ventilation prevent the patient injuring themselves or others, reduces the chance of secondary brain injury, facilitates investigation (CT scanning), allows monitoring of vital signs and provides optimal conditions for any intervention.

Second, we were surprised that the review did not mention the possible use of dantrolene in the management of hyperthermia associated with acute intoxication. The role of this drug in the treatment of hyperthermia secondary to intoxication has been underestimated in the past, but many recent reports of its use in this potentially fatal complication suggest that it may lead to improved outcome [3, 4].

Third, the list of cardiovascular complications of acute poisoning does not include myocardial ischaemia or infarction; these are serious omissions. Reports of more than 50 cases of acute myocardial infarction complicating poisoning have been published. Seventy percent of the victims had no significant cardiac history, the majority being young males who were regular users of cocaine [5]. Recognition of this potential complication has direct consequences on the choice of agents used to manage the cardiovascular complications of cocaine intoxication. Cocaine produces postsynaptic accumulation of catecholamines, resulting in increased sympathetic nervous system effects. Lignocaine is an illogical choice as an anti-arrhythmic in this situation: in common with cocaine, it is also a local anaesthetic and decreases the seizure threshold, and may precipitate convulsions. Propranolol is also an illogical choice; beta block alone may result in unopposed alpha adrenoceptor activity, thus worsening coronary vasoconstriction and hypertension [6]. There are two logical choices available for controlling the cardiovascular effects of cocaine toxicity: labetolol, which the authors did mention, and esmolol, a beta-, adrenoceptor (cardioselective) blocker; both have been reported as being of use in cocaine toxicity [7, 8].

Finally, we were confused by the advice given for the treatment of salicylate poisoning. The fluid regimen called for 1 litre of saline 1 mol litre\(^{-1}\) or 0.3 mol litre\(^{-1}\) every 4-6 h, but that one should not give more than 300 mmol of sodium per 24 h (sic). The initial instruction would result in the administration of 1000 mmol of sodium. One litre of 0.3 mol litre\(^{-1}\) every 4-6 h would provide 1800-1200 mmol of sodium in 24 h, both prescriptions providing considerably more than the stipulated maximum dosage.

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Sir,—Thank you for the opportunity to reply to this letter. The critique of our article by Drs Parr and Craft is helpful, and we agree with several of the issues they raise. Certainly the exclusion of intracranial pathology in cases of acute coma is mandatory, but the circumstances in which an "aggressive approach" to patient management is justified are difficult to define precisely. Clearly, a full clinical assessment by appropriate specialists is preferable to a blanket policy of CT scanning.

Regarding the role of dantrolene in the management of hyperthermia following acute intoxication, we feel insufficient evidence exists at present to recommend its use. In addition to the two case reports cited in the letter from Drs Parr and Craft [1, 2], we discovered only two others before submitting our manuscript [3, 4]. Hyperthermia occurring after 3,4-methylenedioxymethylamphetamine—"Ecstasy"—may be related to its effect on central serotonergic and dopaminergic pathways, or to the consequences of its abuse. The mechanism is not yet known. The prolonged exercise and dehydration resulting from all-night dancing suggest that, in many cases, hyperthermia after Ecstasy may resemble heatstroke in its pathogenesis. Such cases would not be expected to respond to dantrolene [5], nor do the central mechanisms provide an easy explanation of why dantrolene should be expected to be an effective treatment if neuromuscular blockers fail. In one report of its use [3], an initial dose of 1 mg kg\(^{-1}\) failed to reduce the patient's temperature, which remained increased until after a third dose. Dr Parr's own case report of the use of dantrolene in the treatment of a fatal theophylline overdosageremains (as far as we know) the only account of its use in hyperthermia of that aetiology. It seems prudent to await further results. Meanwhile, we feel it more appropriate to emphasize the importance of general supportive measures.

We thank Drs Parr and Craft for pointing out the omission of myocardial ischaemia in the general section on the cardiovascular complications of acute poisoning. Regarding the management of arrhythmias secondary to cocaine poisoning, the role of beta antagonists is undisputed.

The fluid regimen in salicylate poisoning was converted to S.I. units in accordance with editorial guidelines, and it is indeed confusing. An erratum appeared in the August issue of British Journal of Anaesthesia (p. 331).