Delivery of fluids by the subcutaneous route

Editor,—We would like to contribute to the excellent review by Mbamalu and Banerjee on the principles and problems of peripheral venous cannulation. Although peripheral venous cannulation to allow fluid and electrolyte replacement is mandatory in emergencies and in situations where strict fluid balance is required, it is worth noting that it may not always be straightforward. Hypodermocysis, the technique of correcting fluid deficits by subcutaneous infusion popularised initially in the 1950s, serves as a useful parenteral alternative to intravenous cannulation. Isotonic fluids may be introduced into subcutaneous tissues in situations where the aim is to correct mild to moderate dehydration in elderly patients (especially in a chronic care setting where intravenous access in the infirm and elderly is notoriously difficult), in addition to being a less invasive route of drug administration in palliative management where opioid and antieptic treatment is frequently warranted. Fluid replacement by the subcutaneous route is relatively safe, easier to initiate, demands less nursing time, is more cost-effective than intravenous therapy, causes less toxicity in the infused solution and avoids a large number of punctures, and is more cost effective. Intravenous cannulation is relatively safe, easier to initiate, costs more, and causes more toxicity than subcutaneous therapy.7

In my experience, early management with hypodermoclysis, high osmolarity and high flow rates, allows for maximal hydration of patients with cognitive impairment.8

Intravenous cannulation is the preferred route of administration of fluids and other medications; however, the subcutaneous route may be useful in situations where intravenous access is not feasible or when peripheral venous cannulation may cause discomfort to the patient. Anticipated fluid requirements in these situations may be calculated by multiplying the patient’s body weight in kilograms by 0.4 to 0.6, depending on the degree of hydration.7

High dose intravenous glucagon in severe tricyclic poisoning

Editor,—I read with interest the case report by Sensky and Olczak describing the use of glucagon in severe tricyclic antidepressant (TCA) poisoning.1 I am surprised that the patient was not treated with sodium bicarbonate and that there was no mention of the pivotal role of alkalinisation and sodium loading in the management of TCA poisoning.

The cardiotoxicity of TCAs is a consequence of the excitatory effects on the sodium channels in the heart.2 These effects can be alleviated by the administration of sodium bicarbonate, which reduces the frequency of ventricular arrhythmias, decreases the prolongation of the QRS interval, and reduces hypotension.3 These effects have been attributed to the increase in plasma sodium concentration, which competes to reverse the inhibition of sodium conductance by the TCA. Alkalinisation changes the ionisation state of TCA which may facilitate uncoupling of the TCA from the sodium channel.4 Alkalosis induced by hyperventilation can also be effective.

Their patient was acidic with a pH of 7.29 and with evidence of severe cardiac toxicity. Initial resuscitation of this patient should have included aggressive treatment of acidosis, which potentiates any cardiac toxicity, by heparin or nothing?5 Twenty four hour Holter monitoring revealed sinusal bradycardia with more than 900 pauses of >2 sec, but subsequent investigations failed to establish any evidence of coronary artery disease. The patient later had a dual chamber pacemaker inserted for this.

Neuromyotonia and sinoatrial block

Editor,—A recent review article in your journal gives the impression that the case for heparinisation of all patients with cerebral venous sinus thrombosis (CVST) is proved. However, the largest and most robust study did not come to this conclusion and its results did not achieve statistical significance.6 Given that CVST has a spectrum of presentations from headache to coma and death, a blanket approach to treatment in the absence of good evidence and continued debate does not seem sensible.

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intracranial hypertension and hence a much better prognosis anyway; finally a different treatment modality using subcutaneous low molecular weight heparin instead of the intravenous route.

I would agree with Bousser that heparin confers a clinically relevant benefit, justifying the use of this treatment as long as we remain unable to predict the outcome in the individual patient with CVST.


**Hepatocellular carcinoma**

EDITOR,—We read with interest the excellent review article on hepatocellular carcinoma. However Badvie omits to mention Budd-Chiari syndrome and diabetes among the conditions associated with this disease. The evidence of hepatocellular carcinoma in patients with Budd-Chiari syndrome is high. In a study of Japanese patients with chronic Budd-Chiari syndrome in Japan, in a study designed to evaluate the effects of hepatic enzyme inducing drugs, a population based cohort of 153 852 diabetic patients also showed a fourfold risk of liver cancer in these patients, which was greater in males compared with females. The clinical and epidemiological and clinical studies of Budd-Chiari syndrome in Japan. In: Scheur P, Chapman R, editors. International Association for the Study of the Liver, Biennial Scientific Meeting, 3–6 June 1992. Brighon: ISLÉ, 1992: OP-80 (abstr).


**DIARY**

**Falk Symposia**

1/2 October 2000: Non-neoplastic diseases of the anorectum—an interdisciplinary approach (Freiburg, Germany)

3/4 October 2000: Immunosuppression in inflammatory bowel diseases—standards, news, and future trends (Freiburg, Germany)

12/13 October 2000: Biology of bile acids in health and disease (Den Haag, The Netherlands)

4 November 2000: Chronic bowel diseases—progress and controversies at the turn of the century (Bucharest, Romania)

**Columbia University College of Physicians and Surgeons, New York**

28–31 July 2000: 10th Annual course. A comprehensive review of movement disorders for the clinical practitioner

30 July–5 August 2000: 5th Annual course. A comprehensive review of movement disorders for the clinical practitioner

**Ninth International Symposium on celiac disease**

10–13 August 2000: Hunt Valley, MD, USA

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