Anaesthesia, 1998, **53**, pages 511–517

Correspondence

Ephedrine/Epinephrine drug label confusion

When the European Commission's decision [1] to require the UK to label drugs with their 'recommended International Non-proprietary Name (rINN)' was first publicised, there were fears that errors and confusion would result [2, 3]. In particular, the potential for confusion between 'Epinephrine' (adrenaline) and 'Ephedrine' was mentioned [4]. That has now been reported, to our anaesthetic incident audit, twice in one day, from neighbouring operating theatres.

An enthusiastic operating department practitioner was aware of the new nomenclature. When the syringe labels for adrenaline ran out, concerned to avoid the confusion with atropine, he ordered labels reading epinephrine. Twice in one day, anaesthetists who used ephedrine for treating hypotension labelled their syringes 'epinephrine'. In one case, the error was only recognised when the syringe was being discarded. Neither patient came to any harm. The error occurred despite a circular letter, sent to all anaesthetic staff some months earlier, drawing their attention to the new nomenclature. Appeals to 'read the label' [5] will always fail sometimes, as it is well known that we read by recognising word shapes, not their individual letters. Hopefully, as we learn the new shape, the errors will become rarer. Good publicity at the time of any change would seem important. Meanwhile, beware!

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Thoracic epidural analgesia and coronary artery bypass graft surgery

We read with interest the recent retrospective analysis in which Turfrey *et al.* (*Anaesthesia* 1997; **52:** 1090–5) reported a lesser incidence of arrhythmias after thoracic epidural analgesia in patients undergoing coronary artery bypass graft surgery. Broadly similar beneficial results including earlier extubation, attenuated catecholamine response, improved postoperative analgesia and pulmonary function have been shown by several other authors using thoracic epidural analgesia [1, 2]. Despite the purported potential benefits, it needs to be emphasised that only few authors recommend routine use of this technique.

In the above study the epidural catheter was inserted immediately before induction of anaesthesia and prior to full systemic heparinisation while in most other studies the catheters were placed at least 12–24 h before scheduled surgery [1–3]. It raises concern as to the safety of inserting the catheter only 1–2 h before systemic heparinisation. To date there has been no report of epidural haematoma in patients receiving epidural analgesia for coronary artery bypass graft; nevertheless, it needs to be acknowledged that there is such a risk. Spinal haematoma has been reported in many cases in whom diagnostic lumbar puncture was followed by intravenous heparin within an hour [4] and recently in a patient associated with intra-operative use of heparin [5]. No matter how meticulous the technique with which the epidural space is invaded, the incidence of bloody tap – in the order of 1–11% [6] – needs considered evaluation. In the absence of predictability of a difficult or traumatic procedure and hence the risk of epidural haematoma, it would seem prudent to delay surgery for 24 h in such an event to ensure safety of the patients and to avoid the risk of such a devastating consequence.

We are at a loss to understand how ethics committee approval and informed consent were obtained for this retrospective study. Did the authors mean a routine consent for surgery as against one specifically to study the effects of epidural analgesia in this group of patients?

Prospective randomised controlled trials have failed to show any real advantage in terms of outcome and recovery parameters conferred by the addition of thoracic epidural anaesthesia to medium

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or lower dose opioid-based general anaesthesia in patients undergoing coronary artery bypass graft [7–9]. Similar earlier studies have shown that patients receiving epidural infusion for intra- and postoperative analgesia exhibited significantly increased pulmonary artery wedge pressure [10], significant decrease in arterial blood pressure and in coronary perfusion pressure [11] and suppressed cortisol response [12] when compared with patients without epidural analgesia. The authors could have analysed these issues in their epidural group in order to evaluate objectively the benefits of this technique. We feel that the risks, albeit rare, of spinal haematoma and paraplegia are to be taken more seriously.

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In one of the earliest clinical reports of cardiopulmonary bypass for cardiac surgery, a combined epidural and general anaesthetic technique was employed for one of the patients [1]. Yet, in the four decades since this report, the use of epidural anaesthesia for cardiac surgery has found little favour in clinical practice [2]. Recently, there has been an upsurge of interest in this controversial technique. Unfortunately, we fear the paper by Turfrey and colleagues (*Anaesthesia* 1997; **52:** 1090–113) may have done more to muddy, than clarify, the controversy.

Our concerns are the result of the study's methodology and statistical analysis. Retrospective dredging of data, which is a notorious source of error, was employed by Turfrey and co-workers. Using a 5% level of probability, one in 20 variables will be significant by chance and this factor could have contributed to their findings. Another drawback to analysing retrospective data is that it lacks the control of binding. It is well recognised that 'the investigators natural enthusiasm for a new treatment may well influence his judgment of the patients progress and may also be transmitted to the patients and affect their well being, especially for conditions where symptoms are subjective, such as the degree of pain' [3]. Turfrey and colleagues finding of an earlier extubation time in the epidural group may well be a reflection of their enthusiasm for thoracic epidural anaesthesia. An even more serious danger of reviewing data retrospectively is the absence of randomisation which is essential to prevent patient selection bias on the part of the anaesthetists. If the anaesthetists had been less willing to use epidurals on sicker patients, e.g. those with unstable angina, then this may well have produced the difference in rates of arrhythmias. Indeed, the fact that patients with abnormal coagulation studies, presumably due to anticoagulation, were excluded from the combined epidural and general anaesthetic, but not the general anaesthetic group, supports this view. With regards to the statistical analysis, it is unclear from the results whether a correction for multiple testing, such as Bonferroni's, has been applied. If not, then simple multiplication of the probability (0.02) for the difference in the rates of arrhythmias between groups by the number of variables ($n = 11$) gives a nonsignificant value of 0.22. Given these flaws, it is neither possible to draw any valid conclusions from this study nor has it established the need for a large, prospective, randomised study.

Our major misgiving regarding the use of epidurals for cardiac anaesthesia is whether the benefits of the technique outweigh the drawbacks. When assessing the benefits, cardiac anaesthetists should be mindful of high-dose opioid anaesthesia which had many putative beneficial effects but failed to improve

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outcome of surgery [2]. On the other end of the balance is a catastrophic drawback and that is paraplegia resulting from an epidural haematoma. Undoubtedly, Ruff and Dougherty have found an association between diagnostic lumbar puncture and epidural haematoma which is compounded by traumatic puncture, early anticoagulation and aspirin therapy [4]. Although large series have been reported in other areas of anaesthesia without epidural haematoma, reports from cardiac anaesthesia have been in small populations [5]. The incidences that Turfrey and co-workers quote for haematomas resulting from the use of epidurals in other areas of anaesthesia, such as obstetrics, are not extrapolatable to cardiac anaesthesia. Far more profound levels of anticoagulation are used for cardiopulmonary bypass than elsewhere and this is often compounded by the development of serious coagulopathies. In reality, the incidence of epidural haematoma is unknown in the setting of cardiac anaesthesia. What then would be an acceptable incidence of epidural haematoma. Turfey and colleagues suggest that the risk of epidural haematoma is less than 1 in 10 000 which would fall into Calman's verbal description of risk as 'very low' [6]. However, the risk may be in reality low (1 in 1000 to 1 in 10 000) or, worse, moderate (1 in 100 to 1 in 1000), as these levels of risk cannot be excluded by their study. Given the devastating nature of paraplegia, low and moderate risk of epidural haematoma is unacceptable unless there is some great benefit to be gained from the technique such as a reduction in mortality or serious morbidity.

Perhaps, it is time for cardiac anaesthetists to overcome more than four decades of reservation regarding the use of epidural anaesthesia. But if their long-held concern is to be overcome then it will require, as Turfrey and colleagues suggest, a robustly designed trial. The aim of any such trial must be to determine whether combining epidural with general anesthesia reduces mortality, serious morbidity, i.e. myocardial infarction or both and, if so, is the risk of epidural haematoma at an acceptably low level.

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A reply

Thank you for giving us the opportunity to reply to the comments made by Doctors Mallik and Bhaskaran and Doctors Alston, Sinclair and Scott.

Doctors Mallik and Bhaskaran ask about our informed consent. We gain informed consent from all patients in this hospital who undergo any procedure. This involves a description of the type of anaesthetic and surgery they will be receiving. It is the hospital's standard practice that *all* studies are presented to the Ethics Committee to gain approval before data from patients' medical notes are examined and analysed statistically and this was done for our retrospective study.

Drs Alston, Sinclair and Scott worry about bias in the study in favour of the epidural technique, yet our demographic data clearly showed no differences between the two groups and our extubation criteria were well defined. We have only presented objective data. Our 'natural enthusiasm' was tempered by the deliberate omission of such subjective data as the degree of pain and its relief. This did not therefore appear in the paper and we are unsure as to why it is mentioned.

As Drs Alston, Sinclair and Scott state, the risk of epidural haematoma in patients who receive epidural analgesia for coronary artery bypass graft surgery is unknown, but collective data have now been published on nearly 2000 patients without a documented haematoma. In addition, spinal and epidural haematoma can occur spontaneously even in patients who have never received neuraxial blockade or heparinisation [1–5]. The well-documented risk of death (4%) and major complications such as postoperative myocardial infarction and cerebrovascular accident (4%) following this type of surgery is high enough that it requires to be fully explained to patients before they sign a consent form. We believe that the risk of epidural haematoma is much lower than 1% and does not require specific informed consent. Thus, put into context, if, for the sake of argument, the risk of haematoma is 1 in 1500 then for each haematoma that occurred, 60 patients would die and 60 would have a stroke as a direct consequence of the surgery. If it is possible to decrease the risk of common postoperative complications, with only a minimal added risk of epidural haematoma, then we feel the technique should be investigated further.

Doctors Mallik and Bhaskaran state that they feel the catheter should be sited 12–24 h before the onset of surgery. Whilst we accept that some institutions do site their catheters earlier than we do, similarly there are other centres that site their catheters immediately prior to surgery [6–8]. We do not accept that the risk of bloody tap in the hands of experienced operators is in the order of 1–11%. Certainly in our experience of over 300 thoracic epidurals for cardiac surgery to date the incidence of bloody tap through the needle is 1 in 300. Our protocol states that in this situation the operation, and therefore heparinisation, is deferred as agreed in other studies [9–11]. In the presence of normal clinical history and coagulation studies,

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we believe that any bleeding that may have occurred on insertion of the catheter, whether recognised or not, will have ceased prior to heparinisation [10, 12].

There has not yet been a large prospective study performed to look specifically at the differences between the two techniques. When examining relatively rare complications the power of any study involving small numbers is unlikely to be statistically useful. In addition, the abstract by Fillinger *et al.* quoted by Drs Mallick and Bhaskaran as evidence against an epidural technique is very misleading since it did not measure outcome data despite the title! Whilst we are familiar with the papers by Thorelius, Stenseth and Moore, we were not able to collect these data in our epidural patients, as it was a retrospective study. All other studies suggest that there may be benefits to be gained for patients by using this technique and currently we are performing a large prospective study to address this lack of information. We are also assessing some of these missing parameters.

The concern over epidural haematoma has arisen in the literature because epidural techniques were developed initially in the obstetric population. In this situation where pain relief is the major advantage of the technique and mother and child have a combined life expectancy in the region of 120 years, a conservative approach to avoid even one case of epidural haematoma is only right and is completely endorsed and reinforced by ourselves. Major surgery, however, particularly in the elderly or high-risk population, carries significant morbidity and mortality much of which may in fact be diminished by regional sympathetic blockade [13] and cortisol inhibition, in contrast to the thoughts of Doctors Mallik and Bhaskaran. A review of the literature has, therefore, led us to believe that postoperative morbidity may be reduced by careful use of regional anaesthetic techniques.

We do not wish to be seen to be complacent and every possible effort is made to minimise the progression of an epidural haematoma in our patients, should one develop. This can only be achieved by both the full support of one's surgical and nursing colleagues and careful and meticulous monitoring. We have strict protocols for neurological assessment and until further data are collected we would not support the practice without all of these criteria being fulfilled.

That having been said, those of our anaesthetic colleagues who express such concern about so rare an event fail to appreciate that conventional analgesia with opioids carries a significant risk also, especially in the high-risk and elderly populations. Not one outcome study in over 100 in the published literature has demonstrated that narcotic analgesia is safer than, or associated with less morbidity than, regional anaesthesia [13]. Rather than being condemned as 'regional enthusiasts', we are keen to produce objective evidence for or against the use of either technique for this form of surgery. We believe we have tackled this controversy appropriately, namely an initial thorough literature search followed by a pilot group of patients and a subsequent large prospective study.

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A simple approach to dosing in children

Anaesthetists who care for children must frequently calculate dosages of drugs on a $mg.kg^{-1}$ basis. Such calculations are open to error resulting in under or over dosage. A way to avoid this type of calculation altogether is to employ suitable dilutions of drugs and administer them on a $ml.kg^{-1}$ basis.

Rocuronium is a recent example of a drug whose dose ($0.6\,mg.kg^{-1}$) does not lend itself to easy calculation [1]. My

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approach to this is to dilute 30 mg of rocuronium (3 ml) to 5 ml with sterile water and to administer 0.1 ml.kg^{-1} (1 ml per 10 kg). Another example is the dose of glycopyrrolate (10 μg.kg^{-1}) and neostigmine (50 μg.kg^{-1}) [2]. Here I dilute the contents of the standard ampoule (500 μg glycopyrrolate and 2500 μg neostigmine) to 5 ml with water; again the dose is 0.1 ml.kg^{-1}. Morphine is a special case since we must be concerned with both loading and maintenance doses: 100 μg.kg^{-1} and 25 μg.kg^{-1}.h^{-1} respectively for children aged over 6 months [3]. The loading dose can be achieved by diluting 10 mg of morphine to 10 ml with sterile water and giving 0.1 ml.kg^{-1}. Hourly maintenance doses for children weighing less than 40 kg are prepared by discarding all but the initial dose of morphine from the syringe and rediluting this dose to 4 ml. The syringe now contains four maintenance doses of 1 ml each.

Providing syringes are properly labelled, the use of these techniques should reduce the potential for gross error in paediatric dosing.

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Asthma: a plea for commonsense

The report by Padkin *et al.* [1] describing halothane to treat asthma raises important points as it is a 'fringe' treatment. A trend has developed in hospitals to undertreat status asthmaticus, reflecting concern with perceived dangers of the drugs used, but ignoring the high risks of unrelieved asthma caused by mincing about with feeble measures.

Take aminophylline: here it has fallen somewhat from favour due to a narrow therapeutic index, adverse reports and medicolegal cases. The dangers are overstated. It is recommended by the British Thoracic Society guidelines and in higher dosage by the *Oxford Handbook of Critical Care*, *The Merk*' manual and yet higher dosage by *The (Australian) Intensive Care Manual Ed. 3* (with usual cautions). I have used it for 26 years with safety and efficacy, in asthma and chronic obstructive airways disease, as 6–7 mg.kg^{-1} slow bolus followed, if necessary, by infusing 0.5–1.25 mg.kg^{-1}.min^{-1}. Serum levels are unnecessary if usage is under 24 h, unless the patient already takes theopylline; caution then dictates using lower doses and measuring levels.

Persistence with nebulised bronchodilators in patients who have probably already languished for hours shows a poor understanding of the pathophysiology. If no improvement occurs in a reasonable time, say 15 min, then bronchospasm and bronchiolar oedema are so bad that no nebulised drugs will enter these airways to do any good. Intravenous drugs should be given instead – salbutamol, aminophylline or adrenaline. The authors gave a half-dose of aminophylline. Unsurprisingly the lack of benefit seen corresponded with the low serum level (36 μmol.l^{-1}). If a bigger dose of aminophylline had not helped, I would then have given adrenaline 1/10 000 very slowly intravenously at 0.5–1 ml.min^{-1}, titrating rate against tachycardia, hypertension, arrhthymias, bronchospasm and SaO_2. Naturally 100% humidified oxygen, steroids and rapid intravenous rehydration are prerequisites.

The 'fringe' treatments re-reported recently, magnesium, ketamine and now halothane, do work, but are superfluous unless conventional drugs in maximal doses fail. Also halothane is incompatible with adrenaline; cardiac arrest may occur. It is not something every registrar should try! Intravenous aminophylline and adrenaline remain established in treating severe asthma. Adrenaline in particular is life-saving and, when nothing else has helped, can rescue an exhausted patient from the risks and expense of intubation and ventilation.

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Footnote: This letter was shown to Dr Padkin and colleagues who declined the opportunity to reply.

Ilioinguinal nerve block for orchidopexy

Findlow *et al.* (*Anaesthesia* 1997; **52:** 1110–3) state that 'Ilioinguinal nerve block is free of the side-effects of lower limb motor block'. This is not the case. Femoral nerve block has been described after ilioinguinal nerve block in children in two patients [1, 2] and in a series of 81 children having ilioinguinal block, three had femoral nerve block afterwards [3]. In adults this complication is also described [4]. Undetected femoral nerve block after ilioinguinal block for hernia repair has led to serious injury [5] when patients start mobilising postoperatively. It does not seem to be routine practice to test for femoral nerve block after ilioinguinal nerve block even though it is a recognised complication with potentially serious consequences if undetected.

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Self-administration of pre-operative analgesic suppositories

In a recent letter to *Anaesthesia* (1988; **53:** 91), Jolliffe suggests that others might consider adopting the recent change in practice of patient self-administration of pre-operative suppositories. I have been using this method for just over 4 years in all cases where analgesic suppositories are indicated, with the exception of children, in both the day-case unit and in-patient wards. From over 1000 patients self-administering I have had two refusals that I can remember, both of whom were women who did not want a suppository under any circumstances. The usual response when broaching the subject is a smile and all are relieved when they understand its reason and they can give it to themselves. I do not believe there is much additional work for nursing staff and it is quicker for them to supply the suppository, glove and gel and let the patient get on with it. In fact, everybody is happy and the patients are still smiling, pain-free, in recovery.

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Sevoflurane and adult acute epiglottitis

The role of sevoflurane in difficult airway management is already established [1, 2] including its use in a child with acute epiglottitis [3]. We have recently used sevoflurane for induction of anaesthesia to secure the airway in a 35-year-old man with acute epiglottitis. He presented with a typical history in the sitting position with mild stridor and drooling saliva. The gradual deterioration of the airway in the 3 h after presentation necessitated tracheal intubation. He was transferred to the operating theatre and anaesthesia was induced with 3–8% sevoflurane in 100% oxygen. As he rapidly became apnoeic, the inhaled concentration was reduced (4%) and after gentle manual ventilation via the facemask, his trachea was successfully intubated with an 8-mm tube without any difficulty. Later, he was transferred to the intensive care unit for further management. At direct laryngoscopy the epiglottis and arytenoids were noted to be grossly swollen.

Sevoflurane is a more potent respiratory depressant than halothane [4] and its suitability for use in difficult airway management has been questioned [5]. A comparison of induction, however, using a vital capacity breath technique versus a tidal breathing technique found no breath holding in either group despite high concentrations of sevoflurane [6]. Today's trainees probably have greater experience with sevoflurane than halothane for inhalational induction of anaesthesia and they are more likely to use it in potentially difficult situations. Our experience illustrates the need for caution in the use of sevoflurane when the airway is endangered.

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Warning notices above beds

Notices above the beds of patients are widely employed on general wards as reminders to nursing and other health-care staff of particular considerations which apply to that patient (nil by mouth, sips, clear fluids only, named nurse, etc.). While clearly important for correct patient care, none of the signs in common use in our hospital relates to events which are potentially life threatening.

In the Intensive Care Unit, patients are often admitted with acute neurological conditions, such as Guillain–Barré syndrome, in whom the administration of suxamethonium could have fatal results. As suxamethonium is the muscle relaxant of choice for emergency tracheal intubation, it is easy for medical staff to overlook this most serious,

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potential complication at the time of intubation. We therefore employ the warning notice shown at the bedside of at-risk patients, as a simple but effective reminder for all staff. Such warning notices are not currently available on general wards in this hospital, but as it is occasionally necessary to intubate the tracheas of similar patients prior to transfer to the Intensive Care Unit, it is possible that it could also usefully be employed there when patients with acute neurological conditions are admitted.

Intensive Care Units have variable casemix and each unit may be able to identify similar, potentially hazardous interventions for which warning notices may be helpful. In our Intensive Care Unit, we admit many immunosuppressed patients in whom the administration of cytomegalovirus (CMV) positive blood products is undesirable. We have considered producing warning notices to cover this and other similar situations.

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Old habits, the circle system and short procedures

The suitability of the circle system with low flows is not dependent on the blood gas solubility coefficient as claimed by Nel *et al.* [1]. What they had demonstrated is that their technique of low-flow anaesthesia is only suitable for agents with low blood gas solubility coefficient. Their end point for dropping from high to low flow was an F_I/F_E ratio greater than 0.8 and they also considered the ability to maintain this ratio afterwards on a low flow. Essentially they were interested in a technique where the expired concentration – and by implication the brain concentration – is close to that set on the vaporiser dial. By contrast, adopting a low-flow technique which approaches that used in closed circuit anaesthesia, where the aim is to deposit and maintain a given *mass* of agent in the brain, the blood gas solubility of the agent has a minor role to play; there is no clinically significant difference between the handling of enflurane, halothane, isoflurane, sevoflurane or desflurane. Additionally there is no need for prolonged high flows with any of the agents; after partial denitrogenation of 30–60 s, low flow or closed circuit can be commenced immediately for procedures as short as 5 min. For the use of vaporisers outside the circuit, dial settings for the commonly used agents are published, or can be calculated [2].

Low-flow anaesthesia is a foggy domain; it has no underlying physiological principal: the choice of fresh gas flow is completely arbitrary. In contrast, the choice of flow for both high flow and closed circuit is determined by physiological and physical factors; in the former the requirement to eliminate CO_2 and in the latter to match patient gas uptake strictly. I would like to suggest that flows are not viewed as a mere numerical continuum from high flow to closed circuit, but rather as a suspension bridge, with two towers on solid foundations between which is slung the deck of low flow; it is unfortunate that we persist in trying to construct this bridge using only one tower. Closed circuit anaesthesia is a surprisingly simple technique to learn: it holds the promise of exploding many anaesthetic myths; with the current levels of gas and vapour monitoring there never has been a safer time to do so.

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2 Ernst E, Pearson JD. Principles and practice of closed circuit anaesthesia. *Advances in Anaesthesiology* 1987; **4:** 89–122.

New Year, New *Anaesthesia*

Congratulations on the new format which places the Contents on the front cover of the Journal. I look forward to enjoying the improved accessibility.

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