would seem to be indicated and might improve the poor prognosis of severe copper intoxication complicated by acute renal failure.

DAVID E C COLE

Department of Pediatrics,
University of British Columbia,
Vancouver, BC

¹ Cole, D E C, and Lirenman, D S, *Pediatric Research*, 1977, 11, 548.

² Evans, G W, *Physiological Reviews*, 1973, 53, 535.

Allopurinol and urinary stones

SIR,—I read with interest your leading article on the use of allopurinol in urinary tract stone disease (19 November, p 1302). A male patient presented to this department in November 1976 with a stone impacted at the right ureteric orifice which required Dormia extraction. Analysis of this stone showed it to consist of calcium, urate, oxalate, and a trace of ammonia. He had previously passed a stone spontaneously, but as he neglected to retrieve it no information about its composition is available. He had no evidence of parathyroid disease. His blood and urine urate levels were normal.

In accordance with the protocol of a trial we are conducting in the department he was started on allopurinol 100 mg thrice daily. However, he formed another stone, which was removed by left upper ureterolithotomy in November 1976. Analysis of this stone showed that it consisted of calcium ammonium phosphate; neither urate nor oxalate could be detected. He is still on allopurinol and remains well and free from stone.

The patient is one of a group of subjects currently being studied with regard to the effects of allopurinol on urinary oxalate excretion. Most subjects within the group have shown a significant reduction in urinary oxalate values. It is interesting that in the case described there has been a change in stone composition and in particular that no oxalate has been found, although this is a common constituent of stones within this area.¹

P J PATERSON

Urological Department,
Royal Infirmary,
Glasgow

¹ Sutor, D J, and Wooley, S E, *British Journal of Urology*, 1971, 43, 268.

Crush injuries

SIR,—Your leading article on this subject (12 November, p 1244) states that "despite the advent of effective dialysis as treatment for post-traumatic oliguric renal failure, the overall mortality of 60-70% has not changed substantially from that reported 25 years ago."

In 1974 Flynn² reported the UK overall mortality for post-traumatic acute renal failure to be 50%. At Halton we have compared two periods, 1957-64 and 1965-75, with regard to mortality in trauma patients with acute renal failure. While mortality in the first period was 64%, this was reversed in the second period to 34%.^{2,3} The most important difference in management was the institution of daily dialysis, high-calorie feeding, and subsequently total parenteral nutrition in the latter group. Lordon⁴ talks about frequent dialysis in his patients (on whom your statistics are based) but then goes on to say that dialysis in those who died was once every 2-6 days.

He also states that improvement in parenteral nutrition might have been beneficial because of the marked catabolism in his patients. Adequate parenteral nutrition is not possible without daily dialysis because of the obligatory volumes required to provide the necessary calories and protein.

Control of catabolism and adequate dialysis will improve healing⁵ and reduce the susceptibility to sepsis.⁶ If early and adequate attention is paid to debridement and amputation, daily dialysis with total parenteral nutrition to control catabolism offers a much better prognosis than one might be led to believe from your article.

DAVID J RAINFORD

Renal Unit,
Princess Mary's Royal Air Force Hospital,
Halton,
Aylesbury, Bucks

¹ Flynn, C T, in *Acute Renal Failure*, ed C T Flynn. Lancaster, MTP, 1974.

² "Renal Problems in Trauma," Seminar, R N H Haslar, February 1977. Unpublished.

³ Rainford, D J, *Anaesthesia*, 1977, 32, 277.

⁴ Lordon, R E, and Burton, J R, *American Journal of Medicine*, 1972, 53, 137.

⁵ Teschan, P E, et al, *Annals of Internal Medicine*, 1960, 53, 992.

⁶ Parsons, F M, et al, *Lancet*, 1961, 1, 129.

IUCDs—a contraindication to removal

SIR,—I report here a case which shows that if an intrauterine contraceptive device (IUCD) is to be removed for reasons other than cessation of contraceptive measures it should not be removed at the time of expected ovulation.

A 32-year-old woman presented recently with six weeks' amenorrhoea and was found to be pregnant (last menstrual period 17 September 1977). She had had four previous pregnancies, all of which had been normal resulting in normal deliveries at full term, the children's ages spanning 8 to 2 years. She had had no serious illnesses in the past other than two episodes of pneumonia in the last three years, the last attack being in 1975. Investigations had not shown any underlying cause, though she was known to have a sensitivity to house dust which caused rhinorrhoea and occasional wheezing on exposure to dust.

For contraception in the past she had relied mostly on the vaginal diaphragm. Between her first and second pregnancies she had taken oral contraceptives, but these had caused unacceptable side effects and had been discontinued. Following the birth of her last child in November 1975 she again used a diaphragm, but when she had finished breast-feeding the infant, and on resumption of menstruation, she requested a change and in October 1976 she was fitted with a Gravigard Copper-7 IUCD. Her normal menstrual cycle came every 25-28 days, each period lasting six days with a moderate loss. Over the next few months her periods lengthened to 10 days and by September 1977 this had further extended to two weeks and it was decided that her IUCD should be removed. This was done on 30 September, whereupon she resumed the use of her diaphragm. She is a sensible and responsible person who had used this form of contraception successfully for some years previously, but despite this she presented five weeks later and was found to be pregnant.

Close questioning revealed that, although she had been scrupulous in observing contraceptive measures since the removal of the IUCD, she had had intercourse 48-72 h before this was done. Removal was effected 13 days after the first day of her last menstrual period and it therefore seemed likely that the IUCD had been removed just in time to allow implantation of a fertilised ovum. In view of all these circumstances she was referred to a consultant gynaecologist, who agreed to terminate the pregnancy.

Of course it is possible that this unfortunate sequence of events did not occur and that she

became pregnant simply because of failure of her diaphragm. Nevertheless, it is possible that it did occur in the manner suggested and this possibility should be borne in mind in future. In the large majority of women—that is, those with regular menstrual cycles—such an unwanted pregnancy can be avoided simply by ensuring that IUCDs are not removed during a time, say, seven days each side of midcycle. So far as I can tell from inquiries made so far this is not standard practice. I believe that there is a good case for observing this precaution in future.

D F E THALLON

Tring, Herts

Drug-related red-cell aplasia

SIR,—The interesting report by Drs G Reid and A C Patterson (3 December, p 1457) of a case of red-cell aplasia apparently due to gold therapy illustrates the difficulties encountered in identifying cause and effect in this and other marrow hypoplasias. Wintrobe¹ lists 13 drugs believed to cause red-cell aplasia, while another review includes 16.² In some instances the evidence rests on only a very few anecdotal reports. The situation is also confused by the fact that in some cases the patient's underlying disorder was one itself occasionally associated with red-cell aplasia. In the present case the patient had received at least two other drugs known to cause marrow depression and was suffering from rheumatoid arthritis, one of a spectrum of connective tissue disorders in which red-cell aplasia may occur,³ presumably on the basis of autoimmune disease.³ The suspicion that a particular drug is responsible is naturally increased when withdrawal of a single drug, any others being continued at their previous dosage, is followed by remission of anaemia, though for obvious reasons this is rarely, if ever, justifiable in practice. Yet another difficulty is the tendency of red-cell hypoplasia to remit spontaneously without treatment; indeed, it is possible that many cases go unrecognised unless the patient is under haematological surveillance.⁴

These diagnostic pitfalls are further illustrated in the following case, seen in this clinic. A man of 72 was referred by Dr R V Stone because of a history of anaemia of six weeks' duration. There was no history of exposure to drugs apart from chlorotrianisene (TACE), which the patient had taken for 10 years after a clinical diagnosis of carcinoma of the prostate, and prednisolone (40 mg/day), which had been given for a month after the onset of the anaemia. Clinical examination showed mucosal pallor and a moderately enlarged prostate but no other signs. There was no evidence of metastatic disease, and the blood creatinine concentration and serum acid phosphatase activity were normal. The blood count on 21 July 1977 was: haemoglobin 6.6 g/dl, leucocytes $13.5 \times 10^9/l$ ($13\,500/mm^3$) (neutrophils 84%), platelets $224 \times 10^9/l$ ($224\,000/mm^3$), reticulocytes 0.02%. Marrow aspiration confirmed profound red cell hypoplasia, granulopoiesis and thrombopoiesis being normal. Since high-dose oestrogen therapy may diminish erythropoietin production⁵ and there is a report of marrow depression in animals following prolonged oestrogen administration⁶ it was postulated that the patient's red cell aplasia was due to this therapy, which was stopped, as was the prednisolone, to which there had been no response. A blood transfusion was given. Further investigation showed that his serum contained a small quantity of paraprotein, identified as IgG-kappa type, but there was no other evidence of lymphoma or immunoproliferative disease and it was decided that his was an example of "benign monoclonal gammopathy."