Correspondence

Masked bleeding posttonsillectomy with ondansetron

Sir—A 10-year-old girl, ASA I, weighing 23 kg, presented for an elective adenotonsillectomy as an inpatient. She had no significant previous medical or surgical history and no history of a bleeding diathesis. Following intravenous induction of anaesthesia (including a bolus of ondansetron 0.15 mg·kg\(^{-1}\)), a laryngeal mask was inserted. Bleeding from the tonsillar beds was marked intraoperatively and 150 ml blood loss was recorded from the suction apparatus. An infusion of 500 ml of crystalloid was given in theatre. There was no apparent bleeding when the surgery was completed and a full blood count and clotting screen sent intraoperatively were normal (haemoglobin 11.1, INR 1.2, APTT 1.0). In the immediate postoperative period, her recovery was uneventful and she remained in recovery for 1 h before returning to the ward with no evidence of further bleeding and had taken sips of water.

Five hours after surgery, the child vomited 220 ml of fresh red blood and was noted to have passed melaena by the nursing staff. Her observations showed that she had become progressively tachycardic and hypotensive preceding this, suggestive of a gradual bleeding process. On examination, her heart rate was 132 b·min\(^{-1}\) and blood pressure 80/60, she was pale and had cool extremities. A large clot was seen on the left tonsillar bed and active bleeding was visible. Initially, she was resuscitated with intravenous fluids (20 ml·kg\(^{-1}\) of gelofusin) and rewarmed with a forced air warming blanket prior to returning to theatre. A rapid sequence induction and intubation were performed without incident. The surgeon reported five brisk bleeding sites in the left tonsillar bed which were diathermied. A nasogastric tube was passed and 20 ml of blood aspirated. Transfusion of packed red cells 350 ml was commenced in theatre and continued postoperatively. There were no further complications in her recovery and she was discharged home 36 h later. Her postoperative blood count showed a haemoglobin of 10.2 g·dl\(^{-1}\).

Intravenous ondansetron has been shown to be a very effective antiemetic in children postadenotonsillectomy (1, 2). Concern has been expressed over the use of ondansetron for adenotonsillectomy since, in two cases, its use was thought to mask bleeding (3). Such concern was shown that the study was terminated prematurely. In spite of this, ondansetron is used by many anaesthetists for adenotonsillectomy. In our case, the child was observed postoperatively on a ward where regular observations were made. The first manifestation of ongoing tonsillar bleeding was the vomiting of swallowed blood, which occurred 5 h after surgery and was a significantly large volume. Delayed vomiting of blood may have been attributed to the antiemetic effects of ondansetron as described previously. Despite the delayed presentation, immediate resuscitation and rapid surgical correction occurred as the child was an inpatient. Had the child been discharged home from daycase surgery, further delay could have had a potentially more adverse outcome. We suggest that the use of ondansetron as a prophylactic antiemetic for adenotonsillectomy in children should be carefully considered, especially where the child might be a daycase.

SIR—A 3-year-old girl was admitted for removal of a mole from her right temple. She was fit and well apart from a slight cough. She was induced with midazolam 0.5 mg, fentanyl 5 μg, propofol 50 mg, and suxamethonium 25 mg, intubated with a RA 4.5 tracheal tube, and breathed spontaneously on 1–2% isofluorane. All observations were

References

1 Furst S.R., Rodarte A. Prophylactic antiemetic treatment with ondansetron in children undergoing tonsillectomies. Anesthesiology 1994; 81: 799–803.
2 Litman R.S., Wu C.L., Catanzaro F.A. Ondansetron decreases emesis after tonsillectomies in children. Anesth Analg 1994; 78: 478–481.
3 Hamid S.K., Selby I.R., Sikich N, Lerman J. Vomiting after adenotonsillectomy in children: a comparison of ondansetron, dimenhydrinate, and placebo. Anesth Analg 1998; 86: 496–500.
stable until the end of the procedure when suturing commenced and she suddenly became asystolic for 4 s. Cardiopulmonary resuscitation and atropine 200 μg were given, by which time her electrocardiogram (ECG) and cardiac output were returning to normal. She woke up and was extubated uneventfully.

In recovery, after being informed of the period of asystole, her mother volunteered that if her daughter bumped her head she would fall down limply for a few seconds. Being a nurse, her mother had felt the pulse and it did disappear. She was thereafter referred by her general practitioner to a paediatrician who performed a 24 h ECG which was normal. She is at present awaiting provocation tests (ocular pressure).

Anaesthetists commonly encounter bradycardias in response to surgical stimuli in operations on the eye, face, and abdomen (7). Children do have more pronounced bradycardic responses, hence the practice of some paediatric anaesthetists administering atropine as a premedication.

Are reflex anoxic seizures a separate pathology or simply one end of a spectrum of vagal sensitivity? How relevant are they to our anaesthetic practice? If a child has a history of fits prior to anaesthesia, perhaps we should bear in mind that these could be vagal in origin, and give atropine at induction or have it close to hand.

R. C. POLLARD
Department of Anaesthesia
John Radcliffe Hospital
Oxford, UK

Heart failure and ST segment depression in a child aged 6 weeks

Sir—We report a 6-week-old infant of 3.8 kg, who presented with a 4-week history of slow feeding, poor weight gain and breathlessness during feeding. The parents were first cousins by marriage.

The patient was admitted to hospital. Congestive cardiac failure was diagnosed. An electrocardiogram (ECG) showed abnormal T wave pattern across the chest leads (Figure 1). A chest X-ray showed an enlarged heart and an echocardiogram showed a structurally normal heart but a dilated left ventricle with markedly reduced function. The origin of the left coronary artery could not be clearly defined, though both the right and left main coronary arteries themselves appeared enlarged.

The child was commenced on dobutamine 5 μg·kg⁻¹·min⁻¹, diuretics therapy, and fluid restriction. Urgent cardiac catheterization was carried out. We induced with 25 mg of thiopentone and 2 mg of atracurium. The child was uneventfully intubated with a 3.5 mm uncuffed oral tracheal tube. Anaesthesia was maintained without a vapour with nitrous oxide and oxygen 50% to maintain saturation above 95%. 1 μg·kg⁻¹ fentanyl was also given.

CO₂ was maintained at 4 kPa (30 mmHg) and was monitored by capnography.

Angiography showed neither coronary artery arising from the aortic root (Figure 2). Instead, there was a single coronary artery arising from the pulmonary trunk, dividing subsequently into both right and left coronary arteries (Figure 3). The child was transferred intubated and ventilated to Intensive Care for postoperative care. She was transferred for definitive surgery but died in the postoperative period.

The child showed the signs of neonatal heart failure: sweating during feeding and slow and reluctant feeding. The adult signs of dyspnoea, gallop rhythm and raised jugular venous pressure are not seen in this age group.

The ECG demonstrates abnormal T wave pattern across the precordial leads. This reflects mild right ventricular hypertrophy and ischaemic myocardium. It is an uncommon ECG finding in infants. The classic Q waves seen in leads I, aVL and V3–6 were not present in our case. The present picture was of a dilated cardiomyopathy,
the resultant weeks. In the rare setting of total coronary artery perfusion arising from the pulmonary circulation, myocardial blood supply is critically dependent on pulmonary artery pressure and any manoeuvres leading to a reduction in pulmonary artery pressure (such as hypocapnoea, hyperoxia, or respiratory alkalosis) will lead to extensive underperfusion of the myocardium, infarction and death. Increased pulmonary vascular resistance is caused by atelectasis, hyperinflation, positive endexpiratory pressure, hypoxia, hypercarbia and acidosis. Meticulous attention should therefore be paid to these parameters.

During catheterization, the child’s condition was stable so we did not attempt to hypoventilate to raise pulmonary pressure.

For the few reported cases, death is common during the first weeks of life. Operative correction of a single anomalous coronary artery can give good results with resolution of heart failure and recovery of the left ventricular function. However, where the entire coronary artery supply arises from the pulmonary artery, anticipated surgical mortality will be high. Careful consideration should be given to whether corrective cardiac surgery or transplantation in the neonatal period is the best therapy.

An early diagnosis is important so that surgical correction can be offered. Anaesthetists should be aware of the importance of the pulmonary artery pressures in such patients, and avoid manoeuvres to further reduce myocardial perfusion.

OLIVER R. DEARLOVE
GORDON GLADMAN
S. MAGUIRE
Departments of Anaesthesia and Cardiology
Royal Manchester Children’s Hospital,
Hospital Lane,
Manchester M27 4HA, UK

A novel technique for securing lumbar epidural catheters in children undergoing hip surgery

SIR—We would like to recommend a novel technique for securing lumbar epidural catheters in small children undergoing hip surgery when there is limited access to the epidural site.

At our institution, we frequently use epidural catheters for perioperative analgesia in children undergoing open reduction of congenital hip dislocation. These children leave the operating theatre encased in a plaster of Paris hip cast which extends from the umbilicus to the ankles. Although the plaster cast remains on the child for a number of weeks, the lumbar epidural catheter is usually removed 48–72h after surgery. The catheter is initially inserted under
general anaesthesia immediately before surgery and is secured to the skin using a longitudinal strip of adhesive tape. Removal of the catheter on the ward is hampered by the overlying plaster cast and some force may be required to drag the catheter out from beneath the adhesive tape. There is always the fear that the catheter may break leaving a fragment in the patient. Catheter removal is therefore invariably frustrating, uncomfortable and time consuming for all involved.

The following technique has revolutionized our practice. After insertion of the epidural catheter, a small loop is fixed to the skin at the site of entry using adhesive spray (Opsite moisture vapour permeable spray dressing, Smith and Nephew Medical Ltd, UK). The entry point and loop of catheter are then secured to the skin using a small transparent dressing (Cutifilm, Beiersdorf, Germany). The remaining catheter is then secured parallel to the spine and onto the shoulder using the new technique (Figure 1). Starting at the shoulder a continuous strip of adhesive tape 2.5 cm wide (Transpore, 3M, USA) is placed over the catheter, securing it to the skin and overlapping the transparent dressing covering the catheter entry. The tape is doubled back to the shoulder, without it being cut, so that the adhesive surface lies outward. The tape is now doubled back as a third and final layer to just beyond the catheter entry site such that the adhesive surfaces of the two outermost layers of tape are in contact. There is now a ‘ripcord’ consisting of a double layer of adhesive tape at the shoulder.

Removal of the catheter is now simple. The ‘ripcord’ is pulled and the three layers of tape effortlessly appear from beneath the plaster cast. The unsecured epidural catheter is removed separately, in a similar fashion, or may come away with the innermost layer of tape. The Cutifilm dressing may or may not remain adherent to the skin; this is harmless and will invariably ‘come out in the wash’ later. This technique is simple and effective. The only potential disadvantage is the inadvertent pulling of the ‘ripcord’; this complication can be minimized by clear labelling and lightly taping the ‘ripcord’ to the back at the level of the shoulder blade.

**ALISON BLISS**

**IAN LEWIS**

*Shackleton Department of Anaesthetics*

*Southampton General Hospital*

*Tremena Road*

*Southampton SO16 6YD, UK*

© 1999 Blackwell Science Ltd, *Paediatric Anaesthesia, 9, 467–470*