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#### Phenytoin-induced choreoathetosis

Sir,  
Phenytoin-induced movement disorders in patients with brain tumours have been reported rarely and are usually associated with toxic blood levels.<sup>1,2</sup> To the best of our knowledge only one case of choreoathetosis has previously been described in association with therapeutic levels of the drug.<sup>3</sup> Another case is now presented.

A 54 year old woman was admitted with 3 tonic-clonic convulsions in quick succession. A left frontal glioma had been diagnosed in 1987 and a parathyroid adenoma excised in 1989 but she had been asymptomatic until the day of admission. She was given a 700 mg loading dose of intravenous phenytoin (weight 53 kg) and subsequently switched to 300 mg orally, but she received only one dose. The next day she had no further fits and examination revealed no focal neurological signs or evidence of raised intracranial pressure. A computed tomographic brain scan confirmed a left frontal glioma with some surrounding oedema and midline shift. Serum calcium was normal. On the second day she developed sustained choreoathetoid movements affecting her mouth, neck and limbs, disinhibited and bizarre speech and mental confusion. There was no dysarthria, ataxia, nystagmus or diplopia. A phenytoin level at this time was 12 mg/l (therapeutic range 7–17) and an electroencephalogram revealed no significant abnormality. Her phenytoin was stopped and sodium valproate started. Complete resolution of her symptoms occurred within 24 hours.

When confusion develops in a patient with a brain tumour and a therapeutic blood level of phenytoin it is likely to be attributed to the neoplasm. The further complication of choreoathetosis is unusual and likely to prompt more extensive investigation. The awareness that in the presence of intracranial pathology a therapeutic dosage of phenytoin may produce such adverse effects may avoid unnecessary further tests and lead to prompt withdrawal of the offending drug.

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#### Electrocardiographic changes occurring after brief antimony administration in the presence of dilated cardiomyopathy

Sir,  
Sodium stibogluconate remains the first line therapy of choice for Indian kala-azar. Recent reports indicate that prolonged treatment may be required to achieve high cure rates.<sup>1</sup> The electrocardiogram (ECG) changes produced by sodium stibogluconate include sinus bradycardia, T wave inversion in the precordial leads and prolongation of the QT<sub>c</sub> interval.<sup>2</sup> These changes are usually dose dependent and are thought to represent a myocarditis produced by gradual accumulation of antimony in the myocardium.<sup>3</sup> Pandey *et al.*<sup>3</sup> have recently reported that no ECG changes were observed in 50 patients of kala-azar during the first 3 weeks of treatment with sodium stibogluconate.

We treated a 17 year old girl with parasitologically confirmed kala-azar, using 0.1 ml/kg of sodium stibogluconate (10 mg/kg antimony) per dose. This patient had idiopathic dilated cardiomyopathy, proven by endomyocardial biopsy, in addition to kala-azar. She was monitored closely with ECG every alternate day, and was found to manifest symmetrical T wave inversion of 5 mm in the precordial leads V<sub>1</sub>–V<sub>6</sub> on the seventh day of treatment, necessitating drug withdrawal. The QT<sub>c</sub> interval, however, remained constant at 0.42–0.44 s, and the heart rate at 150/min. A repeat MUGA scan revealed that ejection fraction and hypokinesia were unchanged compared to the pretreatment scan. The ECG was unchanged when follow-up ceased two weeks later.

Our experience suggests that in patients with underlying myocardial disease, small doses of antimony may induce ECG changes usually associated only with larger cumulative doses. These early changes may not always be accompanied by deterioration in cardiac function. Such patients, however, should have more frequent monitoring of cardiac status and ECG than patients with normal cardiovascular systems.

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