

the serum 25-OHD levels had risen significantly ( $P < 0.05$ , paired  $t$  test) above cord values by 5 weeks of age, but there were no intragroup differences in the other serum variables measured. In group 1 infants, the serial changes in the serum variables measured had been reported.<sup>1</sup>

Although 4 of the 6 infants in our study who showed radiological skeletal abnormalities received some human milk during their hospitalisation, for reasons stated above, our data neither support nor refute the findings of Sann *et al.*<sup>2</sup>

## Familial benign copper deficiency

Sir,

The case report by Méhes and Petrovicz<sup>1</sup> requires comment. They state that the serum copper of their patient was found to be 49.7 and 44.6  $\mu\text{g}/100\text{ ml}$  (7.8 and 7  $\mu\text{mol/l}$ ) (determined by atomic absorption spectrophotometry) while the serum caeruloplasmin concentrations were 'repeatedly normal, 0.30 to 0.44 g/l.' These figures are mutually incompatible. Caeruloplasmin is a protein of fixed copper content of approximately 0.3%.<sup>2</sup> As the copper in caeruloplasmin is present in a fixed ratio, serum concentrations of between 0.3 and 0.44 g/l of the protein necessitate a copper concentration in the serum of not less than 90  $\mu\text{g}/100\text{ ml}$ , and reaching as high as 132  $\mu\text{g}/100\text{ ml}$  (20.7  $\mu\text{mol/l}$ ). To this must be added a small percentage of 'free copper' always present in plasma, but not exceeding 10%. Thus, if the caeruloplasmin concentrations for the patient quoted are correct his serum copper must have varied between 95 and 140  $\mu\text{g}/100\text{ ml}$  (14.9 and 21.9  $\mu\text{mol/l}$ ), two to three times the reported figure. The only alternative explanation to an analytical error is that this patient has a mutant caeruloplasmin with only half the normal copper content: if such is the case the observation should be adequately documented.

### References

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Dr Méhes and Dr Petrovicz comment:

The discrepancy between low serum copper and normal caeruloplasmin concentrations is certainly remarkable. Although the heterogeneity of caeruloplasmin is well-known and a significant methodical variation is also to be considered, we do not know of an explanation for this phenomenon in the family reported by us. However, since a close positive correlation between blood copper and caeruloplasmin concentrations was obtained in many hundreds of examinations of both normal and hypocupraemic subjects in our laboratory, analytical errors in the given case seem to be most unlikely.

The existence of a mutant caeruloplasmin is a possibility, but at the moment we can only speculate on this.

## Hyperuricaemia as a cause of acute renal failure complicating cardiopulmonary bypass surgery

Sir,

We write in response to the interesting article by Rigden *et al.*<sup>1</sup> on acute renal failure complicating cardiopulmonary bypass surgery. Acute renal failure is an important and potentially very serious complication in children undergoing cardiopulmonary bypass surgery. Although the authors reported a relatively low acute renal failure rate of 5.3% in their 456 children, the mortality rate of those children with acute renal failure was still high at 50%. It is important therefore to identify the adverse predisposing factors so that preventive measures may be taken.

The three factors they identified are young age, complex cardiac lesions, and long overall bypass time. However, they failed to mention hyperuricaemia as a possible contributory factor that may cause acute renal failure in their patients. Hencz *et al.*<sup>2</sup> recently reported that extreme hyperuricaemia occurred in infants and children undergoing open heart surgery. We, too, in a prospective study of 97 children suffering from congenital heart disease (age ranging from 2 weeks to 14 years) found that 42% of our patients had raised serum uric acid concentrations.<sup>3</sup> The adverse factors which predisposed to hyperuricaemia were young age, complex cyanotic congenital heart disease with extreme polycythaemia, hypoxaemia, and low oxygen availability.

Adachi *et al.*<sup>4</sup> have shown that patients treated with allopurinol before open heart surgery were less likely to need DC counter shock to restart the heart beat after extracorporeal circulation. In addition, allopurinol was effective in preventing the damage to cellular structures and in minimising changes in metabolism, including a rise in serum uric acid concentrations in patients undergoing open heart surgery. Hence it is important to identify patients with hyperuricaemia before cardiopulmonary bypass surgery so that measures may be taken to prevent uric acid nephropathy causing acute renal failure.

### References

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