myocardial infarction and distress in pulmonary oedema. The production of a ‘medical phlebotomy’ as part of their therapeutic effect has not been proven. It is just as likely that any haemodynamic change and, in particular, severe hypotension which may occur will have an adverse effect.

I would urge caution in relation to dosage of intravenous opiates in the early stages of treatment.

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REFERENCES


A severe case of hyponatraemia

Sir

Recently, while working in the Accident and Emergency Department, a 49-year-old man presented in a very agitated state making incomprehensible sounds. His wife knew of no recent fall or trauma of any kind. Examination of the patient proved difficult, but venous access and blood tests were eventually obtained. A history of prolonged alcohol intake and protracted vomiting was eventually obtained from his wife. A serum sodium was 100 meq/l and serum urea was 2·2 mmol/l (Anderson et al., 1985).

He was given hypertonic saline intravenously (Ayres et al., 1982). A chest X-ray showed no gross abnormality. Soon after returning to the Department, having had his X-ray, and while still in an agitated state he aspirated and suffered a respiratory arrest followed by cardiac arrest. He was resuscitated and subsequently transferred to the Intensive Care Unit. Unfortunately, later that evening, he suffered several further episodes of cardiac arrest and died. The serum sodium remained at 113 meq/l in spite of intensive therapy (Levensky, 1983). A post-mortem examination revealed cirrhosis of the liver, oesophageal varices, widespread peptic ulceration, cerebral atrophy and cerebral oedema.
Death in patients with hyponatraemia is often related to the underlying medical disorders (Arieff et al., 1976; Kleinfeld et al., 1979; Flear et al., 1981). Delirium and seizures are common presenting features. Hyponatraemia causes neurological symptoms when serum sodium levels are below 120 meq/l and even more frequently when below 110 meq/l. Immediate treatment is indicated when patients present with confusion, seizures, shock and in a comatose state (Flear et al., 1981; Thomas et al., 1978; Ayres et al., 1985). Many of the symptoms which are seen are thought to arise from cerebral oedema (Tomlinson et al., 1979; Arieff, 1985).

We present this case to highlight the importance of measuring serum sodium in all confused patients but particularly in alcoholics, in whom hyponatraemia is associated with a mortality of 86% (Thomas et al., 1978; Baron & Hutchinson, 1983).

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REFERENCES


Hand injury review clinics

Sir

I read with great interest the article by Cutting & McLean (Archives of Emergency Medicine 4, 211–17). I would like to congratulate them on the successful running of their clinic.

The Hand Injury Review Clinic at East Birmingham Hospital runs on very similar
A severe case of hyponatraemia.

P Wilson

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