that could be suppressed voluntarily for a short period. Furthermore, his speech had become strained and low volumed, at times disappearing altogether. The dosage of clonazepam was gradually reduced to 2 mg/day over the following 2 weeks. This resulted in the exacerbation of the HPX. The Tourette-like symptoms gradually diminished over the next month and his speech became more clear and loud.

Serotonergic mechanisms have been implicated in the pathogenesis of Tourette syndrome. Singer *et al.*³ and Crosley⁴ have postulated that serotonin deficiency underlies the major symptoms of this illness. In fact, cyproheptadine (a serotonin antagonist) has been reported to exacerbate Tourette syndrome.⁴ In view of this, it is surprising that our patient developed this syndrome following administration of a serotonin agonist which would be expected if anything to ameliorate such a condition. However, it is significant that the syndrome only appeared approximately 2 months after therapy with clonazepam had been initiated. This may indicate that the agent produced subsensitivity of postsynaptic serotonin receptors during this period resulting in the presentation of a paradoxical Tourette syndrome.

M.A. Gillman
R. Sandyk
South African Research Institute
Suite 9 Highlands House,
(NBS Centre)
173 Louis Botha Avenue,
Orange Grove 2192,
Johannesburg, South Africa

References

1. Fariello, R.G., Schwartzman, R.J., Steven, S. & Beall, L. Hyperexplexia exaggerated by occlusion of posterior thalamic arteries. *Arch Neurol* 1983, **40**: 244–246.
2. Andermann, F., Keene, D.L., Andermann, E. *et al.* Startle disease of hyperexplexia; further delineation of the syndrome. *Brain* 1980, **103**: 985–997.
3. Singer, H.S., Rabins, P., Tune, L.E. Coyle, J.T. Serum haloperidol in Gilles de la Tourette syndrome. *Biol Psychiatry* 1981, **16**: 79–84.
4. Crosley, C.J. Decreased serotonergic activity in Tourette syndrome. *Ann Neurol* 1979, **5**: 596–597.

Hypercalcaemia due to sarcoidosis

Sir,

Dr Gibbs and Dr Peacock¹ maintain that hypercalcaemia in sarcoidosis results from a raised serum 1,25 dihydroxy vitamin D. They do not say, however, why it occurs in the first place. It is likely that the increased vitamin D level is secondary to the apparent disappearance of the parathyroid hormone in these cases. Immunoglobulins secreted by granulomas in the parathyroid glands appear to block the actions of the hormone, unless hydrocortisone is given as well. The hypercalcaemia is then corrected in most cases, and the vitamin D3 falls to normal, also. The abnormality persists, however, in exceptional cases, probably because there is then tertiary hyperparathyroidism or a parathyroid adenoma present.^{2,3} Thirty six cases of this rare syndrome have been reported.

Gerald A. MacGregor
Percy Place
31 Epsom Road
Guildford
Surrey GU1 3LA, UK

References

1. Gibbs, C.J. & Peacock, M. Hypercalcaemia due to sarcoidosis corrects with biphosphonate treatment. *Postgrad Med J* 1986, **62**: 937–938.
2. MacGregor, G.A. Abnormal calcium metabolism in non-metastatic sarcoidosis. *Br Med J* 1977, **1**: 233–234.
3. MacGregor, G.A. Cimetidine in hyperparathyroidism. *Lancet* 1980, **i**, 980–981.