with error bars of 2 SD, the results may be seen in perspective (fig. 1, lower panel).

The investigators described repeat testing for differences between the groups by Student's unpaired t test or by Mann–Whitney U test, and stated that they then confirmed the differences by analysis of variance or by Kruskal–Wallis. However, it is invalid to test repeatedly by Student's or by Mann–Whitney unpaired differences between groups has first been shown by analysis of variance or by Kruskal–Wallis.

The investigators' final assertion is unproven. They imply that a patient who has already had a pressor response to cannulation may have an exaggerated response to a further stimulus, and that this is the reason that high risk patients should receive local anaesthesia before cannulation. The investigators did not describe subsequent laryngoscopy, so do not know that a response would be exaggerated. Patients truly at high risk would almost certainly receive some prophylactic treatment before laryngoscopy, and “will never be included in the control group of an outcome study examining the efficacy of haemodynamic control at...intubation!” [2].

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Sir,—In the paper by Langham and Harrison on the pressor response to venous cannulation [1], the authors concluded that there was a significant response to venous cannulation, as judged by changes in mean arterial pressure (MAP) and rate—pressure product (RPP), which was obviated by prior infiltration with local anaesthetic; they recommended that lignocaine should be considered before cannulation. There are several aspects of this paper which we would like to discuss further.

First, we were not told in which arm cannulation took place. Presumably, it was into the arm opposite to that used for arterial pressure readings. Injection of local anaesthetic into the same arm might, theoretically, affect the neighbouring microvasculature and interfere with arterial pressure readings by digital plethysmography. Second, we were not told if the cannulation site was standardized. It is possible that bias could have occurred if Investigator 1 was free to choose a location for cannulation. The methodology described does not prevent Investigator 1 using a different cannulation site being the dorsum of the non-dominant hand. The Finapres monitor was sited on the middle finger of the contralateral hand; this may have an exaggerated response to a further stimulus, and that this is the reason that high risk patients should receive local anaesthesia before cannulation. Consent was obtained from the patients on the night before surgery, at which time they were told that they may receive one or two “scratches” in the back of their hand. As clearly stated in the paper, time zero was taken as the time of the first stimulus to the patient, be it local infiltration or cannula insertion. The lack of a pressor response to the insertion of the 25-gauge needle is apparent from the results presented.

We agree with Dr Goodman that the most important reason to use local anaesthetic infiltration before venous cannulation is humanitarian and our previous studies [1,2] had already addressed this issue. The fact that a measurable pressor response occurred in normal patients may imply that a clinically important response occurs, for example in those with pre-existing hypertension, and this is currently being studied.

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THE MANAGEMENT OF ACUTE POISONING

Sir,—We read with interest the review article by Collee and Hanson [1], but wish to raise a number of points. We would question the statements that “in unconscious patients organic brain damage should always be suspected if the history of poisoning is unsatisfactory and the depth of the coma does not improve within 12 h” and that “any patient whose level of consciousness appears inappropriately depressed for the history of the poisoning should be suspected of having an intracranial haemorrhage.” In our experience, a satisfactory history of the poisoning in an unconscious patient is rare. In this situation it is essential to exclude a correctable metabolic or intracranial cause for the coma. Even in the presence of a good history, a high degree of suspicion should be maintained, intoxicated patients are often the victims of trauma. It is mandatory to exclude intracranial pathology before ascribing a depressed state of consciousness to the effects of drugs or alcohol. With appropriate neurological examination and the ready availability of CT scanning, we have the means to avoid repeating the lessons of the past [2].

From our experiences with intoxicated patients in North American hospitals, we recognize that the agitated, aggressive, stuporous or comatose intoxicated patient poses many problems in assessment and treatment. Under these circumstances an aggressive approach to patient management may be appropriate. Rapid sequence induction, tracheal intubation and control of