Sedation of children undergoing magnetic resonance imaging

Editor—We were interested to read the correspondence between Allen\(^1\) and Sury and colleagues\(^2\) on paediatric sedation, having reported a series in 1999. Changes in our sedation regime\(^3\) were introduced in 2000. We have since tracked our results for sedation to facilitate MRI scanning in children. In the last five years from 2002 to 2006, we have performed 4165 sedations and 478 general anaesthetics for MRI scan (the general anaesthesia group being a mixture of those who failed sedation and those referred for general anaesthesia directly).

Our sedation technique can be summed up as oral chloral hydrate \(100 \text{ mg kg}^{-1}\) to a maximum of 2 g, with or without rectal paraldehyde \(0.3 \text{ ml kg}^{-1}\) for children weighing \(<20 \text{ kg}\), and oral quinalbarbitone (secobarbital) \(10 \text{ mg kg}^{-1}\) to a maximum of 200 mg for those weighing \(>20 \text{ kg}\). We have had five critical incidents compared with the nil critical incidents of Sury and colleagues,\(^2\) and this may reflect a different population undergoing sedation for MRI in the North West. Most of these were airway-led, as reported by Cote and colleagues.\(^5\)

We had five critical incidents compared with the nil critical incidents of Sury and colleagues,\(^2\) and this may reflect a different population undergoing sedation for MRI in the North West. Most of these were airway-led, as predicted by Cote and colleagues.\(^5\) This means that the critical incidents were, if not unavoidable, then treatable by standard means. During this time, we had one critical incident in the general anaesthetic group. This was a failure to anasthetize.

Our series is large, and critical incidents are to be expected. The correspondence\(^1\) sums up the current tensions in providing a service for sedation of children. Various points arise from it. The demand for sedation in a hospital is huge, and there is little interest in anaesthetists who like to anasthetize rather than sedate or supervise. In order to show safety, it is necessary to perform very large numbers of procedures and audit them as we have done. If ‘safe’ means zero critical incidents, then there will be a pressure to under report. ‘Safe’ we think is a situation where random events are treated by trained personnel in a timely fashion and without an adverse outcome.

Sury and colleagues\(^6\) refer to the use of melatonin, which did not contribute to sedation. The failure rate was 30\% in Wassmer’s series\(^7\) and we would rate this as so high as to make it impractical. Our failure rate was 11\%, 478 of 4165 patients.

Sury suggests that there is a widely held view by anaesthetists that anaesthesia is safer than sedation for MRI in children. MRI in children tends to be a standard non-painful procedure. Many sedations are performed by nurses under supervision, and most anaesthetics are performed by anaesthetists. We do not know how safe sedation services in the UK are, as results are underreported. Clearly, if we do a lot, we should be reporting our results.

We found that there was a group of children for whom it was safer to anaesthetize than sedate—children with mucopolysaccharidosis. Nonetheless, the waiting times for scan become quickly unmanageable if too many well-children are referred directly for general anaesthesia. Secondly, Malik and colleagues\(^4\) have reported a series of children who were assessed for stridor under general anaesthesia and then sedated for MRI scanning, who are included in this series.

Guaranteeing a scan after a single visit to hospital is possible, and general anaesthesia will figure greatly in this system. This is the one that Allen\(^1\) describes. We find that one session is suitable for around 150 scans yr\(^{-1}\). And so if one needs a thousand scans under sedation, to do it in this fashion would require 10 sessions—or the whole week. It is difficult to scale up a service to 10 times the volume.

A sedation service has to be safe; however, safety is open to interpretation and the service must also be available. Allen’s series of 200 children, although admirable, does not show this. The demand for sedation is such that if there is no anaesthetist to provide it or supervise it, the sedation will still go ahead, but provided by someone else without anaesthetic skills. Cote and colleagues\(^5\) have reported a long series showing how things can go wrong and how most critical incidents were airway led. Anaesthetists are well placed to treat those incidents that are most likely to occur during sedation, which are airway problems. Safe sedation services provide a challenge for the anaesthetist, a challenge to which we must rise.

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Acute lung injury and multi-organ dysfunction; an unusual manifestation of leptospirosis

Editor—Leptospirosis is a zoonosis—an infection that can be transmitted from animals to humans—and is primarily a disease of tropical regions. It is an uncommon, but notifiable, disease in the UK, <1 patient million$^{-1}$ yr$^{-1}$, yet two to three people in England and Wales die every year from the disease. Severe forms are associated with a very high mortality rate. Diagnosis is based on epidemiological history, clinical features, and investigations. It is often missed and easily overlooked because of the lack of awareness and the inability to obtain early laboratory confirmation. Adequate antibody titres appear only after a period of 5–7 days$^{2}$ and, in the UK, the specimen needs to be sent to the reference laboratory at Hereford. Leptospirosis should be a differential diagnosis in an acutely ill patient with appropriate risk factors and symptomatology. We present a patient of an uncommon presentation of leptospirosis.

A 59-yr-old male attended the hospital with a 5-day history of fever, lethargy, body pain, headache, productive cough, and decreased appetite. He lived independently, smoked 30 cigarettes a day, and was keen on fishing in the nearby canal. He was in good health with no significant past medical or surgical history and had not recently travelled outside the UK.

On examination, he was alert, oriented, and icteric. Temperature was 37.7°C, heart rate 109 beats min$^{-1}$, arterial pressure 93/53 mm Hg, and well-perfused peripheries. His ventilatory frequency was 19 min$^{-1}$ with an oxygen saturation of 96% on room air. On auscultation, his heart sounds were normal and air entry were equal on both lungs with occasional scattered wheeze.

Initial investigations revealed a white cell count of $7.0 \times 10^{9}$ litre$^{-1}$, platelets $52 \times 10^{9}$ litre$^{-1}$, haemoglobin 13.2 g dl$^{-1}$, urea 22.5 mmol litre$^{-1}$, creatinine 262 μmol litre$^{-1}$, bilirubin 30 μmol litre$^{-1}$, GGT 83 IU litre$^{-1}$, and CRP of 339 mg litre$^{-1}$. ECG showed normal sinus tachycardia and chest X-ray revealed mildly increased bronchoalveolar markings. An initial diagnosis of sepsis with lower respiratory tract infection and acute renal impairment was made and he was transferred to a medical ward.

Within a few hours, his condition deteriorated; he became breathless, hypoxic, and hypotensive (arterial pressure 80/50 mm Hg) despite a litre of colloid and crystalloid infusion. His arterial gases were, pH 7.47, $P_{aCO_2} 3.3$ kPa, base excess $-4$ mmol litre$^{-1}$, bicarbonate 17 mmol litre$^{-1}$, $P_{aO_2}$ 8.6 kPa, $S_{aO_2}$ 94% on oxygen 15 litre min$^{-1}$. He was transferred to a critical care ward.

Facial continuous positive airway pressure (CPAP) and invasive monitoring was initiated. Noradrenaline infusion was started to maintain a mean arterial pressure (MAP) of $\geq 70$ mm Hg. Despite these measures, his condition worsened rapidly. Within 2 h, he became exhaustsed, more hypoxic, and acidotic (blood gas, $F_{IO_2}$ 0.9, pH 7.34, $P_{aCO_2}$ 4.8 kPa, base excess $-6.4$ mmol litre$^{-1}$, bicarbonate 19.4 mmol litre$^{-1}$, $P_{aO_2}$ 7.7 kPa, $S_{aO_2}$ 84%). Investigations and chest X-ray were repeated. Repeat chest X-ray revealed an extensive bilateral alveolar shadowing (Fig. 1), simulating acute respiratory distress syndrome (ARDS). At this point, his trachea was intubated.

Repeat investigations revealed worsening of renal (creatinine 338 μmol litre$^{-1}$, urea 26.2 mmol litre$^{-1}$) and hepatic (bilirubin 114 μmol litre$^{-1}$, GGT 98 IU litre$^{-1}$, alkaline phosphatase 131 IU litre$^{-1}$, albumin 22 g litre$^{-1}$, aspartate transferase 59 IU litre$^{-1}$) function. Platelets fell to $23 \times 10^{9}$ litre$^{-1}$, activated partial thromboplastin time was 35.5 s, prothrombin time 10.4 s, and lactate 1.2 mmol litre$^{-1}$. Abdominal ultrasound revealed no abnormalities, and a cardiac echo showed a dilated and poorly contracting right ventricle with normal left function.

![Fig 1](Acute bilateral alveolar shadowing in leptospirosis.)