CORRESPONDENCE

The care of patients and transport by air

SIR,—I read with great interest the recent review on pre-hospital monitoring of trauma patients. This raised many important issues which need to be addressed further as expectations of out of hospital care increase. In Scotland there has been a fully integrated air ambulance service following a major reorganization in 1993. In addition to providing a primary patient transport service, there are also a large number of secondary inter-hospital transfers. Because of the distances involved and terrain, many of these involve air travel, using fixed-wing or rotary aircraft. In addition to adopting the principles of the Advanced Trauma Life Support program, minimum standards of monitoring, it has also been suggested that the recommendations for assistance should be followed in such situations.

Dr Morley highlighted in his review that monitoring can interfere with aircraft electrical systems. Most monitors give out a small amount of electrical activity to the surroundings; this is not true of defibrillators which, despite having been used successfully in-flight, still pose a greater hazard. It is recommended that authority is gained from the aircraft captain before defibrillators are discharged when airborne.

In addition to the function of monitors in the air environment, associated alarm systems have also come under scrutiny. The noise levels present in the back of an aircraft can render auditory alarms ineffective. It has also been demonstrated that relying on visual scanning for detection of problems leads to delays before alarm conditions are detected. Consideration is currently being given to incorporation of alarms into intercom systems to further increase safety.

All adverse events are more likely with prolonged journey times. With the move to centralize specialist facilities and notably paediatric intensive care units, patient transport requiring sophisticated monitoring is set to increase. Only by continued vigilance and debate will standards improve to keep pace with these developments.

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SIR,—Dr Shirley raises some interesting points which merit further comment. HEMS was certainly preceded by some air ambulance services in the UK, although it differs in several important respects. Most obvious of these is the presence of a full-time medical staff, trained specifically for the purpose of primary retrieval by helicopter of major trauma victims. It is difficult to exclude other factors when considering the specific added effect of a physician on an aeromedical prehospital team. One American study of blunt trauma patients found that patient mortality was 35 % less than predicted by trauma score and injury severity score (TRISS) methodology when the attending aeromedical team incorporated a doctor. In addition, mortality in this group was significantly lower than in a group of patients, with similar TRISS scores, attended by a flight nurse/flight paramedic team. The doctors were able to perform a wider range of practical procedures than their paramedic counterparts, including thoracostomy, cricothyroidotomy and pericardiocentesis.

On the issue of defibrillators, HEMS use the Lifepak LP10-23 (Physio-Control Corporation International, Redmond, WA, USA), which is specifically approved by the Civil Aviation Authority for the HEMS aircraft, an Aerospatiale Dauphin SA 365N. The defibrillator monitoring unit initially caused low level interference in the 120-MHz radio frequency band and this has been resolved by special modifications. Similar problems have been encountered on testing the Lifepak LP10-59 in the Bôlôk 105DSB used by Cornish First Air (personal communication, R. Dawny, Snafleet Aviation Design). Interestingly, in-flight defibrillation can only be heard as a faint click on the aircraft headsets.

Since HEMS began operations in 1989, only 18 patients have undergone defibrillation. In most instances, the defibrillator was used on scene, not in the aircraft. The three survivors had all crashed their cars after sustaining cardiac arrests while driving and their injuries were minor. All were discharged when airborne.

Effect of naloxone on nitrous oxide analgesia

SIR,—We would like to comment on the article by Yagi and colleagues. These authors were unable to show naloxone inhibition of the analgesic effects of nitrous oxide when measured against placebo. We have shown previously that with similar doses of naloxone in human volunteers, the peak effect of naloxone was measurable only within the first 2–3 min after bolus injection. This effect was not apparent after 5 min.

Although in a minority of subjects nitrous oxide analgesia was attenuated by naloxone the majority of subjects showed a transient increase in nitrous oxide analgesia. These observations were confirmed later by others in animals. From our findings we proposed the existence of two opioid systems, in dynamic equilibrium: one analgesic and the other pain producing.

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Sirs,—In a previous article1 we reported that the clinical dose of naloxone did not antagonize the analgesic action of nitrous oxide when the pain threshold test was performed 5 min after injection of naloxone in humans. As Gillman and Lichtigfeld pointed out, the dose (0.01 mg kg\(^{-1}\)) of naloxone used in the study may not have been adequate to fully antagonize the effects of the opioid.2 We chose this dose according to Jaffe and Martin3 who indicated that naloxone 0.4–0.8 mg i.v. in humans can prevent or reverse the effects of mu opioid agonists. The onset of action of naloxone is fast and seen within 1 or 2 min after injection.4 The duration of antagonistic effects is approximately 45 min after an i.v. dose of 0.4 mg\(^2\). In our previous study, measurement of pain threshold was commenced 5 min after administration of naloxone. Measurement of analgesia was obtained during the period of naloxone-induced antagonism. We also evaluated antagonism of nitrous oxide analgesia with higher doses (5 and 10 mg kg\(^{-1}\)) of naloxone by a radiant heat tail-flick test in rats (unpublished data). The results indicated that naloxone did not antagonize the analgesic action induced by nitrous oxide, even at high doses.

Gillman, Kock and Lichtigfeld\(^4\) showed that the peak effect of naloxone was measurable only within the first 2–3 min after bolus injection with clinical doses of naloxone in human volunteers, but not after 5 min. The majority of subjects showed a transient increase in nitrous oxide analgesia. This interesting peak effect of naloxone might be an agonistic effect seen at high doses of naloxone. Naloxone enters the brain very rapidly. The initial brain concentration of naloxone was reported to be much higher (4.6 mg\(^2\)) than that in serum\(^4\). Therefore, the brain concentration of naloxone was much higher (4.6 mg\(^2\)) than that in serum\(^4\). This interesting peak effect of naloxone may transiently reach a high level which may produce agonist effects during the short period after bolus injection of clinical doses. High doses of naloxone produce increased arterial pressure and arrhythmias which may be caused by the release of sudden intense sympathetic activity. Enhancement of sympathetic neurones by naloxone in the central nervous system may explain the transient increase in nitrous oxide analgesia 2–3 min after injection of naloxone.

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**Difficult tracheal intubation**

Sirs,—Difficult tracheal intubation is a major cause of anaesthetic-related morbidity and mortality, and any change which may lead to a reduction is to be welcomed. However, we were interested that when West and colleagues studied a tracheal tube designed specifically for this purpose, they used a size 8 tube for females and size 9 for males. While we know of no firm evidence that decreasing the size of the tracheal tube facilitates intubation in cases of difficulty, it does seem to be true in both our experience and that of others\(^5\). Furthermore, it has been known for many years that small tracheal tubes can be used safely and without complication during positive pressure ventilation\(^6\). It has become routine practice at this institution to use 6-mm tubes in females and 6.5-mm in males. We believe that West and colleagues would have found an increased success rate and shortened learning time had they used smaller tracheal tubes in their simulation of a difficult intubation.

The authors’ incidence of 33% for sore throat is similar to that quoted in other studies\(^7\), but 1–3% of patients may still have hoarseness up to 6 months after intubation\(^7\). This value may be expected to be even higher in cases of difficult intubation where greater degrees of laryngeal trauma are likely to occur. We believe that decreasing the size of the tracheal tube makes intubation easier and reduces the incidence of laryngeal damage. Stout and coworkers\(^2\) have shown that the incidence of hoarseness and sore throat may be reduced substantially by using tracheal tubes of size 7 or less. We believe that small tracheal tubes can be used routinely, and should be used when laryngoscopy is difficult.

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**Difficulty tracheal intubation**

Sir,—Our thanks to Drs Broomhead and Vaughan for their comments. The crucial step is to get the introducer into the right hole; when that is achieved, intubation with our tube is easy; that is its main advantage. It seems unlikely that smaller tubes would have altered the intubation time. With a Magill tube, when the introducer is in place, there is still the problem of preventing the bevel snagging on the cords and a smaller tube is often helpful, but with the new method a smaller tube is needed only if the larynx is abnormally small. The problem is simplified because instead of two possible causes of resistance, there is only one. Occasionally, some resistance is felt and a smaller tube is needed. Recent data\(^8\) suggest that over the past decade the incidence of failed intubation in obstetrics has increased. The authors imply that part of the explanation is that junior staff have less experience of obstetric general anaesthesia than in the past. If that is correct then it underlines our conclusions.

On the other hand, routine use of a small tube implies that there is never any need to change tube and that is an advantage. The authors also refer to Stout and colleagues\(^2\) who found that with size 7 tubes the incidence of sore throat was 22% compared with 33% in our study. But this could be fortuitous (P=0.1); a larger sample might show a real difference. Thus the case for smaller tubes is not proved but we agree that it merits further study.

May we add that the most serious cause of trauma is the novice who attempts to handle difficult intubations with no clear picture of the problem or its solution. This is perhaps the strongest argument in favour of our training drill.

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Cost of volatile anaesthetic agents

Sir,—I read with interest Dr Barker’s letter on the cost of volatile anaesthetic agents1. When the costs of various agents are listed, one is tempted to make comparisons, and economic concerns are of increasing importance in our practice. However, for fair comparison of the available agents, we need to consider the amount of anaesthetic required to provide a similar depth of anaesthesia and this extends beyond making comparisons of similar potencies at set fresh gas flow rates. Differences in solubility of various agents in the blood and tissues become important. The amount of anaesthetic agent required may be divided into that required to provide adequate anaesthesia at the start (to load up the circuit and the patient’s lungs), to that required to replace anaesthetic taken up into the blood and then into the tissues of the body, and also make account of that supplied in excess of uptake and therefore lost from the circuit and wasted. The relative cost of the available agent varies with the fresh gas flow chosen.

Estimates have been made of the volume of liquid anaesthetic required based on uptake kinetics and circuit performance at various fresh gas flow rates to sustain an alveolar concentration of 1 MAC for four of the volatile agents listed by Dr Barker2. I have included the price per millilitre of liquid anaesthetic, from the British National Formulary3, to calculate the cost of the volatile agent required (table 1).

There are limitations to the calculations: at low fresh gas flows, conventional vaporizers may be unable to supply sufficient agent, for example at 0.2 litre min⁻¹ at 1 MAC, isoflurane vaporizers cannot supply a sufficient amount of isoflurane for the first hour, whereas desflurane vaporizers are unable to do so for the first 10 min. The increased fresh gas flow required to compensate for the limitations of the vaporizers would increase the estimated cost noted in table 1, but this would be greatest for the more soluble volatile agents. In addition, there is some concern on the use of sevoflurane at low fresh gas flows4. Table 1 shows, as would be expected, that for any given volatile agent, cost increases with increased fresh gas flow, but also that the relative cost of the less soluble (and newer) volatile agents is reduced at lower fresh gas flows. This illustrates that when comparing the costs of volatile anaesthetic agents, consideration must also be given to uptake kinetics and fresh gas flows used.

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<table>
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<th>Fresh gas flow rate (litre min⁻¹)</th>
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</table>

ERRATUM

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In the methods section of this abstract, the dose of morphine was reported incorrectly: morphine 0.9 mg/kg should read morphine 0.09 mg/kg.

We apologize to the authors for this confusion.