LETTERS TO THE EDITOR

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.¹ 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 necessary. Hypodermoclysis, 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 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 antiemetic treatment is frequently warranted.² 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 discomfort, minimises the risk of intravascular infection,³ does not immobilise a limb (since it may be given into anterior abdominal tissues), and has been found to be less distressing for patients.⁴ The use of hyaluronidase in the infused solution augments the rate of fluid uptake and volumes up to 3000 ml can be delivered over 24 hours.⁵ Evidence suggests that potassium chloride may also be added to the subcutaneous infusion and concentrations up to 34 mmol/l have been given, with the only undesirable effect being that of discomfort at the delivery site.⁶ The chief technical disadvantages of subcutaneous fluid therapy are local oedema and sepsis at the infusion site, but the reported incidence of the latter is exceedingly low.³ Since the welfare of elderly patients in long term care increasingly represents the core of medical workload in Western populations it follows that hypodermoclysis can be an important parenteral route of fluid administration, and may therefore be incorporated as an additional limb in the instructive algorithm proposed by Mbamalu and Banerjee.

JAGDEEP SINGH GANDHI
George Eliot Hospital NHS Trust, Warwickshire

VINOD PATEL
University of Warwick and Diabetes and Asthma Centre,
George Eliot Hospital NHS Trust,
College Street, Nuneaton,
Warwickshire CV10 7DJ, UK

- 1 Mbamalu D, Banerjee A. Methods of obtaining peripheral venous access in difficult situations. *Postgrad Med J* 1999;75:459-62.
- 2 Bruera E, Legris MA, Kuehn N, et al. Hypodermoclysis for the administration of fluids and narcotic analgesics in patients with advanced cancer. *J Pain Symptom Manage* 1990;5:218-20.
- 3 Jain S, Mansfield B, Wilcox MH. Subcutaneous fluid administration—better than the intravenous approach? *J Hosp Infect* 1999;41:269-72.

- 4 O'Keeffe ST, Lavan JN. Subcutaneous fluids in elderly hospital patients with cognitive impairment. *Gerontology* 1996;42:36-9.
- 5 Berger EY. Nutrition by hypodermoclysis. *J Am Geriatr Soc* 1984;32:199-203.
- 6 Rochon PA, Gill SS, Litner J, et al. A systematic review of the evidence for hypodermoclysis to treat dehydration in older people. *J Gerontol A Biol Sci Med Sci* 1997;52:M169-76.

The authors respond:

We appreciate the comments by Drs Gandhi and Patel. The inclusion of hypodermoclysis in the algorithm would certainly be worthwhile and the authors have made a good case for consideration of the procedure.

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 alkalisation and sodium loading in the management of TCA poisoning.

The cardiac toxicity of TCAs is a consequence of the quinidine-like 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. These effects have been attributed to the increase in plasma sodium concentration, which competes to reverse the inhibition of the sodium channel by the TCA. Alkalisation changes the ionisation state of TCA which may facilitate uncoupling of the TCA from the sodium channel.³ Alkalosis induced by hyperventilation can also be effective.

Their patient was acidotic with a pH of 7.29 and with evidence of severe cardiac toxicity. Initial resuscitation of this patient should have included aggressive treatment of the acidosis with sodium bicarbonate. Sodium bicarbonate 1-2 mmol/kg (1-2 ml/kg of 8.4% solution) intravenously is recommended, followed by further doses to maintain an arterial pH between 7.45 and 7.55.⁴ Early intubation and mechanical ventilation is also advised in view of (i) the potential for rapid deterioration in conscious level and risk of gastric aspiration and (ii) the development of a confounding respiratory acidosis, which potentiates any cardiac toxicity.

In my experience, early management with sodium bicarbonate minimises any need for antiarrhythmic or inotropic support.

M J L SCHUSTER-BRUCE
United Bristol Healthcare NHS Trust,
Directorate of Anaesthesia, Level 7,
Bristol Royal Infirmary,
Bristol BS2 8HW, UK

- 1 Sensky PR, Olczak SA. High dose intravenous glucagon in severe tricyclic poisoning. *Postgrad Med J* 1999;75:611-2.
- 2 Frommer DA, Fulig KW, Marx JA, et al. Tricyclic antidepressant overdose: a review. *JAMA* 1987;257:521-6.
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Neuromyotonia and sinoatrial block

EDITOR,—A 70 year old Chinese woman was admitted with symptoms of paraesthesia on her limbs, hypertension, dizziness, extreme bradycardia, and a positive Trousseau's sign. She was on atenolol for hypertension, but she did not recover from the bradycardia even after stopping her antihypertensive. She subsequently developed proximal myopathy and Chvostek's sign.

Results of routine blood tests, including corrected calcium, phosphate, and magnesium concentrations, were within normal limits. Other investigations ruled out the possibility of porphyrias and antibody related diseases.

Electromyography revealed features of "acquired neuromyotonia", but on specific testing the antibody to voltage gated potassium channel was not detected. No other cause could be established for this rare neurological syndrome.

Twenty four hour Holter monitoring revealed sinoatrial block 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 is a membrane disorder and antibody to voltage gated potassium channel has been shown to be one its features. There is no literature describing the association between it and any kind of heart block.

I would be interested to know if there is any relationship between sinoatrial block and neuromyotonia.

PRADIP DE
Department of Medicine,
Princess Alexandra Hospital,
Hamstel Road, Harlow,
Essex CM20 1QX, UK

Cerebral venous sinus thrombosis: heparin or nothing?

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.¹ The largest and most robust study did not come to this conclusion and its results did not achieve statistical significance.² 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 debate³ does not seem sensible.

M B LEWIS
Department of Neurology,
Leeds General Infirmary,
Great George Street, Leeds LS1 3EX, UK

- 1 Allroggen H, Abbott RJ. Cerebral venous sinus thrombosis. *Postgrad Med J* 2000;76:12-15.
- 2 de Bruijn SF, Stam J, for the CVST Study Group. Randomised, placebo-controlled trial of anticoagulant treatment with low-molecular-weight heparin for cerebral sinus thrombosis. *Stroke* 1999;30:484-8.
- 3 Lewis MB. Cerebral venous thrombosis: nothing, heparin, or local thrombolysis? *Stroke* 1999;30:1729.

The author responds:

I acknowledge Dr Lewis' comment with respect to the Dutch CVST Study Group trial.¹ Nevertheless I feel this study in combination with an earlier study by Einhäupl *et al*² allows the conclusion that intravenous heparin should be first line treatment of CVST. The Dutch trial differed in several points from the German study, which may explain its neutral outcome. They are the altogether smaller number of poor outcomes; the larger proportion of patients with isolated

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,³ justifying the use of this treatment as long as we remain unable to predict the outcome in the individual patient with CVST.

- 1 de Bruijn SF, Stam J, for the CVST Study Group. Randomised, placebo-controlled trial of anticoagulant treatment with low-molecular-weight heparin for cerebral sinus thrombosis. *Stroke* 1999;30:484-8.
- 2 Einhäupl KM, Villringer A, Meister W, et al. Heparin treatment in sinus venous thrombosis. *Lancet* 1991;338:597-600.
- 3 Bousser MG. Cerebral venous thrombosis: nothing, heparin, or local thrombolysis? *Stroke* 1999;30:481-3.

Hepatocellular carcinoma

EDITOR.—We read with interest the excellent review article on hepatocellular carcinoma.¹ 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 by Okuda and colleagues,² and commented on by Kashyap and Kashyap,³ an incidence of hepatocellular carcinoma of 6% was found. Lawson *et al* observed a fourfold increased risk of hepatocellular carcinoma in diabetic patients in a study designed to evaluate the effects of hepatic enzyme inducing drugs.⁴ 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.⁵ La Vecchia *et al* in a case-control study reported an odds ratio risk associated with a history of diabetes of 2.3 and felt that in their study population history of diabetes could account for 8% of cases of liver cancer.⁶ It is difficult to speculate whether the fatty liver of diabetes is the hepatocellular injury that predisposes to hepatocellular carcinoma or whether it is the presence of diabetes that leads to increased susceptibility of the damaged liver to progression to hepatocellular carcinoma.

A S KASHYAP
Department of Medicine,
Armed Forces Medical College,
Pune 411040, India

SUREKHA KASHYAP
Department of Hospital Administration,
Armed Forces Medical College,
Pune 411040, India

- 1 Badvie S. Hepatocellular carcinoma. *Postgrad Med J* 2000;76:4-11.
- 2 Okuda H, Yamagata H, Obata H, et al. Epidemiological and clinical studies of Budd-Chiari syndrome in Japan. In: Scheur P, Chapman R, eds. International Association for the Study of the Liver, Biennial Scientific Meeting, 3-6 June 1992. Brighton: IASL, 1992: OP-80 (abstr).

- 3 Kashyap AS, Kashyap S. Hepatocellular carcinoma (letter). *Lancet* 1999;354:253.
- 4 Lawson DH, Gray JMB, McKillop C, et al. Diabetes mellitus and primary hepatocellular carcinoma. *Q J Med* 1986;61:945-55.
- 5 Adami HO, Chow WH, Nyren O, et al. Excess risk of primary liver cancer in patients with diabetes mellitus. *J Natl Cancer Inst* 1996;88:1472-7.
- 6 La Vecchia C, Negri E, Decartie A, et al. Diabetes mellitus and the risk of primary liver cancer. *Int J Cancer* 1997;73:204-7.

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 inflammatory bowel diseases—progress and controversies at the turn of the century (Bucharest, Romania)
Details: Falk Foundation eV—Congress Division, Leinenweberstr 5, PO Box 6529, D-79041 Freiburg, Germany (tel: +49 (0) 761 130340; fax: +49 (0) 761 1303459; e-mail: symposia@falkfoundation.de).

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. Update and intensive review in internal medicine
Details: Center for Continuing Education, Columbia University College of Physicians and Surgeons, 630 West 168th Street, Unit 39, New York, NY10032, USA (tel: +1 212 781 5990; fax: +1 212 781 6047; e-mail: cme@columbia.edu).

Ninth International Symposium on celiac disease

10-13 August 2000: Hunt Valley, MD, USA
Details: Althea Pusateri, Program Coordinator, University of Maryland School of Medicine, 655 W Baltimore Street, Baltimore, MD 21201, USA (tel: +1 410 706 3957; fax: +1 410 706 3103; http://www.celiaccenter.org).

Royal College of Physicians of Edinburgh

2-15 September 2000: Healthcare for older people—the UK experience (course)
7-8 October 2000: Stroke treatment and service delivery (consensus conference)
Details: Education, Audit, and Research Department, Royal College of Physicians of Edinburgh, 9 Queen Street, Edinburgh EH2 1JQ, UK (tel: +44 (0) 131 225 7324; fax: +44 (0) 131 220 4393; web site: www.rcpe.ac.uk).

Royal College of Physicians of Edinburgh/Scottish Intercollegiate Guidelines Network

3 November 2000: Symposium on clinical effectiveness, clinical guidelines and clinical standards
Details: Mrs Anne Fairbairn, Coordinator for Research and EBM, Royal College of Physicians of Edinburgh, 9 Queen Street, Edinburgh EH2 1JQ, UK (e-mail: a.fairbairn@rcpe.ac.uk).

Imperial College School of Medicine, Division of Paediatrics, Obstetrics and Gynaecology

27 June 2000: Caring for sexuality in health and illness (for healthcare professionals and nurses)/jointly with Association of Psychosexual Nursing
29 June-1 July 2000: New horizons in recurrent pregnancy loss
5 July 2000: Bereavement
6-7 July 2000: Advances in obstetric medicine: international meeting of obstetric medicine societies (satellite to ISSHP, Paris)
Details: Symposium Office, Imperial School of Medicine, Queen Charlotte's and Chelsea Hospital, Goldhawk Road, London W6 0XG, UK (tel: +44 (0)20 8383 3904; fax: +44 (0)20 8383 8555; e-mail: sympreg@ic.ac.uk).

St Mark's Hospital & Academic Institute

16-18 October 2000: Frontiers in colorectal disease (lecture course)
Details: The Administrator, St Mark's Academic Institute, St Mark's Hospital, Northwick Park, Harrow, Middlesex HA1 3UJ (tel: +44 (0) 20 8235 4046/8; fax: +44 (0) 20 8235 4039; e-mail: e.power@ic.ac.uk; web site: www.stmarkshospital.org.uk).