges (ml · 100 g^{-1} · min^{-1}) after MCAO

CBF Range	*Control*	*Nitroprusside*	*Isoflurane*	*Hypovolaemia*
*0–6**	*4.6 ± 4.4*	*13.7 ± 2.4*	*12.5 ± 4.0*	*14.2 ± 5.3*
6–15*	5.9 ± 3.8	11.1 ± 3.8	8.6 ± 3.3	12.4 ± 4.9
15–23	8.7 ± 4.4	8.8 ± 4.8	9.5 ± 5.3	12.1 ± 3.4

*$p < 0.02$, analysis of variance.

Results

In the control group (1.2 MAC ISO), MAP was 109 ± 13 mmHg. The requirements for hypotension (MAP 40–50 mmHg) were as follows: nitroprusside – an infusion of 7.7 ± 2.3 μg · kg^{-1} · min^{-1}; isoflurane – 3.56 ± 0.6 per cent end-tidal; Hypovolaemia – removal of 9.3 ± 1.2 ml of blood (approximately one third of total blood volume). Other than MAP, there were no between-groups physiologic differences (Table I).

Induced hypotension resulted in significantly ($p < 0.02$) larger regions falling within the two lowest flow ranges, 0–6 and 6–15 ml · 100 g^{-1} · min^{-1}, while the areas in the range of 15–23 ml · 100 g^{-1} · kg^{-1} were not different (Table II). There were no significant differences between the three hypotensive regimens.

Discussion

The CBF ranges examined were chosen to correspond to thresholds for the sequence of physiologic effects of progressively more severe cerebral ischaemia[4], i.e., 0–6 ml · 100 g^{-1} · min^{-1} – rapid neuronal injury; 6–15 ml · 100 g^{-1} · min^{-1} – delayed neuronal injury; and, 15–23 ml · 100 g^{-1} · min^{-1} – electrophysiologic dysfunction with prolonged neuronal viability. The present data indicate that during MCAO hypotension (MAP 45 mmHg) achieved by any of the three regimens studied (1.2 MAC ISO plus NTP, deep ISO, 1.2 MAC ISO plus hypovolaemia) significantly increases the area of brain tissue within the two lowest and most critical CBF ranges. The data confirm that MAP is an important determinant of CBF in an area of focal ischaemia. In addition, they provide no support for speculation that differences in the neurovascular effects of these regimens can lead to varying degrees of intracerebral shunting of blood flow. In terms of the CBF patterns observed, the data indicate no clear relative advantage to either ISO or NTP. However, differences in the cerebral metabolic state (not examined in this study) produced by these two agents may also influence neuronal survival and may constitute and advantage of ISO.

References

1 *Tamura A et al.* J Cereb Blood Flow Metab 1981; 1: 53–60.
2 *Bederson JB et al.* Stroke 1986; 17: 472–6.
3 *Sakurada O et al.* Am J Physiol 1978; 234: H59–H66.
4 *Olsen TS.* Acta Neur Scand 1986; 73: 321–37.

The effect of high frequency ventilation on cerebral blood flow and intracranial pressure in rabbits

B. Bissonnette, B.A. Hagen, C.A. Richardson, G.A. Gregory
Department of Anesthesia and the Cardiovascular Research Institute, University of California, San Francisco, San Francisco, California

The haemodynamic effects of conventional mechanical ventilation (CV) are well known. The unique haemodynamic effects of high frequency ventilation (HFV), such as decreased brain surface vessel movement, are less well understood. Several authors have studied cerebral blood flow (CBF) and intracranial pressure (ICP), but none have made these measurements across a broad range of blood pressures to determine if HFV alters cerebrovascular autoregulation and none have controlled for changes in intrathoracic volume that may occur when changing from CV to HFV. We hypothesized that HFV alters cerebrovascular autoregulation and cerebral blood flow compared to CV at the same blood pressure and intrathoracic volume.

Methods

The study was approved by the Animal Care Committee. We continuously measured systemic arterial blood pressure (SBP), ICP, and central venous pressure (CVP) during conventional and high frequency jet ventilation of six rabbits (2.5–3.5 kg) who were anaesthetized with urethane and paralyzed with metocurine. For each animal, regional cerebral blood flow of four areas of the brain was computed by H_2 washout technique.[1] Measurements were made over a systemic blood pressure range of 30–175 mmHg so that pressure dependent changes in flow (indicating changes in cerebrovascular autoregulation) could be determined: blood pressure was changed by infusing trimethaphan and/or epinephrine. A jacket plethysmograph[2] was used to continuously measure intrathoracic volume so that it could be kept constant by adjusting ventilator pressure, thus avoiding altering venous return and CVP which in turn could affect ICP and CBF. Baselne mesurements at normal blood pressure were obtained during CV (rate 35 BPM) and after changing to high frequency jet ventilaton (rate 600 BPM); jet ventilator pressure was adjusted to maintain the mean lung volume equal to that during CV by using the plethysmograph volume as guide. After measurements during HFV, the animal was returned to CV and the measurements were repeated. This sequence was repeated across the range of blood pressures for each animal.

Results

In two of the five animals, regional cerebral blood flow was always greater during HFV than during CV at matching blood pressures and intrathoracic volumes (Figure 1, regional CBF for rabbit #1). In two of the remaining three animals, regional cerebral blood flow during HFV was not always greater than during CV. However, in all five animals the ICP during HFV was greater than during CV (Figure 2, ICP for rabbit #3); this suggests that the total cerebral blood flow was greater during HFV, but the measurement of local cerebral blood flow in this instance did not reflect the increase in total cerebral blood flow. In the one remaining animal, regional cerebral blood flow during HFV was similar to CV, and ICP did not suggest a consistent change in total cerebral blood flow. Analysis of cerebral blood flow data for all animals at all blood pressures shows a statistically significant increase in cerebral blood flow during HFV compared to cerebral blood flow during CV (Wilcoxon test, $p < 0.002$).

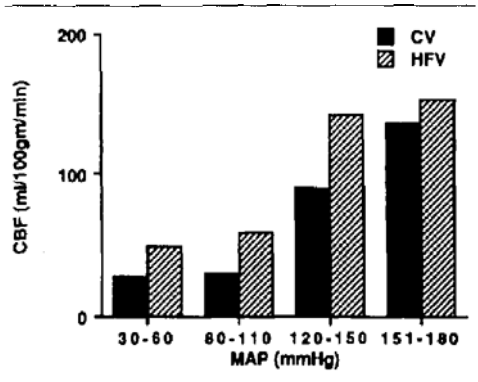


FIGURE 1 Regional cerebral blood flow, Rabbit #1.

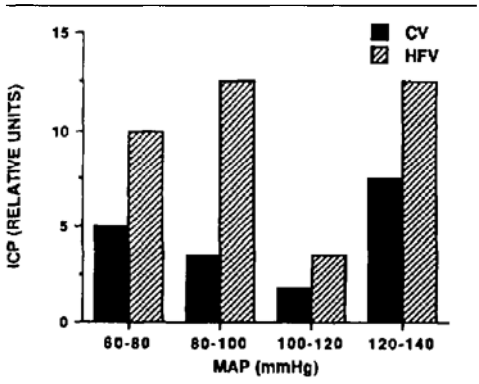


FIGURE 2 Intracranial pressure, Rabbit #3.

Discussion

These results indicate that HFV can increase total, as well as regional, cerebral blood flow when compared to CV at the same

blood pressure and intrathoracic volume (FRC). This increase in CBF (Figure 1) may not always be detected by CBF measurement, but may be reflected in an increase in ICP (Figure 2). The increased CBF may result from dilation of the cerebral vessels rather than a change in cerebrovascular autoregulation since flow during HFV was higher at all blood pressures, even during hypotension; if autoregulation was affected, flow during hypotension would decrease. Increased cerebral blood flow would be particularly dangerous in individual with diminished autoregulation (e.g., during hypercarbia), head-injured patient or neonates whose autoregulation is absent and who have fragile cerebral vessels placing them at risk for spontaneous intracranial haemorrhage.

References

1 *Aukland K, Fowler BF, Berlinger RW.* Measurement of local blood flow with hydrogen gas. Circulation Res 1964; 14: 164–87.

2 *Cartwright DW, Gregory GA, Willis MM.* A respiratory function jacket for measuring tidal volume and changes in FRC. J Appl Physiol 1983; 55: 263–6.

The effect of isoflurane on cerebral blood flow and cerebral blood volume in the hypocapnic baboon

P. Labrecque, D.P. Archer, J.L. Tyler, E. Meyer, W.F. Casey D. Trop

Montréal Neurological Institute and Royal Victoria Hospital, Montréal, Québec

Isoflurane increases cerebral blood volume (CBV) in dogs.[1] Isoflurane also increases intracranial pressure under experimental[2] and clinical conditions.[3] The effect of isoflurane on cerebral blood flow (CBF) varies with the species studied and the underlying anaesthetic conditions. In this study in a hypocapnic primate we have measured both CBF and CBV during neuroleptanaesthesia and after the introduction of isoflurane to determine whether CBF and CBV were similarly affected by isoflurane.

Methods

Four adult female baboons (15–18 kg) were studied. For each study, the animal was restrained with ketamine 10 $mg \cdot kg^{-1}$ IM. Anaesthesia was induced with thiopentone 5 $mg \cdot kg^{-1}$ and atropine 0.01 mg given intravenously. Intubation of the trachea was facilitated with succinylcholine 1 $mg \cdot kg^{-1}$ IV. Neuroleptanaesthesia was maintained with droperidol 1.3 $mg \cdot kg^{-1}$ as a single dose and fentanyl 20 $\mu g \cdot kg^{-1}$ administered as a bolus followed by an infusion of 20 $\mu g \cdot kg^{-1} \cdot hr^{-1}$. In addition to the peripheral venous line placed at induction, a femoral venous cannula was inserted for the administration of angiotensin and maintenance fluids. A femoral artery cannula was used to measure blood pressure and to obtain arterial blood samples. Following the placement of cannulae the depth of anaesthesia was verified to be adequate and muscle relaxation was induced with pancuronium 0.1 $mg \cdot kg^{-1}$, repeated as necessary to abolish spontaneous respiration. Physiologic parameters monitored included oesophageal temperature, direct arterial blood pressure, end-tidal CO_2 levels, and ECG. Infrared spectroscopic monitoring of end-tidal isoflurane concentrations guided isoflurane administration.

Global cerebral blood flow and volume were measured using radioisotope indicator dilution techniques with oxygen-15 labelled H_2O and CO respectively.[4] The tissue radioactivity concentration was measured with the Positome III positon emission tomography scanner. Blood radioactivity was determined in a well counter.

The CBF and CBV were measured during equilibrium conditions at each stage of the anaesthetic protocol: normocapnia (control), hypocapnia, isoflurane 1.3 per cent, and after washout of isoflurane. Blood pressure was maintained with an angiotensin infusion during the isoflurane administration. Following completion of the protocol the animal was allowed to emerge from anaesthesia, muscle relaxants were reversed and the animal returned to its quarters.

CBF and CBV data were analyzed by repeated measures analysis of variance. Individual differences among the groups were isolated by t test, significance if $p < 0.05$.

TABLE Results

Condition	*CBF* $ml \cdot 100\ g^{-1} \cdot min^{-1}$	*CBV* $ml \cdot 100\ g^{-1}$	*MAP* *kPa*	$PaCO_2$ *kPa*
Control	47±5	2.9±0.3	12.6±0.8	5.3±0.1
Hypocapnia	34±8*	2.5±0.1*	15.4±1.1	3.5±0.1
Isoflurane	34±11	2.9±0.5	14.1±2.3	3.6±0.2
Hypocapnia	34±8	2.4±0.1	15.0±2.5	3.6±0.2

*$p < 0.05$.

Results

Physiologic parameters were maintained within ten per cent of control values during the protocol (haematocrit, temperature, blood pressure). Hypocapnia caused a significant decrease in both CBF and CBV ($p < 0.05$); however, the introduction of isoflurane did not cause a significant increase in either CBF or CBV (Table).

Discussion

In this primate model, the administration of 1.3 per cent isoflurane did not cause a significant increase in CBF or CBV. In a similar study in dogs CBV increased.[1] Differences in the present protocol design may have reduced the sensitivity of the method to detect the 10–30 per cent increase in CBV which was anticipated. A redesigned protocol is currently underway.

References

1 *Archer DP, Labrecque P, Tyler JL, Meyer E, Trop D.* Cerebral blood volume is increased in dogs during administration of nitrous oxide or isoflurane. Anesthesiology 1987; 67: 642–8.

2 *Scheller MS, Todd MM, Drummond JC, Zornow MH.* A comparison of ICP effects of isoflurane and halothane after cryogenic brain injury in rabbits. Anesthesiology 1987; 67.

3 *Grosslight K, Roster R, Colohan AR, Bedford RF.* Isoflurane for

neuroanesthesia: risk factors for increase in intracranial pressure. Anesthesiology 1985; 63: 533–6.
4 *Phelps MR, Mazziota JC, Huang S-C.* Study of cerebral function with positron computed tomography. J Cereb Blood Flow Metab 1985; 2: 113–62.

Sufentanil anaesthesia reduces cerebral blood flow and cerebral oxygen consumption

J.M. Murkin, J.K. Farrar, W.A. Tweed
Department of Anaesthesia and Clinical and Neurological Sciences, University Hospital, University of Western Ontario, London, Ontario

Because of its favourable pharmacodynamic and pharmacokinetic characteristics, sufentanil is being increasingly used as the primary anaesthetic for major surgery. A recent report (dogs receiving 2–200 $\mu g \cdot kg^{-1}$ sufentanil) demonstrated a non-dose-related 30 per cent increase in cerebral blood flow (CBF) and no change in cerebral oxygen consumption ($CMRO_2$), raising concern about the use of sufentanil in patients with reduced intracranial comliance or at risk for cerebral ischaemia.[1] The above study, along with an absence of clinical cerebrovascular and metabolic data, prompted the following investigation in order to determine the effects of a clinically relevant dosage of sufentanil on the electroencephalogram (EEG), CBF, and $CMRO_2$ in man.

Methods

Following institutional approval and after obtaining written informed consent, five patients undergoing elective coronary artery surgery were premedicated with lorazepam 0.06 $mg \cdot kg^{-1}$ PO and morphine 0.15 $mg \cdot kg^{-1}$ 90 min prior to surgery. In addition to radial artery and pulmonary artery catheters, a 15 cm 16 Fr catheter was inserted percutaneously via the right internal jugular vein into the jugular foramen for sampling effluent jugular venous blood, 12 AgCl electrodes affixed to the scalp in a 10–20 bipolar parasagittal montage were used for recording a 10-channel EEG, and ten scintillation detectors were arrayed over the cerebral hemispheres. As previously described,[2] CBF was determined form the averaged clearance of 5–10 mCi of ^{133}Xe in 6 ml 0.9 per cent saline IV, and $CMRO_2$ was calculated as the product of the arterial-jugular O_2 content difference and mean right hemispheric CBF.

Following control haemodynamic, CBF and $CMRO_2$ determinations, 10 $\mu g \cdot kg^{-1}$ sufentanil was infused at a rate of 2 $\mu g \cdot kg^{-1} \cdot min^{-1}$ while the patient breathed 100 per cent O_2 by mask. After loss of responsiveness, 0.10 $mg \cdot kg^{-1}$ pancuronium was given and the patient was ventilated by mask until the infusion was completed. The patient was intubated and repeat haemodynamic, CBF and $CMRO_2$ measurements were commenced within 5 min of the termination of the sufentanil infusion. Data were analyzed by paired 2-tailed t test with significance at $p < 0.05$.

Results

All patients were stable during the induction. There were no significant differences between the pre- and post-induction values for $PaCO_2$, temperature, mean arterial pressure, cerebral perfusion pressure, or cardiac output. Following induction of anaesthesia, both CBF and $CMRO_2$ were significantly reduced from respectively 37.2 ± 1.7 to 27.8± 1.9 $ml \cdot 100\ mg^{-1} \cdot min^{-1}$ ($p < 0.01$) and 2.45 ± 0.19 to 1.94 ± 0.16 $ml \cdot 100\ g^{-1} \cdot min^{-1}$ ($p < 0.05$), with a corresponding increase in cerebrovascular resistance (CVR) from 2.2 ± 0.3 to 2.9 ± 0.3 U ($p < 0.005$). Following sufentanil administration, cerebral oxygen extraction ratio ($CERO_2 = CMRO_2/C_aO_2 \times CBF$) was not significantly different from that prior to induction of anaesthesia. The EEG in all patients changed from low voltage beta dominant to frontal dominant, 10–20 mV low frequency activity.

Discussion

A consistent and significant reduction of both CBF and $CMRO_2$ accompanied the production of low frequency, high-voltage EEG activity in all patients. These results are consistent with those reported for an equipotent dosage of fentanyl,[2] and along with the demonstrated increase in CVR and unchanged $CERO_2$, indicate that in this population, sufentanil anaesthesia produces cerebral vasoconstriction because of its primary metabolic depressant effects.

References

1 *Milde LN, Milde JH.* The cerebral hemodynamic and metabolic effects of sufentanil in dogs. Anesthesiology 1987; 67: A570.
2 *Murkin JM, Farrar JK, Tweed WA, McKenzie FN, Guiraudon G.* Cerebral autoregulation and flow/metabolism coupling during cardiopulmonary bypass: the influence of $PaCO_2$. Anesth Analg 1987; 66: 825–32.

The safety of awake tracheal intubation in cervical spine injury

A. Meschino, J.H. Devitt, M.L. Schwartz, J.P. Koch
Sunnybrook Medical Centre, University of Toronto, Toronto, Ontario

Six per cent of all patients admitted to our Regional Trauma Unit have an acute cervical spine and/or cord injury. Many of these patients require intubation either immediately or later during the hospitalization. At our institution, awake intubation is used for securing the airway in these patients with very rare exceptions. We have undertaken a seven-year retrospective study to demonstrate the safety of this approach.

Methods

We reviewed the records of 439 patients with cervical spine and/or cord injury admitted to our trauma unit between 1980 and 1986. Seventy patients were excluded form the study for one of the following reasons: (1) the patient was intubated under general anaesthesia (exceptionally at our institution, mainly at the referring hospital); (2) a severe head injury precluded adequate examination of spinal function; (3) a surgical airway was established; (4) the cervical spine injury was old. The remaining 369 patients were separated into two groups: Group I comprised

TABLE Mean ages and ISS scores in Groups I and II

	Group I	*Group II*	
Age (±SD)	39 ± 20	36 ± 18	p = NS
ISS (±SD)	15 ± 7	24 ± 9	p < 0.0001

233 patients with acute cervical spine and/or cord injury not requiring tracheal intubation. Group II included 136 patients with acute cervical spine and/or cord injury requiring tracheal intubation. All patients in the second group were intubated awake. Comparisons were made between initial and discharge neurological examinations under the following categories: (a) motor level; (b) sensory level; and (c) neurological grade according to a standardized cord injury scale.[1] Both groups were compared with respect to change in neurological status, mean age and mean injury severity score (ISS).

Results

Groups I and II were similar with respect to age but significantly different for severity of injury (Table).

Five out of 233 patients (2.2 per cent) in Group I had a worsening neurological status between initial examination and discharge as compared to three out of 136 patients (2.2 per cent) in Group II. A normal curve test for comparing differences between two proportions (alpha two-tailed = 0.05) failed to show a significant difference between the two groups. Post-hoc power calculations showed that this sample of 369 patients provided the study with 95 per cent statistical power to detect differences in the proportion of worsening neurological deficits when the real differences are in excess of one per cent.

Discussion

Some authors advocate the use of anaesthesia and muscle relaxants for securing the airway in patients with acute cervical spine injury.[2] The safety of that approach relies on anecdotal reports. Ideally a prospective randomized study should compare awake intubation versus intubation with general anaesthesia and muscle relaxants in patients with cervical spine injuries. Ethical considerations preclude this in the acute trauma situation. In spite of the drawbacks of our study design, our data conclusively show that awake intubation did not worsen neurological status in patients with acute cervical spine injury, although a higher ISS in the intubated group indicated a greater severity of injury. Until a study with comparable numbers can demonstrate that intubation with anaesthesia and muscle relaxants is as safe as awake intubation, we recommend awake intubation in all patients who have, or are suspected of having, an acute cervical spine injury.

References

1 *Tator CH et al.* Sunnybrook Cord Injury Scale for Assessing Neurological Injury and Neurological Recovery In Early Management of Acute Spinal Cord Injury (Seminars in Neurological Surgery Series). Raven Press Books 1982; 7–24.

2 *Doolan LA.* Traumatic injury to the cervical spine: recognition of instability and subsequent safe intubation (Abstract). Can Anaesth Soc J 1985; 32: S84.

Comparison of propofol (Diprivan) with thiopentone as induction agent for elective Caesarean section

J.L. Couper, T.P. Lombard

Department of Anaesthesiology, Medical University of Southern Africa

The ideal agent for the production of general anaesthesia for Caesarean section has still to be found. Because of propofol's ready acceptance into anaesthesia we undertook, on behalf of the manufacturers, a study comparing propofol with thiopentone for the induction of general anaesthesia for Caesarean section.

Methods

Fifty ASA physical status I or II patients scheduled for elective Caesarean section were admitted to a randomised, open study. Informed consent, Institutional and Medicines Control Council (equivalent to FDA) approval were obtained. All patients were given 30 ml 0.3 M sodium citrate orally 30 minutes prior to induction and pre-oxygenated for three minutes. Anaesthesia was induced with either thiopentone 5 $mg \cdot kg^{-1}$ or propofol (Diprivan) 2.5 $mg \cdot kg^{-1}$. Thereafter all patients were given succinylcholine 1 $mg \cdot kg^{-1}$, N_2O:O_2 50:50, halothane 0.5 per cent and alcuronium 0.3 $mg \cdot kg^{-1}$. After delivery N_2O:O_2 was changed to 66 per cent:33 per cent. Neostigmine 2.5 mg with glycopyrrolate 0.5 mg was given at the completion of surgery. Time, systolic, diastolic and mean blood pressure, heart rate and end–tidal CO_2 were recorded before and at induction, on intubation, 1 min post-intubation, at surgical incision, uterine incision, delivery of neonate, end of surgery, end of anaesthesia, and at onset of sustained spontaneous respiration. Time to awaken after anaesthesia was recorded. In neonates, Apgar scores at 1, 5 and 20 minutes, time to sustained respiration, venous cord blood pH as well as 24-hour assessment were recorded.

Results

There were no significant differences between the groups in respect of BP or HR changes. Maternal end–tidal CO_2 was kept constant for each patient. Neonates of mothers given propofol had an average Apgar score of 6.4 at 1 min (7.7 for thiopentone), 8.6 at 5 min (9.4 for thiopentone) while all infants scored 10 at 20 minutes. The propofol neonates took 2.7 min to spontaneous respiration compared with 1.6 min for thiopentone.

Discussion

The quality of awakening of the mothers given propofol was far superior to those given thiopentone. Although the propofol neonates were slower to start spontaneous respiration and had lower Apgar scores, by 20 mins and 24 hours there was no difference between the groups. Most mothers given propofol commented on the pleasant quality of recovery compared to previous anaesthetics while those who received thiopentone remarked that there was no difference.

Comparison of ketamine to non-ketamine induction in patients with ruptured aortic aneurysm

W.S. Beattie, D.N. Buckley, S.A. Arciszewski, R.A. Browne, E.J. Ashworth
McMaster University Medical Centre, Hamilton, Ontario

Ketamine is thought to be the induction agent of choice in hypovolaemic states[1] since it retains or restores blood pressure in hypovolaemic situations. This effect on blood pressure is due to the ability of ketamine to augment sympathetic activity. To our knowledge, the effect of ketamine on sympathetic activity and/or blood pressure in the hypovolaemic patient has not been investigated. Animal studies have shown a negative inotropic effect of ketamine. In the hypovolaemic animal blood pressure falls in some cases and blood flow is redistributed away from vital organs.[2,3] These effects may persist for long periods. The nature and urgency of the hypovolaemic state may preclude informed consent, randomization and blinding. We therefore carried out the following retrospective comparison of the effects of ketamine and non-ketamine induction of patients with ruptured aortic aneurysm as a pilot study.

Methods

After approval from our Institutional Ethics Committee, the last 100 cases of ruptured aortic aneurysm (RAA) who came to surgery were reviewed. We defined RAA as any patient with that diagnosis in the operative report. Charts were reviewed for age, sex, mortality, dose of ketamine, systolic blood pressure and heart rate, before induction and at five minutes post-induction of anaesthesia, the number of units of packed cells and plasma required during operation and the need for inotropes to support blood pressure. We compared the use of ketamine to those inductions not utilizing ketamine. For patients with pre-induction systolic pressure above 90 mmHg ketamine was compard to induction with thiopentone. This arbitrary BP level of 90 is used in most definitions of shock. Statistical analysis was by t test (paired and unpaired) and Chi square where appropriate.

Results

The results are seen in the Table.

The dose of ketamine in 51 patients ranged from 0.5 $ml \cdot kg^{-1}$ to 1.3 $ml \cdot kg^{-1}$. Pre-induction BP were not significantly different between the two groups. Results of the patients with pre-induction BP > 90 mmHg show a higher mortality associated with ketamine induction ($p < 0.05$). Ketamine induction was associated with an average blood pressure decrase of greater than 24 mmHg ($p < 0.01$). This decrease was not seen in the group induced with a non-ketamine regime. The group induced with ketamine used significantly more packed cells and plasma. Sixty-two per cent of the patients induced with ketamine required inotropes to support blood pressure whereas the non-ketamine group required inotropic support only 11 per cent of the time ($p < 0.01$). In patients with preinduction blood pressure below 90 mmHg there were no differences between groups. There was a significant rise in pressure in the group not receiving ketamine ($p < 0.05$). Most of these patients received only muscle relaxant.

TABLE

	No.	*Mortality (%)*	*BP (preind)*	*BP (post)*
BP > 90 (Ket)	33	41.3	129.1 ± 25.1	104.8 ± 38.4
BP > 90	39	19.9	142.6 ± 30.8	146.8 ± 26.1
BP < 90 (Ket)	18	67.0	60.4 ± 24.5	62.0 ± 24.6
BP < 90	9	67.0	41.6 ± 22.4	71.7 ± 26.4

	No.	*PC's (units)*	*FFP (units)*	*Inotropes (%)*
BP > 90 (Ket)	33	10.1 ± 4.1	5.5 ± 2.6	62.1
BP > 90	39	6.8 ± 2.7	3.4±1.3	11.4
BP < 90 (Ket)	18	10.9 ± 3.3	6.1 ± 1.7	72.2
BP < 90	9	9.3 ± 3.1	5.0 ± 1.6	66.7

Discussion

The data presented in this paper is not definitive since it is retrospective and relies on the known inaccuracies of the anaesthetic record for haemodynamic data. We believe the baseline blood pressure to be accurate.

The results of this study show no clear benefit for ketamine. The use of this agent does not appear to offer any benefit over other regimes in maintaining blood pressure. The use of ketamine may in fact cause further deterioration in patients with RAA. On the basis of this study the use of ketamine in RAA should be further evaluated in a double-blind randomized trial. This analysis supports the contention from animal studies that if further sympathetic stimulation is not possible ketamine may in fact cause further harm.

References

1 *Chasapakis G, Kekis N, Sakkalis, Kolios D.* Use of ketamine for anesthesia for patients in hemorrhagic shock. Anesth Analg 1973; 52: 282–7.
2 *Weiskopf RB, Bogetz MS, Roizin MF, Reid IA.* Cardiovascular and metabolic sequelae of inducing anesthesia with ketamine or thiopental in hypovolemic swine., Anesthesiology 1984; 60: 214–9.
3 *Longnecker DE, McCoy S, Drucker WR.* Anesthetic influence on response to hemorrhage in rats. Circ Shock 1979; 6: 55–60.

Does aging affect alfentanil disposition in healthy volunteer and surgical patients?

P.C. Duke, D.S. Sitar, J.L. Benthuysen, N.T. Smith
Departments of Anesthesia and Pharmacology, University of Manitoba, Winnipeg, Manitoba and Department of Anesthesiology, University of California, San Diego, La Jolla, California

We studied the kinetic disposition of alfentanil in young and old healthy subjects and male surgical patients to see if age-associated changes with other narcotics extended to this drug. The alfentanil dose was 20 $\mu g \cdot kg^{-1}$ in the healthy volunteers and 100–180 $\mu g \cdot kg^{-1}$ in surgical patients. Plasma concentrations of alfentanil were measured by gas–liquid chromotog-

TABLE Results

	YS	OS	YP
N	10	10	4
Age (yr)	26±1	65±4	32±7
Weight (kg)	70.2±5.2	64.6±3.3	71.7±5.9
Alpha (min^{-1})	0.438±0.076	0.877±0.537	0.282±0.048
Beta (min^{-1})	0.021±0.004	0.014±0.002	0.097±0.063[a]
Vdss ($L \cdot kg^{-1}$)	0.391±0.056	0.340±0.077	0.554±0.130
Vp ($L \cdot kg^{-1}$)	0.270±0.047	0.257±0.068	0.225±0.079
Vc ($L \cdot kg^{-1}$)	0.131±0.028	0.083±0.011	0.329±0.079[c]
Clp ($ml \cdot kg^{-1} \cdot min^{-1}$)	9.3±2.0	4.1±0.8	39.6±19.0[d]
ke (min^{-1})	0.092±0.019	0.062±0.014	0.124±0.064

	MP	OP
	6	5
Age (yr)	52±5	65±3
Weight (kg)	85.1±5.5	78.4±8.3
Alpha (min^{-1})	2.75±1.79	0.398±0.148
Beta (min^{-1})	0.035±0.009[b]	0.010±0.007
Vdss ($L \cdot kg^{-1}$)	0.434±0.090	0.404±0.040
Vp ($L \cdot kg^{-1}$)	0.339±0.064	0.280±0.027
Vc ($L \cdot kg^{-1}$)	0.096±0.034	0.125±0.032
Clp ($ml \cdot kg^{-1} \cdot min^{-1}$)	12.8±2.5	4.6±1.6[e]
ke (min^{-1})	0.282±0.109	0.098±0.097

[a]Greater than OS and OP.
[b]Greater than OP.
[c]Greater than all other groups.
[d]Greater than all others excepting MP.
[e]Less than YP and MP.

raphy. Thirty-five subjects were grouped by age for comparison of the pharmacokinetic characteristics of alfentanil in healthy volunteers and in surgical patients. Subjects were classified as young if less than 40 years, middle-aged from 40 to 60 years of age, and old if beyond 60 years of age. This resulted in two groups, young and old for the healthy volunteers (YS, OS), and three groups for the surgical patients (YP, MP, OP).

Results

Comparison of the pharmacokinetic characteristics of alfentanil among these groups of subjects is presented in the Table. The initial drug distribution into the peripheral kinetic compartment, alpha, was not different with respect to age or health status. This finding was also true for apparent volume of distribution at steady-state (Vdss), and for the peripheral compartment space (Vp). However, the volume of the central compartment (Vc) was larger in young surgical patients than in any other group of subjects ($p = 0.013$). Regression analysis of Vc versus age indicated a significant decline with a modest inverse correlation ($r = -0.323$, $p < 0.05$). Kinetic constants associated with the elimination process showed significant differences among groups for beta ($p = 0.039$) and plasma clearance (Clp) ($p < 0.001$) but not for the elimination rate constant (ke) ($p = 0.151$). Clp was significantly reduced in the elderly healthy subjects and surgical patients, and regressions of beta and Clp versus age showed a significant decline with age ($r = -0.39$, $p < 0.01$ and $r = -0.54$, $p = 0.001$ respectively).

Discussion

This is the first concurrent comparison of the kinetic disposition of alfentanil in young and old healthy volunteers with surgical patients. The data suggest that older patients metabolize alfentanil more slowly than their younger counterparts. The more consistent disposition characteristics for alfentanil in elderly subjects suggest that dose–response relationships will be the most predictive in patients older than 60 years, and that surgical stress does not have a significant effect on alfentanil kinetics in this older group.

A quantitative description of the interactions of fentanyl and midazolam in reducing enflurane MAC in dogs

R.I. Hall, C.C. Hug Jr.
Cardiothoracic Anesthesia Division, Department of Anesthesiology, Emory University School of Medicine, Atlanta, Georgia

Fentanyl (FEN) and midazolam (MID) are combined to produce general anaesthesia. The nature of their interaction (i.e., additive, synergistic) is unknown. Neither drug alone is capable of replacing enflurane (ENF) as an anaesthetic.[1,2] We examined the interaction of FEN and MID in terms of their ability to reduce enflurane MAC (EMAC) in the dog.

Methods

Following determination of EMAC by the tail-clamp method, ten mongrel dogs were each given MID 9.6 $\mu g \cdot kg^{-1} \cdot min^{-1}$ to reduce EMAC by approximately 40 per cent for the duration of the experiment.[2] Following a 60 min observation period, EMAC was determined and then FEN was administered to each animal in a series of three incremental infusions (0.05, 0.2, 3.2 $\mu g \cdot kg^{-1} \cdot min^{-1}$) designed to produce 30, 50, and 65 per cent reductions of EMAC, respectively.[1] EMAC was determined following a 60 min observation period at each infusion rate. If no movement occurred following discontinuation of ENF the observation was confirmed by application of the tail-clamp stimulus every 10 min for 60 min. Then the FEN infusion was discontinued and naloxone (NOX) 1 $mg \cdot kg^{-1}$ was administered every 10 min during a final determination of EMAC. Arterial blood was taken for analysis of plasma concentrations of MID (by GLC) and FEN (by RIA) 45 min after the initation of any infusion rate and every 15 min until EMAC was determined. T-tests determined the difference ($p < 0.05$) between the degree of MAC reduction observed and that extrapolated from the concentration vs EMAC reduction curve obtained in previous experiments.[1,2] All results represent the mean ± SEM.

Results

MID plasma concentrations (360 ± 40 $ng \cdot ml^{-1}$) remained stable throughout the experiment and reduced EMAC by 38 ± 3 per cent (predicted EMAC reduction = 40 per cent).[2] The addition of FEN produced further reductions in EMAC (Table). When the further degree of EMAC reduction produced by any concentration of FEN was compared to that expected to occur

TABLE EMAC reduction and [FEN] produced by incremental infusions of FEN in dogs receiving enflurane and a continuous infusion of MID (values = mean ± SEM) (control EMAC = 2.28 ± 0.10%)

Infusion rate ($\mu g \cdot kg^{-1} \cdot min^{-1}$)	*% EMAC reduction*	*N**	*[FEN]* ($ng \cdot ml^{-1}$)	*% EMAC reduction by FEN*	
				Predicted†	*Actual‡*
MID 9.6	38 ± 3	0/10	0	0	0
MID + FEN 0.05	58 ± 6[a]	0/10	1.4 ± 0.1	10	20
MID + FEN 0.2	80 ± 5[a,b]	1/10	4.0 ± 0.4	38	42
MID + FEN 3.2	89 ± 3[a,b]	5/9	48 ± 5.1	59	51
MID + NOX 1 $mg \cdot kg^{-1}$	38 ± 5	0/10	—	0	0

*N = No of animals with >90% EMAC reduction and no movement in response to tail-clamp stimulus for 60 min.

†Predicted reduction of EMAC due to FEN based on extrapolation from log conc vs EMAC reduction curves obtained in previous experiments.[1]

‡Actual reduction of EMAC is that attributed to FEN after subtracting the contribution of MID alone (38%). No significant difference from predicted EMAC reduction.

[a]$p < 0.05$ vs MID alone.

[b]$p < 0.05$ vs MID + FEN 0.05.

based on extrapolation from a previously determined log FEN vs EMAC reduction curve,[1] no significant differences were observed, thus indicating an additive interaction of FEN and MID. In six animals a combination of MID-FEN completely replaced enflurane as demonstrated by no movement in response to tail-clamping repeatedly over a 60-min period. Movement occurred in four dogs in response to tail-clamping while they were receiving the highest FEN infusion rate. The degree of EMAC reduction achieved in these four dogs (84 ± 7 per cent; FEN = 51 ± ng · ml^{-1}) did not differ from that achieved in the six dogs (94 ± 1 per cent; FEN = 45 ± 10 ng · ml^{-1}) which did not move during the extended period of observation. After discontinuation of FEN and administration of NOX, the degree of EMAC reduction returned to that produced initially by MID alone (Table).

Discussion

FEN and MID interacted in an additive fashion to reduce EMAC in the dog. Combinations of MID-FEN were capable of completely replacing EMAC in 6/10 dogs. Possible reasons for the failure to completely replace EMAC in some animals include the development of acute tolerance to the opioid, as has been demonstrated to occur over time when sufentanil is infused continuously at a low rate in dogs studied under similar experimental conditions.[3] Tolerance may also develop to the effects of MID, but this explanation seems unlikely since administration of NOX returned EMAC reduction to that produced by MID alone.

References

1 *Murphy MR, Hug CC Jr.* Anesthesiology 1982; 57: 485–8.

2 *Hall RI, Lasker S, Hug CC Jr.* Anesthesiology 1987; 67: A657.

3 *Hall RI, Szlam F, Hug CC Jr.* Anesthesiology 1987; 67: 518–25.

Pharmacokinetics and pharmacodynamics of sufentanil in patients undergoing abdominal aortic surgery

R.J. Hudson, R. Bergstrom, I.R. Thomson, M.A. Sabourin, M.R. Rosenbloom, L. Strunin

Departments of Anaesthesia, St. Boniface General Hospital and the University of Manitoba, Winnipeg, Manitoba and Foothills Hospital at the University of Calgary, Calgary, Alberta

Sufentanil is becoming widely used during anaesthesia for cardiovascular surgery. Ideally, dosing regimens should be based upon pharmacokinetic principles. Accordingly, we undertook this investigation to determine the pharmacokinetics of sufentanil in patients undergoing abdominal aortic surgery, and to estimate the sufentanil concentration required to prevent haemodynamic responses to surgical stimulation.

Methods

After approval by the Human Subjects Committee, informed consent was obtained from eight patients scheduled for elective abdominal aortic surgery. Morphine 0.15 mg · kg^{-1} IM and scopolamine 0.006 mg · kg^{-1} IM were given preoperatively. Anaesthesia was induced with sufentanil 7.5 $\mu g \cdot kg^{-1}$, given by continuous infusion over 3 min. Metocurine 0.6 mg · kg^{-1} was given concomitantly for muscle relaxation. A second dose of sufentanil, 5 $\mu g \cdot kg^{-1}$ over 2 min, was administered just prior to skin incision. No additional anaesthetic agents were given unless the heart rate or mean arterial pressure increased to 120 per cent of the preincision values. At that time, nitrous oxide, diazepam, morphine, or appropriate vasoactive drugs were given at the discretion of the attending anaesthetist. Intraoperative fluid therapy consisted of sufficient crystalloid to maintain the pulmonary artery wedge pressure near preinduction values and transfusion of packed red blood cells as needed. Arterial blood samples for subsequent sufentanil analysis were drawn 1, 3, 5, 10, 15, 20, 30, and 45 min and 1, 1.5, 2, 2.5, 3, 3.5, 4, 6, 8, 10, 12, 14, 16, 18, and 24 h after the first infusion. The serum was separated and stored at −20° C. Serum sufentanil concentrations were determined in triplicate by radio-immunoassay. The coefficient of variation of the assay was 12 per cent at 0.15 ng · ml^{-1} and three per cent at 8.2 ng · ml^{-1}. A three-compartment model, allowing for the two infusions, was fit to the data using nonlinear regression. The distribution and elimination half-times ($t_{1/2}$), clearance (Cl), the volume of the central compartment (Vc), and the volume of distribution at steady state (Vdss) were calculated using standard formulae.[1]

Results

The mean (±SD) age of our patients was 68.9 ± 8.8 years and their mean weight was 74.7 ± 15.6 kg. Cl was 15.2 ± 3.4 ml · $min^{-1} \cdot kg^{-1}$, Vc was 0.28 ± 0.09 L · kg^{-1}, Vdss was 9.2 ± 4.5 L · kg^{-1}, the rapid and slow distribution $t_{1/2}$'s were 1.7 ± 0.5 and 23.7 ± 4.3 min, respectively, and the elimination was $t_{1/2}$ 12.3 ± 5.2 h. All eight patients required additional anaesthetic agents prior to the end of surgery. The mean sufentanil concentration associated with increases in heart rate or blood pressure was 1.3 ± 1.1 ng · ml^{-1} (range 0.56–3.7).

Discussion

The mean elimination $t_{1/2}$ of sufentanil in patients undergoing general surgery is 2.7 h, with mean Cl of 12.7 ml · min^{-1} · kg^{-1} and Vdss of 1.7 L · kg$^{-1.2}$ Cl is similar in our patients. However, the Vdss is much larger. The elimination $t_{1/2}$ of any drug is inversely proportional to its clearance and directly proportional to its Vdss. Therefore, the prolonged elimination $t_{1/2}$ of sufentanil observed in our patients is solely due to a large Vdss. This long elimination $t_{1/2}$ is clinically important. Recovery after large doses of sufentanil will take longer than would have been predicted from previously published data.

References

1 *Gibaldi M, Perrier D.* Pharmacokinetics. 2nd ed. New York: Marcel Dekker, 1982; 45–143.

2 *Bovill JG, Sebel PS, Blackburn CL, Oei-Lim V, Heykants JJ.* The pharmacokinetics of sufentanil in surgical patients. Anesthesiology 1984; 61: 502–6.

Optimal sufentanil dosage – haemodynamic control or respiratory depression?

D.C.H. Cheng, F. Chung, K.R. Chapman, J. Romanelli, F.H.H. Leenen

Departments of Anaesthesia and Medicine, Toronto Western Hospital, University of Toronto, Toronto, Ontario

In order to attenuate the pressor response to laryngoscopy and tracheal intubation with the recommended dosage of sufentanil, the risk of postoperative respiratory depression is relatively high.[1] Recently, we have shown that the optimal time to blunt the cardiovascular response by intravenous lidocaine is three minutes prior to tracheal intubation.[2] In this double-blind study, we compared the effect of low-dose sufentanil, with or without lidocaine on cardiovascular and plasma catecholamine responses to endotracheal intubation and postoperative respiratory depression by respiratory inductive plethysmography.

Methods

Human Experimentation Committee approval and informed consent were obtained. Twenty adult ASA physical status I–II patients scheduled for elective surgical procedures were studied. No premedication was given. Baseline flow-volume spirometric and respiratory inductive plethysmographic measurements were done preoperatively. Patients were randomized into three groups for induction: control (C); lidocaine 1.5 mg · kg^{-1} followed with sufentanil 0.5 μg · kg^{-1} (LS); and sufentanil 1.0 μg · kg^{-1} alone. Thiopentone was titrated until loss of consciousness and succinylcholine 1.5 mg · kg^{-1} was administered. The trachea was intubated. Anaesthesia was maintained by controlled ventilation with 70 per cent nitrous oxide and 30 per cent oxygen. Measurement of heart rate (HR) and blood pressure (BP) were recorded continuously 5 min before and after intubation. Then anaesthesia was supplemented with vecuronium ± isoflurane. Plasma catecholamine levels were assayed at baseline, 1 min and 5 min post-intubation. Postoperatively, patients were monitored continuously by the respiratory inductive plethysmograph and arterial blood gases were measured at 30 min post-extubation. Data were analyzed as a repeated measures analysis, and differences across study groups and over time point were assessed with multiple comparisons using the Bonferroni correction and Chi-square analysis.

TABLE I Comparison of study groups

	C (n = 7)	*LS (n = 7)*	*S (n = 6)*
Age (yrs)	32 ± 5	31 ± 5	28 ± 6
Wt (kg)	75.5 ± 4.8	71.7 ± 4.8	72.7 ± 5.2
Duration (mins)	73.1 ± 13.8	82.4 ± 13.8	78.7 ± 14.9
Thiopentone (mg)	4.8 ± 0.2	3.9 ± 0.2*	2.9 ± 0.2*†
$PaCO_2$ (mmHg)	40.3 ± 3.0	44.4 ± 2.7	53.2 ± 3.0*†

*p < 0.01 (vs C).
†p < 0.05 (vs LS).

TABLE II Haemodynamic response

	Group	*Baseline*	*Intubn*	*1 min*	*3 min*	*5 min*
HR	C	66 ± 5	88 ± 7†	89 ± 5†	82 ± 5	75 ± 3
(bpm)	LS	72 ± 5	89 ± 7	77 ± 5	67 ± 5*	63 ± 3*
	S	70 ± 5	75 ± 7	70 ± 5*	62 ± 5*	57 ± 3*
SBP	C	122 ± 8	131 ± 7	162 ± 8†	143 ± 7†	135 ± 6
(mmHg)	LS	131 ± 8	123 ± 7	115 ± 8*	104 ± 7*†	96 ± 6*†
	S	137 ± 9	122 ± 7	108 ± 9*	105 ± 8*†	101 ± 7*†

*p < 0.05 (vs C).
†p < 0.005 (vs baseline intra-group.)

Results

There were no differences among the three groups in age, weight, and duration of surgery. The thiopentone dose requirement was significantly lower in both LS and S groups (Table I). The control group had significantly higher HR, SBP and DBP post-intubation (Table II). Epinephrine and norepinephrine levels were lowered post-intubation but not significantly different from the control group. No narcotic antagonist was required postoperatively. $PaCO_2$ was markedly elevated in group S 30 min post-extubation (Table I). Morphine requirement for postoperative analgesia was significantly higher in control group (57 per cent), when compared with LS(0 per cent) and S (17 per cent). The apnoea index indicated that group S had persistent apnoea, while group LS showed a decreasing rate of apnoea over an hour postoperatively (Figure). Apnoea was attributed to prolonged expiratory time interval.

Discussion

This study demonstrated that 1 μg · kg^{-1} of sufentanil on induction was associated with a persistent apnoea rate and CO_2 retention postoperatively in healthy patients undergoing 1.5 hr operations. The optimal drug doses in blunting haemodynamic response upon intubation and exhibiting minimal respiratory depression was found to be lidocaine 1.5 mg · kg^{-1} with sufentanil 0.5 μg · kg^{-1}. Therefore, the recommended the dose of sufentanil used in operations of less than 2 hr duration should

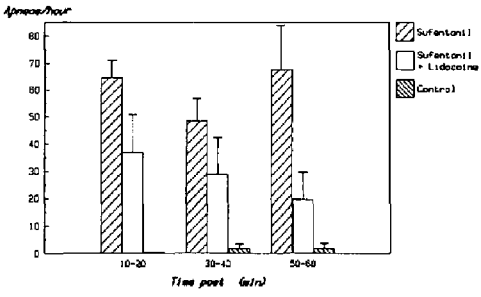


FIGURE Apnoea index.

be re-evaluated in view of the potential for respiratory depression.

References

1 *Murkin JM.* Multicentre clinical trial: sufentanil for major surgery. Can J Anaesth 1987; 34: S84.

2 *Tam S, Chung F, Campbell M.* Attenuation of circulatory responses to endotracheal intubation using IV lidocaine: a determination of the optimal time of injection. Anesth Analg 1987; 66: 1036–8.

Comparison of a physiological and a visual analogue pain scale in children

L.M. Broadman, L.J. Rice, R.S. Hannallah
Children's Hospital National Medical Center and the George Washington University, Washington, District of Columbia

The 0–10 Linear Analogue Pain Scale (LAPS) is the standard instrument for pain measurement in adults. However, since its use requires patient comprehension and cooperation, it is difficult to use in studies involving young or nonverbal children, even when modified with pictures depicting facial expressions of pain or its absence (Figure). Alternatively, an Objective Pain Scale (OPS) has been used in several recent studies[1–3] to equate pain and discomfort in young children with changes in such physiologic variables as blood pressure elevation, crying, movement, agitation, and either body language or verbal complaints of pain (Table).

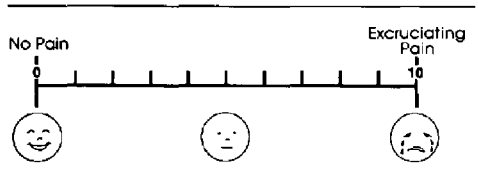


FIGURE Linear analogue pain scale.

TABLE Objective pain scale

Observation	*Criteria*	*Points*
Blood pressure	±10% pre-op	0
	>20% pre-op	1
	>30% pre-op	2
Crying?	Not crying	0
	Crying but responds to tender loving care (TLC)	1
	Crying and does not respond to TLC	2
Movement	None	0
	Restless	1
	Thrashing	2
Agitation	Patient asleep or calm	0
	Mild	1
	Hysterical	2
Verbal evaluation	Patient asleep, or states no pain	0
	Mild pain (cannot localize)	1
	Moderate pain (can localize verbally or by pointing)	2

Although the OPS has been repeatedly used to determine the need for the administration of analgesics in previous studies,[1–3] its validity has never been verified.

Methods

We prospectively studied 29 verbal children and adolescents ranging in age from 13–18 years who underwent arthroscopy under general anaesthesia. Postoperative pain and discomfort were evaluated by the patient as well as an observer using both LAPS and OPS at five-minute intervals for the first 30 minutes and at ten-minute intervals for another 30 minutes. Verbalization of pain was deemed to be appropriate for the children participating in this study, and was used instead of body language. 0–2 points were assigned for each of the five OPS categories (Table). The LAPS scale was simultaneously scored on a 0–10-point basis. The paired pain scores on the LAPS and OPS pain scales were analyzed using Spearman's rank correlation.

Results

There was an excellent correlation between LAPS and OPS in children having intense pain (score ≥ 6) (spearman's $r = 0.695 \pm 0.068$). There was less agreement, however, in children having only mild or moderate pain (score < 6) ($r = 0.479 \pm 0.083$).

Discussion

Until recently, the diagnosis and management of postoperative pain in children has been a neglected dimension of paediatric anaesthesia and surgery. Methodological difficulties as well as the inability to apply adult data to children have so far complicated and stifled research in paediatric pain management. Objective pain assessment is important for diagnostic and treatment purposes, and to determine analgesic efficacy of different therapeutic interventions. Several methods of quantifi-

cation have been attempted in children; few have been validated against accepted adult pain scales. While direct validation of OPS is not possible in nonverbal infants and very young children, this study has demonstrated in older children that OPS gives similar results to those obtained with LAPS. It is therefore inferred that the Objective Pain Scale (OPS) is a valid instrument for objectively assessing the intensity of pain in very young infants and nonverbal children.

References

1 *Hannallah RS, Broadman LM, Belman AB, Abramowitz MD, Epstein BS.* Comparison of caudal and ilioinguinal/iliohypogastric nerve blocks for control of post-orchiopexy pain in pediatric ambulatory surgery. Anesthesiology 1987; 66: 110–2.

2 *Broadman LM, Hannallah RS, Belman AB, Elder PT, Ruttiman U, Epstein BS.* Post-circumcision pain – a prospective evaluation of subcutaneous ring block of the penis. Anesthesiology 1987; 67: 399–402.

3 *Wolf AR, Lerman J, Valley R, Fear DW, Roy WL.* Minimal effective concentration of bupivacaine for caudal blocks in paediatrics. Can J Anaesth 1987; 34: S92.

Pharmacokinetics of two per cent rectal methohexitone in children

R.B. Forbes, K.L. Croskey, D.L. Dull, D.J. Murray, J.B. Dillman, K. Murphy

Department of Anesthesia, University of Iowa College of Medicine, Iowa City, Iowa

Rectal methohexitone is a safe, pleasant technique for the induction of anaesthesia in young children and use of a ten per cent solution results in a rapid, reliable onset of sleep.[1,2] Some investigators have reported that 15 mg · kg^{-1} of a less concentrated solution was as effective as 25 mg · kg^{-1} ten per cent methohexitone for induction anaesthesia[3] and that use of a two per cent solution resulted in higher peak plasma methohexitone concentrations.[4] Because changes in the concentration of the methohexitone solution alter its pharmacokinetics, the optimal dose of two per cent methohexitone for induction of anaesthesia has not been determined. The purpose of this investigation was to compare the effectiveness of plasma methohexitone concentrations achieved following rectal administration of four different doses of two per cent methohexitone.

Methods

Forty-one children were studied after obtaining informed parental consent. Each child was randomly assigned to receive 15, 20, 25 or 30 mg · kg^{-1} of a two per cent methohexitone solution. The drug was administered rectally while a parent comforted the child until asleep. Sleep was defined as loss of consciousness, unresponsiveness to verbal stimuli and absence of voluntary movement when unstimulated. Time from administration of methohexitone until the onset of sleep was recorded. Children not asleep 15 minutes following methohexitone administration were considered failed inductions. All children were then transferred to the operating room and anaesthesia was continued with nitrous oxide and halothane in oxygen. Blood was collected at 15, 30 45 and 120 minutes following drug administration, then centrifuged, and the plasma frozen for later determination of methohexitone concentration using gas chromatography. Results are expressed as mean ±SE.

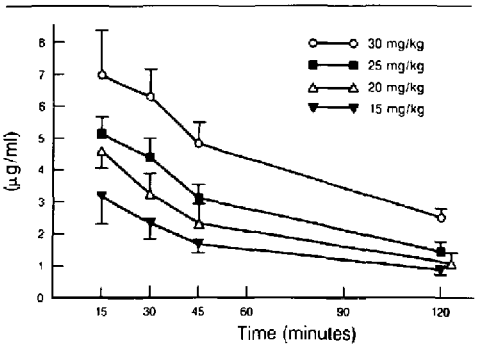


FIGURE 1 Plasma methohexitone concentration.

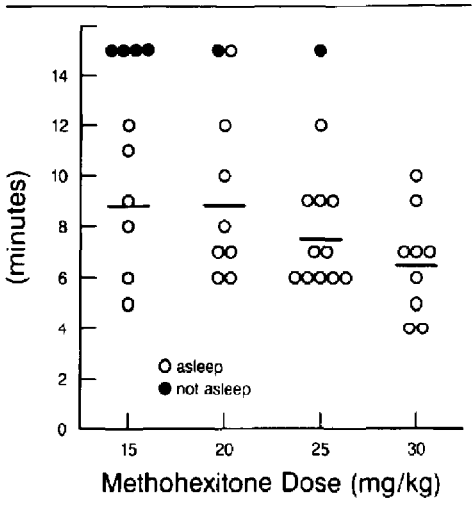


FIGURE 2 Time to onset of sleep.

Results

Results are shown in Figures 1 and 2.

Discussion

The effectiveness of rectally administered methohexitone depends upon complex interaction between the volume and concentration of the solution administered and the site of absorption within the rectum. Use of a larger volume of a less concentrated solution results in higher plasma methohexitone

concentrations and may decrease the risk of damage to the rectal mucosa that has been reported following the use of ten per cent rectal methohexitone in mice.[5] The results of this study indicate that 15–30 $mg \cdot kg^{-1}$ of two per cent rectal methohexitone produces plasma concentrations that consistently produce sleep. We conclude the two per cent rectal methohexitone is an effective alternative to use of a ten per cent solution for induction of anaesthesia in children.

References

1 *Goresky GV, Steward DJ.* Can Anaesth Soc J 1979; 26: 213–5.
2 *Liu LMP, Goudsouzian NG, Liu PL.* Anesthesiology 1980; 53: 343–5.
3 *Laishley RS, O'Callaghan AC, Lerman J.* Can Anaesth Soc J 1986; 33: 427–32.
4 *Forbes RB, Vandewalker GE.* Anesthesiology 1986; 65: A420.
5 *Hinkle AJ, Weinlander CM.* Anesthesiology 1985; 63: A461.

The cardiovascular effects of rectal methohexitone in children

R.B. Forbes, D.L. Dull, D.J. Murray, K.L. Croskey
Department of Anesthesia, University of Iowa College of Medicine, Iowa City, Iowa

Rectal methohexitone is an effective technique for the induction of anaesthesia in young children.[1,2] However, the cardiovascular changes associated with this technique have not been fully investigated. The purpose of this study was to evaluate the cardiovascular effects of rectal methohexitone in children using two-dimensional (2-D) and pulsed Doppler echocardiography.

Methods

After obtaining institutionally approved informed parental consent, 12 children, ASA physical status I or II, less than five years of age scheduled for elective surgery were studied. Fifteen minutes prior to the induction of anaesthesia, each child was evaluated using 2-D and pulsed Doppler echocardiography. The cardiovascular parameters obtained included blood pressure and heart rate, left ventricular length and area in systole and diastole, pulmonary artery diameter and pulsed Doppler echocardiographic determination of the velocity of pulmonary blood flow. Following the initial echocardiogram, anaesthesia was induced using 25 $mg \cdot kg^{-1}$ two per cent methohexitone administered rectally. Immediately following loss of consciousness, a second 2-D and pulsed doppler echocardiogram was obtained. The children were then transferred to the operating room and anaesthesia was continued with halothane and oxygen.

The data obtained by 2-D and pulsed doppler echocardiography was used to calculate cardiac output, cardiac index, stroke volume and ejection fraction. Changes in cardiovascular function that occurred following administration of rectal methohexitone were compared to control values using two-way analysis of variance. Significance was accepted at $p < 0.05$. Results are expressed as mean ± SEM.

TABLE Hemodynamic data (mean ± SEM)

	Pre-induction	*Post-induction*
Heart rate (beat · min^{-1})	113.9 ± 6.6	126.1 ± 4.5*
Mean arterial pressure (mmHg)	72.9 ± 2.4	68.2 ± 2.0
Cardiac index (ml · $min^{-1} \cdot m^{-2}$)	5046.8 ± 723.5	5184.6 ± 334.0
Stroke volume (ml)	12.6 ± 1.7	13.8 ± 2.2

*$p < 0.05$.

Results

The results are shown in the Table. Following induction of anaesthesia with methohexitone there was a significant increase in heart rate. Mean arterial pressure, left ventricular end diastolic and systolic volume, cardiac output, cardiac index, stroke volume and ejection fraction did not change significantly following 25 $mg \cdot kg^{-1}$ rectal methohexitone.

Discussion

Previous investigators have reported that rectal administration of methohexitone in children caused an increase in heart rate and no change in systolic blood pressure.[2] However, children in that study also received atropine and no direct determination of cardiac function was made. In this investigation administration of 25 $mg \cdot kg^{-1}$ rectal methohexitone also caused tachycardia, but arterial pressure, cardiac output and stroke volume were maintained. While respiratory depression can occur following use of barbiturates in children, this study indicates that rectal administration of methohexitone for induction anaesthesia had minimal cardiovascular effects in healthy paediatric patients.

References

1 *Liu LMP, Gaudreault P, Friedman PA, Goudsouzian NG, Liu PL.* Anesthesiology 1985; 62: 567–70.
2 *Laishley RS, O'Callaghan AC, Lerman J.* Can Anaesth Soc J 1986; 33: 427–32.

Accuracy of capnographs in a neonatal lung model

R.F. Kaplan
Departments of Anesthesiology and Paediatrics, University of Florida College of Medicine, Gainesville, Florida

Large gradients between peak expired (P_ECO_2) and arterial ($PaCO_2$) carbon dioxide tensions in anaesthetized neonates have been noted with various capnographs. We examined capnograph accuracy in a neonatal lung model with various mainstream and sidestream capnographs.

Methods

One side of a test lung (Vent-Aid) was modified with an internal fan and ports to instill and sample CO_2 from the bellows ("alveolus").[1] A "trachea" was simulated with a paediatric pneumotachograph, pressure gauge, and neonatal airway adapter (Hewlett Packard used for capnograph). A 3.5-kg newborn lung was simulated by infusing CO_2 into the bellows with a

TABLE Measurements by different monitors of Alveolar-Peak expired PCO_2 gradient ($P[A-E]CO_2$) and inspired PCO_2 ($PiCO_2$) in a neonatal lung model at different respiratory rates with and without sighs

	Capnographs						
		*Sidestream**				*Mass spectrometer*	
Respiratory rate (breaths · min⁻¹)	*Mainstream*	*High*	*High with sigh*	*Low*	*Low with sigh*	*Without sigh*	*With sigh*
Mean $P(A-E)CO_2$ (mmHg)							
10	0.33	−1.17	—	−0.6	—	1.93	—
	(0.58)	(1.99)	—	(1.6)	—	(1.66)	—
80	0.00	7.4†	0.8	11.87†	−0.8	15.53†	2.9
	(1.0)	(2.5)	(1.39)	(3.06)	(0.72)	(2.32)	(0.9)
Mean $PiCO_2$ (mmHg)							
10	0.23	0.0	—	2.67	—	4.3	—
	(0.40)	(0.0)	—	(0.23)	—	(0.3)	—
80	6.5†	11.7†	0.0	17.5†	2.0	20.8†	3.6
	(3.1)	(2.1)	(0.0)	(2.26)	(0.68)	(3.2)	(0.9)

Numbers in parentheses are 95% confidence limits.
*High sampling rate: 250 ml · min⁻¹; low sampling rate: 120 ml · min⁻¹.
†Mean ± 95% confidence limits > 5 mmHg (arbitrary limit of clinical acceptability).

calibrated flowmeter at 6 ml · kg⁻¹ · min⁻¹; "tracheal" dead space was 2 ml · kg⁻¹ and lung compliance, 10 ml/cm H_2O. "Alveolar" gas sampled at 6 ml · kg⁻¹ · min⁻¹ was analyzed with an infrared capnograph (HP-47210A, Hewlett Packard). The "trachea" was connected to a disposable circle system via a 5-ml elbow with a sampling port for sidestream capnographs on the tracheal end. The test lung was ventilated at 10, 20, 30, 40, 60, or 80 breaths · min⁻¹ with an inspiratory-to-expiratory ratio of 1:2 by a prototype Ohmeda anaesthesia volume ventilator. Tidal volume was adjusted to keep simulated alveolar CO_2 at 40 ± 1 mmHg (SD) for 5 min at each respiratory rate. Inspired and peak expired CO_2 were sampled with a mainstream infrared capnograph (HP 78354A), a sidestream capnograph at 250 and at 120 ml · min⁻¹ (Saracap, 10-ft sampling tube), or a mass spectrometer at 240 ml · min⁻¹ (Perkin-Elmer, 120-ft sampling tube). Additional measurements were made at respiratory rates of 40, 60, and 80 breaths · min⁻¹ immediately after the initiation of "sighs" (10 breaths · min⁻¹ with equivalent tidal volumes). Capnographs were applied in random order. Paper recordings verified reproducible waveforms; each measurement was repeated three times. Before and after each series of measurements, capnographs were calibrated with a five per cent CO_2 calibration gas. Significant differences among data were determined by analysis of variance with post hoc testing and Duncan's multiple range test; values > 5 mmHg were arbitrarily selected as clinically unacceptable.

Results

Both $P(A-E)CO_2$ and $PiCO_2$ were significantly affected by respiratory rate, sigh, and capnograph ($p > 0.0001$ for all) (Table), values being more accurate with slow respiration (values at lowest and highest rates shown in Table), sighs, or the mainstream capnograph and less accurate with the sidestream capnograph at a low sampling rate.

Discussion

The accuracy of capnographs is adversely affected by shallow, rapid respiration, as often used for neonates, and can be improved by adjusting ventilation, using a mainstream capnograph, or both.

Reference

1 *Lampotang S, Gravenstein N, Banner M et al.* A lung model of carbon dioxide concentrations with mathematical or spontaneous ventilation. Crit Care Med 1986; 14: 1055–7.

The control of postoperative apnoea in the ex-premature infant: experience with a high caffeine dose

L.G. Welborn, R.S. Hannallah, R. Fink
Children's Hospital National Medical Center and George Washington University, Washington, District of Columbia

The use of caffeine as a respiratory stimulant has been shown to be effective in the management of neonatal apnoea and postoperative ventilatory dysfunction in former premature infants.[1,2] In a recent study, the administratoin of 5 mg · kg⁻¹ caffeine resulted in a significant reduction in the severity of postoperative apnoea; complete abolition of all types of apnoea, however, did not occur.[2] We designed this double-blind, randomized, prospective study to examine the effectiveness of a higher dose of caffeine in the control of postoperative apnoea in ex-premature infants.

Methods

Informed consent and institutional approval for the study were obtained. Former premature infants (≤37 weeks gestational age) undergoing general anaesthesia for minor surgery were studied. All were ≤44 weeks conceptual age at the time of surgery. Infants with cardiac, neurologic, endocrine or metabolic diseases and patients already receiving methylxanthines were excluded. All infants received inhaled endotracheal anaesthesia with neuromuscular blockade. No barbiturates or narcotics were given. Heart rate and sounds, blood pressure, electrocardiogram, temperature, respiration, $ETCO_2$ and oxygen saturation were monitored. Infants were randomly divided into two groups. Group I received IV caffeine 10 mg · kg⁻¹. The drug was administered slowly immediately following induction so that its peak effect would manifest at the end of surgery. Group II received IV saline (controls). The solutions were supplied by the hospital pharmacy. At the completion of surgery, the trachea was extubated in the operating room when the patient was fully awake and a venous blood sample was drawn to measure caffeine level. The pattern of respiration, heart rate and oxygen saturation were monitored and recorded for at least 12 hours postoperatively using a pneumogram with a magnetic tape recorder and Nellcor® N-100 pulse oximeter respectively. The recorded data were analyzed by the pulmonologists for evidence of apnoea, periodic breathing, bradycardia and desaturation. Brief apnoea was defined as a respiratory pause < 15 seconds; prolonged apnoea was a respiratory pause ≥ 15 seconds *or* < 15 seconds if accompanied by bradycardia; bradycardia: heart rate

TABLE Age, incidence of apnoea, periodic breathing (PB) and desaturation

	Group I $n = 7$	*Group II* $n = 6$	
Gestational age (weeks)			
(mean ± SD)	31.0 ± 3.2	31.7 ± 2.6	
(range)	26–34	28–35	
Conceptual age (weeks)			
(mean ± SD)	41.9 ± 2.0	40.8 ± 1.8	N.S
(range)	38–44	39–44	
History of pre-op apnoea	3(43%)	3(50%)	
Post-op apnoea < 15 sec (no bradycardia)	1(14%)	none	
Post-op prolonged apnoea with bradycardia	none	5(83%)	$p < 0.005$
Post-op PB > 1%	none	none	
Post-op desaturation < 95%	none	4(67%)	$p < 0.021$
Post-op caffeine level $mg \cdot L^{-1}$ (range)	15–19	zero	

< 100 bpm for at least five seconds; periodic breathing (PB): three or more periods of apnoea 3–15 seconds separated by < 20 seconds of normal respiration; desaturation was an O_2 saturation <95 per cent. The difference in the incidence of apnoea and desaturation between the two groups was compared using Fisher's exact test.

Results

Thirteen premature infants were studied. Seven received caffeine, and six received saline. There were no significant differences between the two groups in gestational or conceptual ages. The incidence of apnoea, PB and/or desaturation in the two groups is shown in the Table. None of the patients in either group required endotracheal intubation or controlled ventilation postoperatively. The difference in the incidence of prolonged apnoea with bradycardia and desaturation between the two groups is statistically significant ($p < 0.005$ and < 0.021 respectively).

Discussion

Both theophylline and caffeine have been extensively usd for the therapy of neonatal apnoea. Caffeine 5 $mg \cdot kg^{-1}$ has also been shown to be useful in the control of prolonged postoperative apnoea but not brief apnoea in premature infants.[2] The data from this study indicate that caffeine 10 $mg \cdot kg^{-1}$ is effective in the control of all types of apnoea in those infants. It is still recommended, however, that until a larger sample of infants has been studied, all infants at risk be monitored for apnoea and/or bradycardia following general anaesthesia.

References

1 *Arranda JV, Grondin D et al.* Pharmacologic considerations in the therapy of neonatal apnea. Pediat Clin N Am 1981; 28: 113–33.

2 *Welborn LG, DeSoto H, Hannallah RS et al.* The use of caffeine in the prevention of postanesthetic apnea in premature infants. Anesthesiology 1987; 67: A507.

Preoperative fasting in children: how long is enough?

B.K. Sandhar, G.V. Goresky, E.A. Shaffer, L. Strunin
Departments of Anaesthesia and Gastroenterology, University of Calgary, Calgary, Alberta

Traditionally paediatric patients are starved for four to six hours before elective surgery to allow adequate time for the stomach to empty. During this period the stomach continues to secrete fluids. Measurement of gastric volume at induction does not distinguish between secretions and residual ingested fluids. Administration of a marker dye, such as phenol red, with the ingested fluids enables assessment of the extent to which these have been cleared by the stomach.[1]

Methods

Written, informed consent was obtained from the parents of 84 healthy children, aged 1–14, admitted for elective, minor surgery. Children with a history of gastrointestinal disease or those receiving drugs which affect gastric secretion or motility were excluded from the study. The children were randomised into four groups, A, B, C and D. At two hours before the scheduled time of surgery, 1 ml of phenol red solution was administered to each child. At this time, the children in groups A and B received 5 $ml \cdot kg^{-1}$ of orange juice whereas those in groups C and D continued to fast. In addition, ranitidine 2 $mg \cdot kg^{-1}$ was given to groups B and D and placebo was given to A and C. The children were then kept nil by mouth until surgery. The stomach contents were aspirated within ten minutes of induction of anaesthesia using a 14-gauge multi-orifice orogastric tube and a syringe. The volume obtained was recorded and the sample frozen for subsequent dye analysis. A Beckman U-50 spectrophotometer was used to measure the phenol red concentration.[2] Then this was used to calculate the percentage recovery of the dose administered preoperatively and hence the percentage contribution of the oral fluids to the total gastric volume at induction.

Results

The groups were comparable with respect to age, weight and duration of fast before entering the study. Analysis of the phenol red showed that recovery of the original dose depended upon the interval between ingestion and sampling. The highest recovery, 33 per cent, was from one group C patient in whom the sample was taken one hour after ingestion. Samples taken between one and two hours after ingestion showed less than five per cent recovery in all groups. The recovery was zero per cent in all patients when more than two hours had elapsed since ingestion. These results demonstrated that oral fluids administered in this study were completely cleared by the stomach within two hours. Therefore the volume aspirated after this time interval reflects endogenous gastric secretions. We conclude that two hours of fasting following clear oral fluids in healthy children is an adequate period of starvation preoperatively.

References

1 *George JD*. New clinical method for measuring the rate of gastric emptying: the double sampling test meal. Gut 1968; 9: 237–42.

2 *Scheld HP*. Use of polyethylene glycol and phenol red as unabsorbed indicators for intestinal absorption studies in man. Gut 1961; 7: 159–63.

Effect of volume of bupivacaine on the effectiveness of caudal analgesia in children

S. Malviya, D.W. Fear, W.L. Roy, J. Lerman

Department of Anaesthesia and the Research Institute, The Hospital for Sick Children and the University of Toronto, Toronto, Ontario

Caudal blocks are widely used for postoperative analgesia after penoscrotal and inguinal surgery. Although the optimal concentration of bupivacaine with epinephrine for caudal analgesia has been investigated, the optimal volume remains unclear.[1] In order to determine the optimal volume of bupivacaine for adequate caudal analgesia, we compared the effectiveness of two volumes of 0.125 per cent bupivacaine with 1:200,000 epinephrine for caudal analgesia in children.

Methods

This prospective, randomized, double-blind study was approved by the local institutional ethics committee. Informed, written consent was obtained from the parents of 23 children scheduled for hypospadias repair, non-plastibell type circumcision, inguinal hernia repair and orchidopexy. The patients were one to ten years of age, ASA physical status I or II, fasting and unpremedicated. Anaesthesia was induced in all patients with an intravenous technique and maintained with a volatile anaesthetic with or without a muscle relaxant. Narcotics were not administered to these patients preoperatively or during surgery.

A caudal block was administered at the completion of surgery. Each patient was randomly assigned to receive either 0.5 $ml \cdot kg^{-1}$ or 1.0 $ml \cdot kg^{-1}$ of 0.125 per cent bupivacaine with 1:200,000 epinephrine. After awakening from anaesthesia the patient was transferred to the recovery room.

Patients were evaluated at 1, 2, 4 and 12 hours after administration of the block. Postoperative pain, lower extremity motor function and urinary retention were assessed by a blinded observer at one hour and by blinded nurses on the ward thereafter. Pain was assessed using a scoring system described previously.[2] Motor strength was evaluated on a four-point scale ranging from paralyzed (one) to forced movement against gravity (four).[1] Analgesic medications were administered according to the surgical standing orders at the discretion of the nurses if the pain score exceeded three.

Statistical significance ($p < 0.05$) was determined using the Fisher exact test, Mann-Whitney U test and Students' t test.

Results

Twenty-three children were studied: 13 received 0.5 $ml \cdot kg^{-1}$ and 10 received 1.0 $ml \cdot kg^{-1}$ of the caudal solution. One patient (0.5 $ml \cdot kg^{-1}$ bupivacaine) was excluded from the analysis because he returned to the operating room for a second procedure within six hours of the caudal block. The mean age, weight and type of surgery did not differ significantly between the two groups (Table). The duration of analgesia and the number of patients who required postoperative narcotic analgesics did not differ significantly between the two groups (Table). All patients were able to stand with minimal assistance one hour after the caudal block.

TABLE Results

	Volume of caudal solution	
	0.5 $ml \cdot kg^{-1}$	*1.0 $ml \cdot kg^{-1}$*
Number of patients	12	10
Weight (kg)*	16.4 ± 4.5	15.8 ± 4.8
Age (years)*	4.03 ± 2.25	3.21 ± 1.43
Per cent of patients given narcotics	25%	20%
Median motor score (range)	4	4

*Data are means ± SD.

Discussion

The preliminary results of this study indicate that both 0.5 and 1.0 $ml \cdot kg^{-1}$ of 0.125 per cent bupivacaine with 1:200,000 epinephrine provide adequate postoperative analgesia with minimal side effects in children undergoing penoscrotal and inguinal surgery.

References

1 *Wolf AR, Valley RD, Fear DW, Roy WL, Lerman J*. The minimum effective concentration of bupivacaine for caudal analgesia after surgery in pediatrics. Anesthesiology 1987; 67: A485.

2 *Hannallah RS, Broadman LM, Belman AB et al*. Comparison of caudal and ilioinguinal/iliohypogastric nerve blocks for control of post-orchidopexy pain in pediatric ambulatory surgery. Anesthesiology 1987; 66: 832–4.

Effects of metoclopramide and ranitidine on gastric fluid pH and volume in children

J. Lerman, S.K. Christensen, A.C. Farrow-Gillespie

Department of Anaesthesia and the Research Institute, The Hospital for Sick Children and University of Toronto, Toronto, Ontario

Seventy-five per cent of fasting children are at risk for the development of pneumonitis following pulmonary aspiration of gastric contents: that is, the gastric fluid pH is less than 2.5 and the gastric fluid volume is greater than 0.4 $ml \cdot kg^{-1}$.[1] Although metoclopramide and ranitidine increase the gastric fluid pH and decrease the fluid volume in adults,[2] similar data in children are unavailable. Therefore, we compared the effectiveness of these drugs in fasting children undergoing elective surgery.

Methods

With approval from the Human Review Committee, written informed consent was obtained from the parents of 40 children, ASA physical status I or II, between the ages of two and eight years scheduled for elective surgery. Children with known oesophageal or gastrointestinal diseases were excluded. The children were randomly assigned to receive one of four oral treatments with water (2 ml · kg^{-1}) four hours before surgery: no premedication (control) (n = 10), metoclopramide (0.1 mg · kg^{-1}) (n = 10), ranitidine (2 mg · kg^{-1}) (n = 10) and the combination metoclopramide (0.1 mg · kg^{-1}) and ranitidine (2 mg · kg^{-1}) (n = 10).

After induction of anaesthesia with thiopentone, atropine and succinylcholine, and tracheal intubation, the lungs were ventilated with nitrous oxide, oxygen, and halothane or isoflurane. Before skin incision, an orogastric tube (Salem 16 gauge) was inserted into the stomach. The pH of a 2 ml sample of gastric aspirate was determined using a calibrated pHM62 Radiometer pH meter. Bromosulphthalein (BSP) (2 mg · kg^{-1}) was then injected into the stomach. After the gastric contents were mixed, the stomach was emptied as thoroughly as possible and the concentration of BSP in the aspirate was analyzed by colorimetry using a calibrated Gilford Staser III Spectrophotometer. Gastric fluid volume was determined by BSP dilution. Measurements of pH and BSP concentration were performed by a blinded observer. Data were tested for normality using the Kolmogorov-Smirnov test. Statistical significance ($p < 0.05$) was determined using one-way ANOVA and the Student's t-test.

Results

The mean ages and weights of the four groups of patients did not differ significantly. Gastric fluid pH was significantly greater with ranitidine alone and in combination with metoclopramide than with the control and metoclopramide alone groups (Figure 1) ($p < 0.05$). Gastric fluid volume was significantly reduced with metoclopramide, ranitidine, and the combination (metoclopramide and ranitidine) compared to controls (Figure 2) ($p < 0.05$).

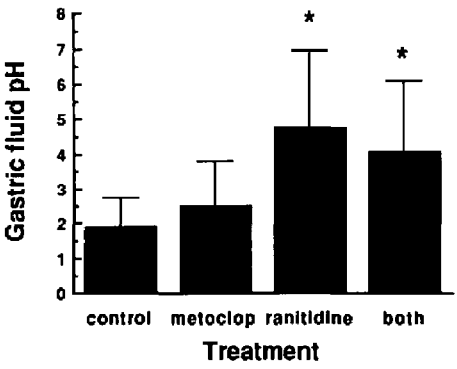


FIGURE 1 Gastric fluid pH.

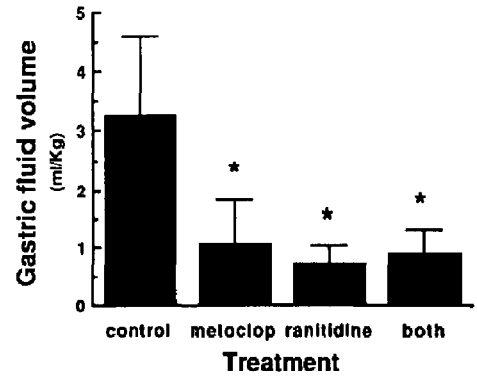


FIGURE 2 Gastric fluid volume.

Discussion

We found that ranitidine alone or in combination with metoclopramide when given orally four hours before surgery significantly increases the gastric fluid pH and decreases the gastric fluid volume in children. Oral metoclopramide (0.1 mg · kg^{-1}) alone does not significantly increase gastric fluid pH but does significantly decrease gastric fluid volume.

Supported in part by A. H. Robins, Canada, Inc.

References

1 *Coté CJ, Goudsouzian NG, Liu LMP et al.* Assessment of risk factors related to the acid aspiration syndrome in pediatric patients: gastric pH and volume. Anesthesiology 1982; 56: 70–2.

2 *Manchikanti L, Colliver JA, Marrero TC et al.* Ranitidine and metoclopramide for prophylaxis of aspiration pneumonitis in elective surgery. Anesth Analg 1984; 63: 903–10.

Lidocaine en aerosol après une amygdalectomie chez l'enfant

B. Bissonnette, J. M. Plante, P. Denault

Département d'anesthésie-réanimation, Hôpital du St-Sacrement, Université Laval, Québec

L'amygdalectomie chez les enfants est une intervention très douloureuse et peut même être quelques fois vraiment traumatisante. Depuis plusieurs années, de nombreuses études ont été publiées au sujet de l'analgésie post-amygdalectomie.[1–3] Par contre, aucune de ces études n'a pu mettre en évidence un analgésique capable de fournir une bonne analgésie sans pour autant modifier le cours normal de la période postopératoire.[4] Par conséquent, nous avons fait cette étude pour déterminer si l'utilisation de la lidocaïne en aérosol sur les fosses amygdaliennes après une amygdalectomie pourrait fournir une analgésie adéquate durant la période postopératoire immédiate.

Méthodes

Après l'accord du Comité d'éthique sur la recherche clinique, on a obtenu un consentement écrit de la part des parents de 30 enfants, de classification ASA I ou II, âgés de deux à dix ans. Aucune prémédication n'a été donnée et tous étaient à jeun. Tous les patients ayant des antédécents connus d'hypersensibilité aux anesthésiques locaux de type amide et/ou de sensibilité connue à l'excipient propylène glycol étaient exclus de l'étude. Dans cette étude prospective à double insu, chaque enfant était aléatoirement affecté à un des traitements suivants au moment de l'extubation: Groupe A: codéine 1.5 $mg \cdot kg^{-1}$ IM, Groupe B: lidocaïne aérosol dix pour cent. L'induction anesthésique se faisait par voie intraveineuse avec atropine (0.02 $mg \cdot kg^{-1}$), thiopental (5.0 $mg \cdot kg^{-1}$), et succinylcholine (2.0 $mg \cdot kg^{-1}$). Ensuite, tous étaient intubés et la ventilation spontanée était permise. Le maintien de l'anesthésie était fait avec un mélange de protoxyde d'azote dans l'oxygène et d'halothane en concentration clinique. A la fin de l'intervention et juste avant l'extubation, on administrait, sous vision directe, de la lidocaïne dix pour cent en aérosol (dose maximale de 2.0 $mg \cdot kg^{-1}$) sur les fosses et les piliers amygdaliens de chaque côté. Tous les patients étaient éveillés lorsqu'extubés. Une mesure de la lidocaïnémie était faite chez tous les patients à la 7ème, 10ème, et 15ème minutes après l'application. La lidocaïnémie a été mesurée par la méthode immuno-sérologique enzymatique homogène de Syva. (Emit®, Palo Alto, Calif.). Les observations postanesthésiques dans la salle de réveil étaient faites par l'infirmière responsable des soins au patient. Les infirmières ignoraient le régime thérapeutique utilisé chez le patient. Nous avons évalué l'incomfort et la douleur selon une échelle globale utilisant les points suivants: confortable = 1, agitation modérée = 2, incontrôlable = 3. De plus, l'âge et le poids des patients et la période de temps passée à la salle de réveil étaient notés. La signification statistique ($p < 0.05$) a été déterminée par détermination du Chi-carré avec facteur de correction de Bonferroni pour la continuité et par le test statistique de Student.

Résultats

L'âge moyen et le poids ne différaient pas entre les deux groupes (Tableau). Aucune complication, telle une augmentation de l'incidence d'hémorragie postopératoire ou une perturbation du réflexe de protection des voies respiratoires, n'a été observée. Le temps passé à la salle de réveil n'a pas été statistiquement différent entre les deux groupes (Tableau). Cependant, tous les patients ayant reçu de la lidocaïne en aérosol ont démontré un état de confort statistiquement différent de ceux ayant reçu de la codéine IM au moment de l'extubation ($p < 0.05$). La concentration systémique moyenne de lidocaïne à la 7ème minute était: 1.6 ± 0.3 $\mu g \cdot ml^{-1}$, 10ème minute: 1.3 ± 0.2 $\mu g \cdot ml^{-1}$, 15ème minute: 1.0 ± 0.4 $\mu g \cdot ml^{-1}$. La précision des résultats de licocaïnémies était confirmée par l'utilisation d'une solution-témoin (TDM, American Dade): trois niveaux, soit: C_1 = sub-thérapeutique 1.5 $\mu g \cdot ml^{-1}$, C_2 = thérapeutique 3.2 $\mu g \cdot ml^{-1}$ et C_3 = toxique 8.3 $\mu g \cdot ml^{-1}$.

TABLE Résultats

	Group A	*Group B*
Nombre de patients	15	15
Age (ans)	6.0 ± 2.3	5.9 ± 2.8
Poids (kg)	25.8 ± 7.4	23.7 ± 9.3
Séjour en salle de réveil (min)	71.4 ± 8.4	69.9 ± 7.1

Résultats: moyenne ± dév. std.
*$p < 0.05$ comparé au groupe A.

Discussion

Les résultats ont démontré qu'il y avait une différence significative entre les deux groupes quant au confort du patient et à l'absence de douleur, mais aucune différence significative n'a été observée quant au temps passé à la salle de réveil, ce qui est probablement attribuable au fait qu'il a été nécessaire d'utiliser de la codéine IM après environ 30–45 minutes. Toutefois, il est évident que l'utilisation topique de la lidocaïne dix pour cent en aérosol sur les fosses et les piliers amygdaliens est efficace dans la réduction de l'inconfort associé à la douleur durant la période postopératoire immédiate.

Références

1 *Rundle FW.* Post-tonsillectomy morbidity: a clinical trial of a local penecillin-steroid-anesthetic mixture. Laryngoscope 1968; 78: 1060–6.
2. *Sarnivaara I.* Comparison of paracetamol and pentazocine suppositories for pain relief after tonsillectomy in adults. Acta Anesthesiol Scand 1984; 28: 315–8.
3. *King JT.* Dangers of injections into the tonsillar fossae after tonsillectomy. Laryngoscope 1963; 73: 466.
4. *Crysdale WS.* Complications of tonsillectomy and adenoidectomy in 9409 children observed overnight. Can Med Assoc J 1986; 135: 1139–42.

Effects of age and weight on the measured rate of uptake of nitrous oxide in normal children

I.A. Sloan, G. Koren, A.C. O'Callaghan
The Hospital for Sick Children, University of Toronto, Toronto, Ontario

The effect of age on the uptake of nitrous oxide (N_2O) has been previously determined by using the time required to reach a steady state.[1] This methodology may be inaccurate since the steady state is often not achieved during the study period. The time to reach a steady state depends on elimination half-life ($t_{1/2}$) 95 per cent steady state = 5 × $t_{1/2}$. We therefore developed a method to calculate the $t_{1/2}$ of N_2O from the inspired and expired concentrations.

Methods

With approval from the ethical committee, N_2O uptake was measured in 31 normal children undergoing elective surgery. Anaesthesia was induced with thiopentone (5–6 $mg \cdot kg^{-1}$), atropine (0.02 $mg \cdot kg^{-1}$) and succinylcholine (2 $mg \cdot kg^{-1}$). Children were manually ventilated with 100 per cent oxygen, intubated, and then ventilated with a Sechrist ventilator delivering 30 per cent (inspired) O_2 in N_2O. N_2O was sampled continuously at a point 9 cm down the tracheal tube and the

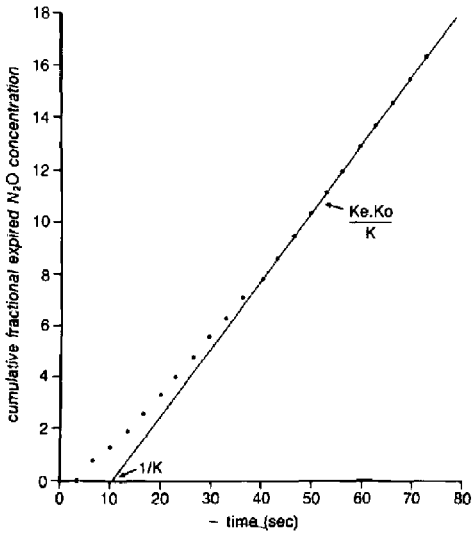


FIGURE 1 Expired N_2O concentration.

concentration was measured with a Beckman LB2 medical gas analyzer. The results were plotted on a Hewlett Packard 7004B X-7 chart recorder. When N_2O is administered at a constant rate and eliminated through the lungs, the pharmacokinetics may be described by:

$$X_l = \frac{Ke \cdot Ko}{K} \cdot t - \frac{Ke \cdot Ko}{K^2}(1 - e^{-Kt})$$

where: X_l = cumulative amount of N_2O excreted by the lungs, Ke = elimination rate constant via the studied organ (i.e. the lungs), K = total elimination rate constant (for N_2O, Ke = K); Ko = administration rate constant of N_2O; t = time from beginning of administration of N_2O.[2] The cumulative fractional exhaled N_2O concentration was plotted against time, where:

$$\text{fractional exhaled } (N_2O) = \frac{[N_2O] \text{ exhaled}}{[N_2O] \text{ inhaled}}.$$

Extrapolation of the linear part of the curve to the x-axis yields 1/K (Figure 1). This permits calculation of $t_{1/2}$; $t_{1/2}$ = 0.693/K. Statistical significance ($p < 0.05$) was determined using least square linear regression analysis or the unpaired t test wherever applicable.

Results

The $t_{1/2}$ of N_2O correlated significantly with age. Correlation between body weight and $t_{1/2}$ was less strong. The $t_{1/2}$ is significantly longer in normal children who are more than eight years of age ($t_{1/2}$ = 14.91 ± 1.21 sec) compared to children less than 8 years of age ($t_{1/2}$ = 8.3 ± 0.37 sec; $p < 0.0001$).

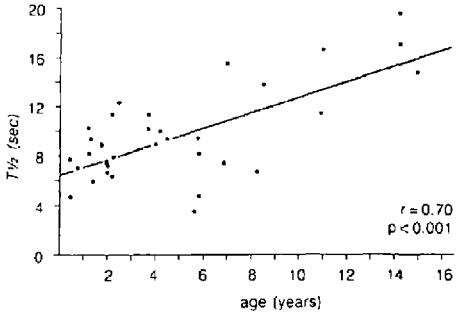


FIGURE 2 Elimination half-life for uptake of nitrous oxide in normal children.

Discussion

The $t_{1/2}$ of N_2O is significantly shorter in children < 8 years of age compared to children > 8 years of age. Consequently younger children will achieve a steady state more rapidly. This quantitative measure is in agreement with previous qualitative measures of N_2O uptake. We conclude that elimination half-life ($t_{1/2}$) is an accurate method of quantitating the time to reach steady state in children inhaling N_2O. This method may be employed with other inhaled anaesthetic agents to characterize their uptake rate.

References

1 *Salanitre E, Rackow H.* Anesthesiology 1969; 30: 388–94.
2 *Gibaldi M, Perrier D.* Pharmacokinetics. Marcel Dekker Inc., New York, 1982, pp. 31–33.

The effects of induction of anaesthesia with halothane on respiratory mechanics in infants

A. Goodman, D. Shulman, S. Godfrey
Hadassah University Hospital, Mount Scopus, Jerusalem, Israel

The induction of anaesthesia results in a decrease in respiratory compliance in adult subjects.[1] The effects of induction of anaesthesia with halothane on respiratory mechanics in infants have not been studied. We have applied a new, non-invasive technique for measuring C_{rs} in sedated infants,[2] termed "volume recruitment," to healthy infants while sedated and then following induction of anaesthesia with halothane in order to quantitate the changes in respiratory mechanics caused by this anaesthetic technique.

Methods

Four infants from 2.5 to 7 months of age (height 57 to 64 cm), who were scheduled to undergo general anaesthesia for elective surgery, were studied. They were all ASA physical status I and were delivered at full term, with no subsequent history of

TABLE Results

	Sedation	*Anaesthesia*
τ(s)	0.65 ± 0.25	0.30 ± 0.05*
C_{rs} (ml·cm^{-1} H_2O)	12.1 ± 3.3	6.4 ± 0.6*
R_{rs} (cm $H_2O \cdot L^{-1} \cdot s^{-1}$)	52.6 ± 7.2	47.3 ± 8.6

*Significant difference in comparision with sedation.

cardiopulmonary illness. The protocol received approval from the Human Subjects Review Committee of the hospital and informed consent was obtained from the parents of each child prior to the study. The infants received pre-anaesthetic sedation with triclofos 70 mg·kg^{-1} body weight orally one hour prior to induction. In the operating room anaesthesia was induced and maintained with halothane in oxygen via a Jackson-Rees modified Ayre's t-piece. Following the test procedure, surgery commenced.

Each child was tested while sedated and then during maintenance anaesthesia while breathing spontaneously. The apparatus consisted of a Rendall Baker mask, sealed to the face with soft silicon putty, with a port for measuring mask pressure (P). A pneumotachograph was connected to the mask and its output was connected to a pressure transducer whose signal was amplified and integrated. The flow ($\dot{V}$), volume (V) and P signals were displayed on an oscilloscope and recorded on magnetic tape. At end-inspiration the pneumotachograph was briefly occluded until P plateaued and then the occlusion was released. The passive expiratory time constant (τ) was calculated by the method of Lesouef *et al.*[3] from at least four end-inspiratory occlusion manoeuvres. Then a unidirectional valve was placed on the exit port of the pneumotachograph. The anaesthetic circuit was connected to the inspiratory port of this valve and the expired gases were vented to the room via the expiratory port. During quiet breathing at end-inspiration, the expiratory port was occluded which prevented expiration while inspiration of anaesthetic gases continued. When the subject had inspired at least three times the occlusion was released and C_{rs} was calculated by the method of Grunstein *et al.*[2] Total respiratory system resistance (R_{rs}) was calculated as τ/C_{rs}.[3] C_{rs}, R_{rs} and τ in each sedated infant were compared with the results for that subject during halothane anaesthesia by the paired t test and $p < 0.05$ was considered to indicate a significant difference.

Results

C_{rs} decreased approximately 48 per cent following induction of anaesthesia, and τ also decreased significantly (Table). R_{rs} did not change significantly following induction of anaesthesia with halothane.

Discussion

Using the new technique of volume recruitment to measure C_{rs} in healthy infants, we have shown that halothane anaesthesia decreases C_{rs} following induction, but does not affect R_{rs}. The decrease of approximately 48 per cent in C_{rs} was similar to the estimated increase in lung elastance in adults when anaesthetized with halothane.[1]

References

1 *Behrakis PK, Higgs BD, Bevan DR, Milic Emili J.* Partitioning of respiratory mechanics in halothane-anesthetized humans. J Appl Physiol 1985; 58: 285–9.

2 *Grunstein MM, Springer C, Godfrey S et al.* Expiratory volume clamping (EVC) a new method to assess respiratory mechanics in sedated infants. J Appl Physiol 1987; 62: 2107–12.

3 *Lesouef PN, England SJ, Bryan AC.* Passive respiratory mechanics in newborns and children. Am Rev Respir Dis 1984; 129: 552–6.

Timing of caudal block in relation to surgery does not affect recovery in paediatric ambulatory patients

M.A. Pudimat, L.J. Rice, R.S. Hannallah
Children's Hospital National Medical Center and George Washington University Medical Center, Washington, District of Columbia

In several recent studies caudal analgesia performed at the end of surgery was found to be an effective treatment for pain following hernia repair, orchiopexy or hydrocelectomy.[1,2] It has been proposed, however, that it may be preferable to perform such a block after induction of anaesthesia, but prior to the start of surgery. The purpose of this study is to determine if the timing of caudal block placement significantly affected postoperative pain or discharge time in ambulatory surgical patients.

Methods

Institutional approval and informed parental consent were obtained on 40 ASA physical status I or II children (ages 18 mo–11 yr) undergoing hernia repair, orchiopexy or hydrocelectomy. Anaesthesia was induced and maintained with N_2O/O_2/halothane in all cases. Children were randomly assigned to one of two groups. Group I received a caudal block with 0.5 ml·kg^{-1} of bupivacaine 0.25 per cent after the child was asleep but prior to the beginning of surgery, while Group II patients received a similar block at the completion of surgery but prior to the emergence from general anaesthesia. All patients were monitored in the Post Anaesthetic Recovery Room (PARR) by a research associate blinded to the timing of block placement. Pain was assessed upon arrival to the PARR and at 15-minute intervals, using a previously described Objective Pain Scale.[1] Fentanyl 1–2 μg·kg^{-1} was administered to any patient who complained of pain or who achieved a pain score of six or greater. Time of discharge from PARR, time of ambulation and time to meet criteria for discharge from the SSRU were all recorded. All parents were telephoned at home the day following surgery and questioned as to the need for further pain medication, presence of nausea and vomiting, and the return of the child's usual "bright and alert" status. Fisher's Exact Test and the t test were used to study the differences between the groups with respect to postoperative pain, need for additional pain medications, and time to meet discharge criteria.

Results

Patients in both groups were comparable with regards to age, duration of anaesthesia and surgery. No significant differences

TABLE Results

	Group I	*Group II*
Age (mo)	53.8	54.7
Duration of anaesthesia (min)	67.3	60.5
Duration of surgery (min)	37.0	33.1
Pain score in PARR	2.7	1.2
Fentanyl required (♡)	2(7%)	none
Discharge time (min)	198	204
Time until oral analgesic requested (min)	96	98

in postoperative pain/discomfort scores, need for additional pain medication (i.e., fentanyl), or time to discharge home were found between the two study groups (Table). There were no anaesthetic complications.

Discussion

Multiple studies have demonstrated the analgesic advantages of caudal blocks. However, some authors have raised the question of whether a caudal done prior to the surgical procedure would be more beneficial for ambulatory surgical patients than that done at the end of surgery. By contributing adjunct operative anaesthesia, such a block would reduce the general anaesthetic requirements, obviate the need for endotracheal intubation, and possibly shorten the recovery and discharge times. The results of this study failed to show any difference in the duration of postoperative analgesia and therefore suggest that timing of caudal placement did not alter the effectiveness of caudal analgesia for the time to recovery for these three commonly performed outpatient surgical procedures.

References

1 *Hannallah RS, Broadman LM, Belman AB et al.* Comparison of caudal and ilioinguinal/iliohypogastric nerve blocks for control of post orchiopexy pain in pediatric ambulatory surgery. Anesthesiology 1987; 66: 832–4.

2 *Wolf A, Valley RD, Fear DW, Roy WL, Lerman J.* The minimum effective concentration of bupivacaine for caudal analgesia after surgery in pediatrics. Anesthesiology 1987; 67: A509.

Computerized schedules – an answer to variable workstyles

C. Chong, H.K. Andrien, D.K. Rose

St. Michael's Hospital, University of Toronto, Toronto, Ontario

The increasing complexity within our own group of thirty anaesthetists, some full-time and some part-time, each with different areas of expertise, led to the development of a computerized call schedule. This assures coverage of all anaesthesia services: the general, cardiovascular and trauma operating rooms, obstetrics and the intensive care units.

There are three alternatives in developing a computerized system. The first involves a system to track calls assigned manually. The second, totally automated, generates a completed schedule if all call rules and doctor commitments are programmed. Unfortunately, rules cannot be formulated for every situation. The third alternative, our system, gives a priortized list of doctors available for a particular anaesthesia service and tracks calls assigned. Availability is dependent on holidays, time-off and the number and sequence of previous calls. Calls are equally distributed on a daily basis throughout the year. An anaesthetist is chosen dependent on hospital requirements, part-time variations, no call requests and updating. A five-week summary of an individual's schedule is displayed before a call is assigned.

Our hospital requires the first call anaesthetist to be in the hospital overnight and the second call anaesthetist available for emergency cases 24 hours, seven days a week. One of these two has expertise in trauma and cardiovascular anaesthesia. Six other anaesthetists (calls three through eight) are available five days a week after 4:00 pm to continue surgical cases. One anaesthetist provides 24 hour Intensive Care coverage seven days a week.

Part-time variations include a group of five anaesthetists who each work four out of five days a week, another group of five individuals who each work four out of every five weeks, one anaesthetist who works three days a week, another who takes no overnight calls and one who works no weekends or overnights. The number of calls is proportioned dependent on full-time or a percentage of part-time practice. One anaesthetist works no third through eighth calls to compensate for administrative time. The Pain Clinic anaesthetist takes no third or fourth calls. The six anaesthetists who cover the intensive care unit on a weekly rotation do not take operating room calls on weekends or during their intensive care unit week. Only half of the group does major cardiovascular and trauma cases.

Calls are not scheduled the day following late nights (first, second and third calls), weekends worked, and weekends prior to a holiday week.

If an individual is not available to do a scheduled call, updating is done, the one who takes the call is compensated and the "missed" call is added to a future schedule.

The program is written from "Revelation," a data base system made up of data entry templates and programs written in R/Basic. This system uses complex algorithms in assigning calls required to fulfill daily shift requirements. All information is stored on a hard disk. The program requires a fast personal computer, monitor and printer.

The reports generated include: a monthly schedule for calls one through eight and the intensive care unit, a personalized monthly schedule for each individual of the days on call, days away and holidays, a grid developed to show who is available to do calls each day, a call tracking report which lists the calls required to be done for fair proportioning and those which have been scheduled and a call updating print-out which lists the number of calls actually completed and the number of calls scheduled.

This computerized program has worked statistically for six months to schedule calls in this large complex group with multiple responsibilities. Advantages of the system include better distribution and proportioning of assigned calls, easier tracking, fuller reports, and minimal time to update or reschedule. The program allows flexibility in scheduling different workstyles and has the potential to be expanded for other part-time arrangements or different subspeciality coverage.