Letters to the Editor

An oral treatment for lead toxicity

Sir,

We read with interest the paper by Thomas and Ashton¹ which described a patient with lead poisoning in whom comparison was made between the efficacy of intravenous sodium calciumedetate and oral DMSA. The authors claim that 'an equal amount of the lead is excreted by the two drugs'. Although both chelating agents produced similar falls in the blood lead concentration, the limited data given in Figure 1 by the authors¹ suggest that the excretion of lead during treatment with sodium calciumedetate was approximately five times greater than with oral DMSA, notwithstanding the fact that both treatments were continued for a similar period.

The efficacy of chelation therapy in lead poisoning cannot be judged by estimating blood lead concentrations alone; determination of urine lead excretion is mandatory. A critical review of the two comparative clinical studies published in the English literature^{2,3} does not support the view that DMSA is superior to sodium calciumedetate in promoting urinary lead excretion. Even though only 12–16 mg/kg/day sodium calciumedetate was given to intoxicated smelter workers,² lead excretion was significantly greater than after DMSA administration (18–42 mg/kg/day). In a paediatric study,³ sodium calciumedetate (29 mg/kg/day) also produced greater urinary lead excretion than DMSA (30 mg/kg/day). Our own data (to be published) demonstrate that intravenous sodium calciumedetate (75 mg/kg/day) is approximately four times more effective in promoting lead excretion than DMSA (30 mg/kg/day). Taking into consideration not only 'therapeutic' dose but molar equivalents, we conclude that oral DMSA is less effective than intravenous sodium calciumedetate in promoting urinary lead excretion.

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Profound prolonged hypotension following captopril overdose

Sir,

We would like to report a case of captopril overdose complicated by prolonged hypotension and the failure of naloxone administration as a therapeutic modality. A 22 year old man, with a past history of intravenous drug abuse and several episodes of self-poisoning, presented to our hospital following ingestion of 30, 25 mg tablets captopril and possibly intravenous narcotics. On admission he was unconscious (Glasgow Coma Scale 6), pale and diaphoresing. Systolic blood pressure was 80 mmHg on admission falling to 40 mmHg with a relative bradycardia (60 beats/min). There was no clinical evidence of alcohol ingestion, opiate overdose or recent intravenous injection. Physical examination was otherwise normal. Urea and electrolytes, electrocardiogram and chest radiograph on admission were normal. The patient was given intravenous plasma protein substitute at a rate of 1 litre per hour over 8 hours. A bolus (10 mg) and infusion (0.04 mg/min) of naloxone was given over 1 hour with no effect on conscious level or hypotension. After 8 hours a systolic blood pressure of 90 mmHg was maintained with a concomitant improvement in conscious level.

There are 6 reports of angiotension converting enzyme inhibitor overdose^{1–6} with variable severity of hypotension and altered conscious level. The duration and severity of the hypotension was unexpected in view of the pharmacokinetics of the drug. Supportive therapy has been advocated with intravenous fluid and, rarely, inotropic support and renal-vasodilator therapy.

It is unlikely that in our patient a concomitant intravenous narcotic overdose may have contributed to the hypotension and the altered conscious level. There was no clinical improvement with intravenous naloxone. Naloxone pre-treatment has been used to block the hypotensive response associated with captopril treatment⁷ and we report that, in our patient, naloxone did not reverse captopril-induced hypotension.

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Percutaneous endoscopic gastrostomy in elderly patients

Sir,

I read 'Percutaneous endoscopic gastrostomy in elderly patients'¹ with interest. I was surprised the authors did not mention percutaneous non-endoscopic gastrostomy, which is probably the best method of gastrostomy for enteral feeding. The technique was first described independently by three radiologists in 1983.^{2–4} The stomach is initially inflated with air via a nasogastric tube. The stomach is located by fluoroscopy and the feeding tube is introduced under local anaesthesia using the Seldinger technique.

The technique has a number of advantages over percutaneous endoscopic gastrostomy: (1) It can be performed quickly and easily by one operator without the need for endoscopy, intravenous sedation or general anaesthesia; (2) it is simple and can be learned more easily as it does not require endoscopy skills; (3) because the feeding tube is introduced percutaneously under sterile technique rather than via the oesophagus, the incidence of

wound infection and aspiration is much lower; (4) a larger tube can be placed, permitting easier feeding of the patient; (5) the technique can be used even in the presence of severe oesophageal narrowing; (6) it is easier to change the tube when it becomes obstructed; (7) it is easier to advance the tube into the jejunum if required.⁵

The largest series to date reports a procedure-related mortality of 0.8% and a major complication rate of 1.2% which compares favourably with the figures quoted for percutaneous endoscopic gastrostomy.⁵ Percutaneous gastrostomy using the Seldinger technique would therefore appear to be the preferred method of gastrostomy for enteral feeding.

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