



Figure 1 Haemodynamic changes after first dose of enalapril 10 mg. ○—○, BP (mmHg) (supine); ●—●, pulse (min⁻¹).

should be used in 'at risk' patients. This line of action should ensure that any symptomatic first-dose responses are short-lived, but it may lead to a false sense of security. This case demonstrates that the pharmacological differences of captopril and enalapril are more profound than may be realised, and I would therefore suggest that 'test-dosing' should be done with a small dose of the same ACEI that is intended for long-term therapy.

The relative bradycardia seen during this patient's hypertensive reaction is noteworthy, and may suggest that tests of autonomic function could be used to screen for at-risk patients.

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Facioscapulothoracic syndrome with cardiomyopathy

Sir,

Cardiomyopathy and electrocardiogram (ECG) abnormalities have been associated with several neuromuscular disorders.¹ In facioscapulothoracic FSH syndrome, cardiac involvement may be present in some patients in the form of atrial paralysis or even generalized cardiomyopathy.²

Patients with genetic (FSH) weakness may have histological evidence of myopathy, neuropathy and inflammation. The term FSH syndrome is presently used to designate this entity.

We have investigated a 12 year old boy with clinically and electromyographically proven FSH syndrome who had normal early motor development until age 6 years when mild weakness in his arms was noted.

On examination, he appeared healthy and showed marked muscle wasting and weakness of the upper arms, the neck flexors and shoulder girdle. He also had a mild facial weakness when whistling. There was no pelvic or peroneal muscle involvement. Tendon reflexes were slightly reduced.

Serum cardiac enzyme levels were within normal limits. Electromyogram (EMG) examination indicated a myopathic process with a normal motor nerve conduction velocity. A muscle biopsy was normal apart from a few central nuclei. Chest X-ray was normal. The surface ECG showed only inverted T waves. The echocardiography findings were suggestive of a cardiomyopathy. The posterior ventricular wall motion was found to be slightly diminished. Cardiac catheterization revealed a probable cardiomyopathy.

To verify the diagnosis an endomyocardial biopsy was done from the right ventricle during catheterization. Findings disclosed unusual morphological changes; fatty infiltration amongst the bundles and hyperkinetic nuclei along with increase in cardiac connective tissue.

Our case shows that the presence of cardiac involvement in muscle disorders cannot always be detected by performing solely simple-to-perform cardiac evaluation studies with chest X-ray, ECG and echocardiography. Cardiac muscle biopsy is an invasive test. We do not propose it should be done more routinely, but the point should be kept in mind that normal findings in the screening tests mentioned above do not necessarily exclude possible cardiac involvement.

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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.^{1,2} To the best of our knowledge only one case of choreoathetosis has previously been described in association with therapeutic levels of the drug.³ 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.¹ The electrocardiogram (ECG) changes produced by sodium stibogluconate include sinus bradycardia, T wave inversion in the precordial leads and prolongation of the QT_c interval.² These changes are usually dose dependent and are thought to represent a myocarditis produced by gradual accumulation of antimony in the myocardium.³ Pandey *et al.*³ 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 V₁–V₆ on the seventh day of treatment, necessitating drug withdrawal. The QT_c 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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