concerned mainly with the rapid decrease in K\(^+\) concentration which takes place during extracorporeal perfusion when the pump is primed with K\(^+\)-rich blood. The recommendation in that paper, to limit administration of i.v. K\(^+\) to 70-100 mmol over 24 h was not based on any relevant experimental evidence, or referenced. The only other reference given by Vaughan which looks, by title, to be promising, is Vitez [4], which proved to be an abstract of a refresher course lecture and recommends not exceeding 10-20 mmol h\(^{-1}\) again, solely on the author’s own authority. (This reference is incorrect as printed: pages 530-531 should read S30-331). Dr Vaughan has kindly given me a reprint of the actual lecture which says "Potassium infusions can be tolerated at rates as high as 0.5 mEq kg\(^{-1}\) h\(^{-1}\); most guidelines list safe rates at 10-20 mEq h\(^{-1}\). This is referenced to a 1978 paper which is not readily available in the United Kingdom [5].

In real life, experienced intensivists sometimes exceed these recommendations, and this disjunction between reality and “standard” advice puts examination candidates into a difficult position when asked about the preoperative management of a patient with dehydration and potassium depletion. Such a patient may have large total body potassium losses, with a plasma concentration of potassium about 3.0 mol litre\(^{-1}\). Let us assume a situation in which the cardiac output is 3 litre min\(^{-1}\), with oliguria. If potassium is infused at twice the "recommended" rate (40 mmol h\(^{-1}\) diluted in a volume of 1000 ml) 0.67 mmol will be infused in 16.7 ml each 1 min. The total volume of fluid going through the heart every minute, therefore, will be 0.0167 litre, consisting of 3 litre at a concentration of 3.0 mmol litre\(^{-1}\) (i.e. 9 mmol of K\(^+\) and 0.0167 litre containing 0.67 mmol of K\(^+\). The final concentration would therefore be 9.67 mmol of K\(^+\) dissolved in 3.0167 litre, or 3.2 mmol litre\(^{-1}\), an increase of 0.2 mmol litre\(^{-1}\).

The build up, minute by minute, of the K\(^+\) concentration depends on its distribution volume. Even a severely dehydrated patient, if still conscious, is likely to have at least 20% of the body weight in rapidly exchangeable potassium, so that in a 60-kg patient, the total increase in concentration during 1 h could not be more than of the order of 3.3 mmol litre\(^{-1}\); but this assumes that there would be no transfer into cells. The load of K\(^+\) introduced by 72-h-old blood (14 mmol litre\(^{-1}\) in a heart-lung machine was shown to be cleared from the circulation within a few minutes of starting bypass, and this was not caused by renal excretion [3]. I suspect that 90% of any increase would be transferred into the cells which are, by definition, grossly depleted, and the plasma concentration might increase by 0.3 mmol litre\(^{-1}\) (from 3.0 to 3.3 mmol litre\(^{-1}\)), a prediction which, I venture to suggest, accords with experience and with what experimental evidence exists. For example, when potassium 0.5 mmol kg\(^{-1}\) was given to normal volunteers, the plasma concentration of potassium increased by 0.6 mmol litre\(^{-1}\) [8]. However, the current recommendations do seem extremely conservative.

Wherever one turns in the literature, one finds that experimental evidence is discounted in favour of opinions. For example, relying on Marindale sounds impressive, but even here [9] the sources are not treated evenhandedly. Reference is made to Clementsens [10], who gave two severely depleted patients respectively 335 mmol and 375 mmol of potassium i.v. over less than 6 h and reported no ill effects; more weight, however, is given to a subsequent letter from Bradfield [11], who offers no evidence whatever for his opinion that is should have been given more cautiously. Clementsens traces the then current recommendations (20 mmol h\(^{-1}\)) back to a 1957 book [12] and goes on to say: "It is my impression that too much attention is paid to these upper limits, so that intravenous potassium is often given with too much hesitation and in inadequate dosage...". After nearly 30 years, perhaps it is time to say ‘Amen’ to that.

M. D. Vickers
Cardiff

REFERENCES

Sir,—I thank you for allowing me to respond to Professor Vicker’s letter. There is no doubt that he has a point.

However, in a review one can only recommend “standard” or accepted safe practice. In this particular context, the advice regarding potassium therapy is that which is accepted around the world. By coincidence, the September 16, 1991 issue of Drug and Therapeutics Bulletin [1] has also addressed the issue of i.v. potassium replacement therapy. The advice is that K\(^+\) only 80 mmol/24 h is required for maintenance—that is, about 3-4 mmol h\(^{-1}\). It is also interesting that there is no reference cited in relation to this advice. Certainly, hypokalaemia does not cause undue problems and is rarely life-threatening, whereas hyperkalaemia does cause problems and is life-threatening. These differences most probably account for the conservative approach to i.v. potassium therapy.

The calculations made for the dehydrated patient have assumed that the infusion, dilution and redistribution rates of potassium all remain constant. The evidence, suitably referenced, does not support this assumption [2,3]. In addition, the experimental data cited in normal volunteers may not be applicable to the dehydrated patient. Professor Vickers, to be fair, quotes the papers in which cardiac arrest is reported to have been associated with i.v. potassium therapy. He also admits that there is an associated danger of excessive therapy.

Therefore, it seems to me that, until correct clinical trials regarding i.v. potassium therapy are completed, well founded and safe advice cannot be disregarded. To do so may lead to the “Amen” being pronounced over the patient and not over the safe, tried and trusted therapy, albeit partly opinion, partly evidence and considerable experience.

R. S. VAUGHAN
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REFERENCES
and in cancer chemotherapy [3]. Recently, we have reviewed the published evidence for an antiemetic action of stimulation of P6 [4], and it is becoming clear that all methods of stimulation are not equally effective.

An invasive approach (acupuncture with manual rotation or electrical stimulation of the needle) has a much more consistent effect than non-invasive methods (transcutaneous electrical stimulation or acupressure). This is evident particularly when there is a strong emetic stimulus such as with meptazinol [5] or nalbuphine premedication [6], or highly emetic chemotherapy [7]. Strabismus operations also fall into this class. There is one report showing a reduction in postoperative sickness when acupuncture was combined with neuroptanaesthesia in eye surgery [8]. In contrast, indirect stimulation such as transcutaneous electrical stimulation and acupressure, is shorter acting and less effective than acupuncture [3, 6]. Surprisingly, we have not been able to find any scientific evidence for the efficacy of acupressure in travel sickness [4].

In common with many others, we have included both emesis and nausea as an "emetic response" and this may not be appropriate with acupressure. Barsoum, Perry and Fraser [9] found a significant reduction of postoperative nausea (but not vomiting) after the use of acupressure, and a recent study of chemotherapy sickness has shown a reduction of nausea with acupressure [10]. While acupressure alone produces little benefit in reducing sickness from clavipain-like drugs [5], transcutaneous electrical stimulation has a synergistic action with ondansetron in treating residual nausea [11].

We are now accumulating evidence on the efficacy and possible mode of action of P6 antiemesis. This effect may be blocked by local anaesthesia [12], it has to be given before the emetic stimulus [13], and it now appears that different methods of stimulation produce differing effects [1, 4].

J. W. DUNDEE
C. M. McMillan
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REFERENCES


Sir,—Thank you for the opportunity to respond to the letter by Professor Dundee and Dr McMillan. Earlier investigations of P6 acupressure [1–3] are not directly comparable with our study [4]; there are differences in study design, population, emetic stimulus, measured responses and statistical analysis. All the previous studies investigated adults, and none defined an antiemetic effect that was considered clinically significant (our study was designed to detect a 50% difference in the incidence of vomiting between two groups), or discussed the calculation of sample size. Nonetheless, our findings were different from these earlier studies: we were unable to demonstrate an antiemetic effect using P6 acupressure.

We would agree that the available evidence suggests that invasive acupuncture is more effective and has a greater duration of action compared with non-invasive methods. However, for P6 stimulation to be effective it must be applied before the emetic stimulus [5]. Clearly, acupuncture would not be tolerated by the majority of awake children. Although we chose to use acupressure before anaesthesia and surgery, an alternative may have been to administer acupuncture immediately after induction of anaesthesia.

Finally, Professor Dundee suggests that acupuncture may be effective in reducing the incidence of nausea, but less effective in reducing the incidence of vomiting. We recorded the incidence of postoperative nausea and vomiting during recovery in hospital; only vomiting was recorded at home by the parents. In fact, only one child in the study felt nauseated without vomiting in hospital and that child later vomited at home; the results for nausea or vomiting were therefore almost identical.

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REFERENCES


NO HEPARINIZATION WITH THE CELL SAVER

Sir,—Dr Columb's letter [1] may mislead those unfamiliar with cell salvage as an autotransfusion technique. The "Cell Saver" is a machine made by Haemonetics which we have used extensively over 8 years [2]. Blood coagulation profiles are monitored routinely and no evidence of heparin overspill has been seen.

The problems Dr Columb describes are with the "Kardiothor" system. They emphasize the importance of machine design and maintenance. Bedside (in-theatre) monitoring of activated partial thromboplastin time and prothrombin time is now available (512 Coagulation Monitor, Ciba Corning), and where cell salvage machines are used with large amounts of heparin, with risk of