The patient was a 19 year old previously healthy man who was injured while skiing and sustained complete tetraplegia. X-ray examinations disclosed fractures of C4 and C5. On admission to hospital he had normal serum concentrations of calcium, phosphate, creatinine, and parathyroid and thyroid hormones. Repeated chest X-ray films were normal, as were X-ray films of the skull, lumbar spine, and pelvis.

Two months after the accident the serum calcium concentration increased above 3 mmol/l (12 mg/100 ml) (figure); he developed anorexia, malaise, vomiting, and abdominal pains, and his general condition declined. His daily fluid intake was increased (3 l/day), and frusemide was given; the serum calcium concentration and abdominal complaints remained unchanged. Treatment with salcolamin (Miacalcic), 100-200 MRC units/h intramuscularly or subcutaneously, was started, and the serum calcium concentration decreased to 2.5 mmol/l (10 mg/100 ml) but then increased again. Sodium acid phosphate was given by mouth; symptoms resolved, and near normal serum calcium concentrations were established.

Four months after the start of the combined treatment his symptoms and hypercalcaemia recurred, serum calcium concentrations exceeding 3 mmol/l (12 mg/100 ml) being recorded. It was then decided to try disodium etidronate (Didronel) 5 mg/kg/24 h by mouth; salcolamin and sodium acid phosphate were stopped. Within a month he had essentially normal serum calcium concentrations, his gastrointestinal complaints had resolved completely, and he began to gain weight. Two months after disodium etidronate was stopped he still had normal serum calcium concentrations.

The effects of disodium etidronate on urinary hydroxyproline excretion, a finding reported previously with treatment with disodium etidronate and of unknown importance. The possibility that the doses of the drug used in our patients were too low does not seem likely.

Treatment with disodium etidronate confers a risk of developing osteomalacia, and long term use of high doses should be avoided. Disodium clodronate and disodium aminohydroxypropildenediphosphate show a lesser tendency to cause osteomalacia. Disodium clodronate prevents bone mineral loss and hypercalcaemia in paraplegic patients but has now been abandoned because of potentially carcinogenic effects.

In conclusion, we suggest that disodium etidronate (5 mg/kg/24 h for less than six months) or, preferably, disodium aminohydroxypropildenediphosphate be used in hypercalcaemia due to immobilisation when conventional treatment is insufficient.

OTHER FINDINGS
Urinary hydroxyproline concentrations were raised in both patients and did not change during treatment with disodium etidronate. No side effects of disodium etidronate were noted.

Comment
In both these patients serum calcium concentrations returned to normal within one month after the start of treatment with disodium etidronate. Interestingly, no changes occurred in urinary hydroxyproline excretion, a finding reported previously with treatment with disodium etidronate and of unknown importance. The possibility that the doses of the drug used in our patients were too low does not seem likely.

Treatment with disodium etidronate confers a risk of developing osteomalacia, and long term use of high doses should be avoided. Disodium clodronate and disodium aminohydroxypropildenediphosphate show a lesser tendency to cause osteomalacia. Disodium clodronate prevents bone mineral loss and hypercalcaemia in paraplegic patients but has now been abandoned because of potentially carcinogenic effects.

In conclusion, we suggest that disodium etidronate (5 mg/kg/24 h for less than six months) or, preferably, disodium aminohydroxypropildenediphosphate be used in hypercalcaemia due to immobilisation when conventional treatment is insufficient.

CORRECTIONS