CORRESPONDENCE

ERYTHROCYTE STABILITY IN ETHANOL–SALINE SOLUTIONS

Sir,—In a clinical evaluation of ethanol as an anaesthetic agent, Isaac and Dundee (1969) and Dundee and co-workers (1970) established that 8% w/v (10% v/v) ethanol in Hartmann’s solution was the most satisfactory concentration for i.v. infusion in man and was associated with a low frequency of thrombophlebitis. Sanderson and co-workers (1970) confirmed that in vitro ethanol solutions in concentrations of 2.0–2.5 mol litre⁻¹ (approximately equivalent to 10% v/v) in Hartmann’s solution are least likely to cause erythrocyte lysis. Studies in our laboratory have shown that in vitro haemolysis of human erythrocytes is prevented (less than 5%) in 0.9% sodium chloride solution containing 0.0–10.0% v/v ethanol (Ku and Cadwallader, 1974). Also, our determination of “haemolytic” isotonic coefficients indicated that ethanol at the 10% v/v concentration provided a stabilizing effect on erythrocytes. Higher concentrations of ethanol, however, caused haemolysis; haemolysis was initiated in 11% v/v ethanol and was complete when the concentration reached 14% v/v. To examine this more closely, we obtained from the forearm veins of several 22–46-yr-old Caucasian and Oriental subjects 10 different blood samples, which were placed in 0.9% (0.154 mol litre⁻¹) sodium chloride solutions containing various concentrations of ethanol. After 45 min at 37 °C, the maximum ethanol concentration which was not associated with haemolysis was 10.1% v/v ± SD 0.52. These data confirm that 10% v/v is an optimum infusion concentration for ethanol in an isotonic vehicle and that higher concentrations cause haemolysis.

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REFERENCES


ANTAGONISM OF KETAMINE BY PHYSOSTIGMINE

Sir,—The preliminary report of Balmer and Wyte (1977) describing a ketamine/physostigmine combination to provide awake analgesia without psychological upset was noted with interest. However, this combination may produce convulsions in some patients. Ketamine alone produced generalized convulsions in 0.28% of 4265 patients, and tonic and clonic movements, without e.e.g. seizure activity, have also been reported on induction of anaesthesia with ketamine (Parke–Davis monograph).

Physostigmine salicylate is an anticholinesterase which penetrates the blood–brain barrier and possesses analeptic properties (Goodman and Gilman, 1975). It is used in recovery rooms in North America (Brebner and Hadley, 1976) and for rousing patients who have poisoned themselves with tricyclic antidepressants (Newton, 1975). Although imipramine, in large doses, may provoke grand mal seizures, the addition of physostigmine occasionally precipitates them. Convulsions occurred twice in Newton’s study of 21 patients and other instances have occurred in a pilot study (personal communication).

It appears, therefore, that the combination of ketamine with physostigmine could produce convulsions and caution is advisable.

ALBAN HOUGHTON

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REFERENCES


Sir,—Thank you for the opportunity to reply to Dr Houghton’s letter. His comments are entirely justified, but in our defence may I plead that the rate of dosage of ketamine which we employed is very low indeed. In addition, the very small doses of diazepam administered i.v. may have afforded the patient a degree of protection from seizure activity.

Barnard’s group have experience of approximately 5000 administrations of physostigmine in the past 10 years with no serious sequelae (DiLiberti and O’Brien, 1975) and although ketamine has been associated with e.e.g. evidence of seizure activity or even frank convulsions, there is a possibility that it may act occasionally as an anti-convulsant (Rucci and Caroli, 1974).

In presenting the paper, I emphasized that we were not recommending the use of physostigmine for awakening patients who had received normal anaesthetic doses of ketamine, and I regret that this stricture was not included in the abstract.

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REFERENCES
