OVERDOSE OF OPIOID FROM PATIENT CONTROLLED ANALGESIA PUMPS

Sir,—In our hospital there have been two episodes, at an interval of 1 year, of Patient Controlled Analgesia (PCA) pumps delivering the entire contents of a 60-ml syringe of opioid as a bolus (diamorphine 60 mg over approximately 40 min). Both patients were receiving PCA for control of postoperative pain and were being managed on general surgical wards according to our standard policy [1]. The nurses were alerted by the “Syringe Empty” alarm of the pumps. Both patients suffered severe respiratory depression, but were successfully resuscitated. They made satisfactory recoveries and suffered no ill effects.

Both the episodes were reported to the Department of Health, who inspected the pumps. The pump manufacturers, Graseby Medical, Watford, England, traced both faults to corruption of the pumps’ software by either mains electricity fluctuations or static electricity discharges from the patients’ beds. Corruption of the software from both these sources had often been observed in our hospital. However, the pumps had previously always “failed-safe.” After both incidents, Graseby carried out modifications to all their pumps.

PCA has been widely used for many years. There are only two other such episodes described in the literature. The first related to syphoning from a cracked syringe and the second is of uncertain aetiology [2, 3]. A third such incident occurred in Tasmania in 1990 [personal communication]. Nearly 5000 patients have used PCA from 31 PCA pumps in this hospital, reflecting some 200000 h use. However, this must be set against the huge worldwide usage of PCA and the Graseby pumps.

We are continuing to review our management of PCA as part of a wider review of patients requiring High Dependency Care on general wards. We have continued to use PCA as our main postoperative analgesic technique for severe pain. We urge manufacturers to continue to improve the fail-safe mechanisms in their equipment.

Full details of both incidents will be published as a Case Report.

W. NOTCUTT
Great Yarmouth


THE EPIC STUDY

Sir,—On behalf of the EPIC Study Advisory Committee, I would like to draw the attention of all specialists in intensive care to the European Prevalence of Infection in Intensive Care (EPIC) Study and urge all intensive care units to participate in this study on April 29, 1992.

The EPIC Study is the largest of its kind to look at the problem of nosocomial infection in intensive care units from a European perspective and will take place in 17 countries throughout Western Europe. This will be the first time that the prevalence of infection in the ICU has been related to the severity of illness of the patient on admission, in combination with an assessment of the adequacy of treatment in patient survival. The results published will be a major contribution to the literature on the subject, and should provide many new insights into the course of infection in susceptible patients, and the likely outcome.

This is a one-day prevalence study which has been chosen as the simplest, most cost effective and least disruptive study method. The guidelines developed by the Centers for Disease Control for the diagnosis of nosocomial infection will be used. These definitions can be used for prevalence surveys and outbreak investigations in addition to routine surveillance of nosocomial infections [1]. Using the same definitions and similar surveillance methods will enable comparisons to be made with NNIS data.

It is hoped that the European database generated by the EPIC Study will have a significant role in the assessment of future infection control procedures. Nosocomial infections in the intensive care unit have a major cost in terms of patient morbidity and mortality and also in economic terms. In 1989, the average associated cost for a nosocomial infection was estimated at US$1800 [2]. More recently in Switzerland, hospital infection has been estimated to cost in the range of SF 100-300 million per year [3].

The EPIC Study is being planned and directed by the EPIC Study Advisory Committee, made up of 17 members from Western Europe. The study is funded by Roussel Uclaf as part of their commitment to the control and management of hospital infection.

It is particularly important that as many units as possible contribute data, so that the results are representative. For further information, please write to EPIC Study Co-ordinator, Medical Action Communications Ltd, Action International House, Crabtree Office Village, Eversley Wey, Thorpe, Egham, Surrey TW20 8RY (U.K.); Fax: 0784 431323. Units which have already registered will receive information automatically.

D. BIHARI
EPIC Study Advisory Committee


COMBINED SPINAL—EXTRADURAL ANAESTHESIA

Sir,—Having interests similar to those of Dr Carrie, I was interested to read his review [1] and the subsequent associated correspondence [2, 3]. I should like to respond to Dr Carrie’s final paragraph, in which he invited further revelations of the combined spinal and extradural (CSE) technique.

Prompted by Brownridge [4], I believed that a low spinal combined with a higher extradural block would be efficacious during elective Caesarean section, but it seemed more logical to use the Tuohy needle as the introducer. Difficulty was experienced in obtaining long enough spinal needles but eventually, with a Steriseal 110-mm length 25-gauge metal spinal needle, the CSE was underway; the earliest anaesthetic record I can find is dated November 2, 1981.

I embarked on a formal study in 1982-83, accumulating approximately 80 cases and presented the work at an open meeting of the Munro Kerr Society (for the Study of Reproductive Biology) in association with the Sesquicentennial anniversary of the Glasgow Royal Maternity Hospital in 1984. It is pertinent to state that, in our hospital at least, the advent of spinal anaesthesia as first choice for elective Caesarean section stems from this introduction of CSE and, from 1983 onwards, this form of block superseded the extradural variety.

I now use CSE for deliveries for which the duration of operation may be difficult to judge, in which assessment of spinal dosage may be problematic because of abdominal size (for example prematurity) and in multiple pregnancy.

I commend Dr Carrie for his work and writings on this topic and thank him for this chance to contribute towards it.

D. J. M. FERGUSON
Glasgow

Sir,—On reading Dr Saddler's editorial on masseter spasm, I was surprised to find no mention of relevant studies on muscles of the hand [1]. I was also a little puzzled by this, as recent work from his own department has shown that there is an excellent correlation between the tension changes occurring in the muscles of the hand and those in the jaw [2].

It seems likely that the high incidence of clinically evident masseter spasm which has been reported in some children's hospitals [3, 4] involves a difference in response to suxamethonium between children and adults, together with differences in clinical practice. Studies at our hospital suggest that children require at least 2 mg kg⁻¹ of suxamethonium to produce conditions for tracheal intubation comparable to those obtained with a dose of 1 mg kg⁻¹ in adults [5, 6]. In addition, the previously recommended dose for children of 1 mg kg⁻¹ [7] was shown to produce a very brief period of neuromuscular block in some patients, while producing the maximum initial increase in muscle tone. Taken together, these results indicate that use of suxamethonium 1 mg kg⁻¹ could give rise to inadequate neuromuscular block in some children who would, nevertheless, exhibit maximum muscle spasm. It is not difficult to see how, in the presence of light anaesthesia, such children might be thought to exhibit an abnormal response to suxamethonium.

G. MEASKIN
Manchester


Sir,—Thank you for the opportunity to reply to Dr Meakin's letter.

I did mention that the increase in stiffness may occur in other muscle groups, but did not go into great detail as the editorial was concerned primarily with the behaviour of the jaw muscles.

Much of Dr Meakin's argument is probably valid. However, the masseter muscle may not necessarily behave in the same way as the adductor pollicis muscle. For example, we found that the masseter muscle in children was more sensitive to suxamethonium, with an ED₅₀ of 0.25 mg kg⁻¹ [1]. We concluded at the time that it is very unlikely that masseter spasm is due to a failure to block neuromuscular transmission at the massetermuscle.

In addition, although Dr Meakin demonstrated that tone changes at adductor pollicis do not increase with large doses of suxamethonium, we found that suxamethonium increased masseter tone in a dose-related fashion up to 1 mg kg⁻¹. It would be interesting to assess tone changes at the jaw with larger doses.

The point that some children metabolize suxamethonium very quickly is a good one. In this situation, the jaw may appear stiff because the muscle has already begun to recover.

J. M. SADDLER
Southampton


TENSION PNEUMOTHORAX—DETECTION AND INCIDENCE

Sir,—We read with great interest the article by Laishley and Aps [1] and the subsequent comment by Kneeshaw [2]. The former, however, did not discuss methods of early detection and preventative measures.

It is difficult to maintain the head in a neutral position when a patient is moved from the supine to a lateral position, as the head tends either to flex or to extend. It is well known that flexion or extension of the head in patients with a tracheal tube may cause considerable movement of the tube [3] and may dislocate the tip of a left-sided, double-lumen tube over a distance of 27 mm in either direction [4]. Therefore one should check for displacement of the tip of the tube after a patient is placed in the lateral position, even if correct placement had been confirmed in the supine patient, by bronchoscopy or x-ray.

One can easily detect the onset of a ball valve mechanism by a change in lung compliance during manual ventilation, inability to perform right bronchial catheterization, and deflation of the cuff of the endobronchial tube before tension pneumothorax is established. These manoeuvres may be performed within minutes. Had this been the case in the patient reported, tension pneumothorax might have been prevented.

Fortunately, we have encountered only two cases of development of pneumothorax among some 30000 general anesthetics given at our hospital. We were able to detect pneumothorax in its early stages by minor changes in pulmonary compliance during manual ventilation and via frequent and deliberate auscultation of the lungs. These occurrences were treated successfully before circulatory derangements could ensue.

Y. KUBOTA
Y. TOYODA
H. KUBOTA
Osaka, Japan


Sir,—While it is pleasing to see that our article has raised some thought and correspondence, it must be remembered that it is always easy to be wise and critical after the event. We concluded, in addition to the other correspondents, that malposition of the endobronchial tube and cuff herniation, acting as a ball valve, were likely to be responsible for the development of tension pneumothorax. We hope that the publication of our article has at least served its purpose in reminding anaesthetists that tension pneumothorax should always be considered in the differential diagnosis of muscle arteriole desaturation during anaesthesia. Moreover, it underlines the value of pulse oximetry as mandatory monitoring during anaesthesia.

R. S. LAISHLEY
London