Dr Etzioni and Dr Benderley comment:

We agree with Lapatsanis *et al.* that cystic fibrosis should be included in the differential diagnosis of unexplained hypochloraemia. On the other hand, we do not think that one should exclude cystic fibrosis in cases of water intoxication, as was our case.

In the case reported by Lapatsanis *et al.*, and in the case reported by Barbero and Sibinga,¹ the patients suffering from cystic fibrosis who showed hyponatraemia and hypochloraemia also had severe dehydration. The reason for the hyponatraemia was excessive loss of fluids containing large amounts of salt.

In water intoxication the situation is the opposite. These children are overhydrated and not dehydrated. While correcting abnormal electrolytes in our patient, she also lost 5% of her body weight. She had dilutional hyponatraemia.

Thus, when dealing with hyponatraemia and hypochloraemia in patients suffering from water intoxication, one does not have to rule out cystic fibrosis if the patient is overhydrated and not dehydrated.

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Effect of puberty on rates of bone growth and mineralisation

Sir,

In their recent paper Krabbe *et al.*¹ described a prolonged acceleration of bone mineralisation in girls and boys towards the end of their pubertal growth spurt in height and after it. The data on which this finding rests were obtained by measuring bone mineral content in the forearm. As growth in width (or cross-sectional area) of the long bones is considerably slower by that time,² it seems reasonable to suggest that most of the increase in bone mineral content is due to a greater degree of mineralisation (or mineral density) of bone tissue.

To explain the prolonged increase in bone mineral content Krabbe *et al.*¹ invoked the action of several hormones, but I feel this is not quite correct. In periods of rapid growth—such as during the pubertal growth spurt—bone tissue turnover is high, as shown by the high level of plasma alkaline phosphatase³ which indicates an increased rate of bone formation, and the high rate of excretion of hydroxyproline,⁴ which indicates increased bone resorption. This increased formation and resorption of bone and the pubertal growth

spurt all depend on the change in endocrine activity which is the hallmark of puberty. However, the prolonged increase in bone mineral content occurs at the end of the period of increased turnover and beyond it and therefore must be caused by a different mechanism.

When bone turnover is high, a large proportion of the bone tissue consists of new, incompletely mineralised lamellar bone of low mineral density.⁵⁻⁶ After growth in stature is complete, rates of bone accretion and resorption diminish sharply, and the mineral density of the bone gradually increases by secondary mineralisation of the incompletely mineralised new bone.⁷ This process is responsible for the increase in bone mineral content. It is dependent on the physicochemical properties of bone matrix and bone mineral, and there is no evidence that bone mineral content increases by the action of endocrine substances in the manner suggested by Krabbe *et al.*¹

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Dr Krabbe and co-workers comment:

We thank Dr Steendijk for his interest in our paper.¹ His main point is that the prolonged phase of accelerated mineralisation that follows the pubertal growth spurt is determined by *local* factors—that is the physicochemical properties of bone matrix and bone mineral. Teleologically, this view is a very restricted one. In our opinion it is not likely that local bone factors alone take care of the intense mineralisation of the entire skeleton. More than half of all the minerals finally deposited in the skeleton have to traverse the blood stream within the relatively few years after the pubertal growth spurt.¹ This task, ideally, should be governed by one hormone which produces the bone matrix and keeps the supply of minerals at a suitably high level. In a forthcoming paper we present a unifying concept of bone growth and mineralisation, and describe blood mineral homeostasis during childhood and