

*israelii* is a normal mouth commensal the significance of a positive finding on sputum culture and cytology may be difficult to assess. Bronchoscopy may be unhelpful<sup>3</sup>. Also since pulmonary actinomycosis may not be suspected because of its rarity, the appropriate transport media and culture techniques may not be utilized<sup>4</sup>.

The computed tomogram may prove to be useful in the diagnosis of this disease in its early stages. It showed the presence of a pulmonary infiltrate with air bronchograms and also showed that the disease did not cross the oblique fissure or involve the pleural space or chest wall. These features appear to correspond to the initial lesion of pulmonary actinomycosis as described by Bates and Cruickshank<sup>1</sup>. The operative specimen showed extensive involvement of the lower lobe and thus differed from the minor involvement seen on CT. This difference can be accounted for by the time interval of one month between CT and operation during which time the disease progressed.

There are few CT descriptions of pulmonary actinomycosis in the literature. Webb and Sagel<sup>5</sup> in two patients with actinomycosis involving the chest wall drew attention to the pulmonary consolidation with involvement of adjacent pleura and chest wall. They also observed the presence of

a pulmonary infiltrate with air bronchograms. James *et al.*<sup>6</sup> described alveolar consolidation; an air bronchogram could be seen on their accompanying CT. Stanley and Lusk<sup>7</sup> described an irregular tissue density containing small air bubbles. We would like to suggest that a pulmonary infiltrate with an air bronchogram is a CT feature of the initial lesion of pulmonary actinomycosis and the diagnosis should be considered when this pattern is seen.

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## Lightning strike and autonomic failure - coincidence or causally related?

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Keywords: autonomic failure; lightning-strike; corticosteroids

This report describes the investigation and management of a severe case of idiopathic orthostatic hypotension exacerbated by paroxysmal atrial fibrillation. A trial of 7 days prednisolone therapy increased 24-h blood pressures recorded with an ambulatory monitor but did not improve orthostatic changes on a tilt table. The possible contribution of a telephone-mediated lightning strike to the pathogenesis of these disorders is discussed.

#### Case report

In 1974, a 36-year-old man experienced a telephone-mediated lightning strike, losing consciousness for 5 min. His history included two unexplained syncopal episodes 5 years before. A few days after the lightning strike, he noticed occasional missed heart beats that developed over the years into rapid, irregular palpitations lasting hours.

In 1979, he began to experience frequent episodes of severe postural faintness, and postural hypotension was demonstrated (135/80 mmHg lying, 85/50 mmHg standing after 2 min). In the absence of other abnormalities, a clinical diagnosis of idiopathic autonomic neuropathy was made and led to treatment with 9  $\alpha$  fluorohydrocortisone, subsequently supplemented with sodium chloride tablets and then, because of continuing postural faintness, with dihydroergotamine. Ephedrine was ineffective and he was unable to tolerate indomethacin, propranolol or pressure stockings.

In 1980, features of penile erectile difficulty and urinary retention suggested widespread autonomic dysfunction. By 1986 his palpitations were symptomatically troublesome and atrial fibrillation was documented during an attack. An echocardiogram showed localized septal thickening without outflow tract obstruction. Digoxin, quinidine, procainamide,

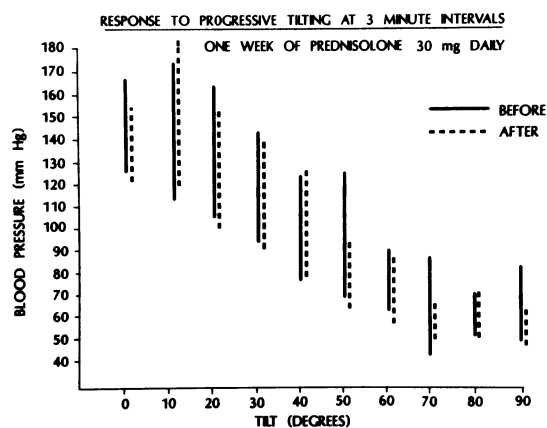


Figure 1. 1988 data whilst on therapy with NaCl 1800 mg three times a day, 9  $\alpha$  hydrocortisone 100  $\mu$ g three times a day and dihydroergotamine 10 mg three times a day

flecainide and sotalol were either ineffective or worsened his dizzy spells.

In July 1988, he was still experiencing frequent faintness despite compliance with his medications (see Figure 1). He was in sinus rhythm. The diagnosis of autonomic failure was then further refined. Sinus arrhythmia was absent and the Valsalva manoeuvre exhibited a clear denervation pattern. Plasma noradrenaline was exceedingly low with a super-sensitive pressor response to infused noradrenaline. The radiolabelled noradrenaline uptake across the heart was 25% (normal 50-65%) in keeping with lack of sympathetic innervation. An MRI scan of the brain and spinal cord was normal.

A trial of prednisolone 30 mg daily was undertaken for a week in the hope that steroids would sensitize the blood vessels to circulating catecholamines<sup>1</sup>, and/or that there was a novel auto-immune basis to the disorder. The 24-h ambulatory blood pressure recordings (Accutracker) showed an overall upward shift (see Figure 2) but with more severe nocturnal hypertension. As the magnitude of the blood pressure fall on the tilt-table was unchanged, the prednisolone was ceased.

In November 1988, rapid atrial fibrillation recurred. He was commenced on amiodarone with reversion to sinus rhythm and a prompt improvement in his symptoms and blood pressures (144/88 lying, 106/76 standing).

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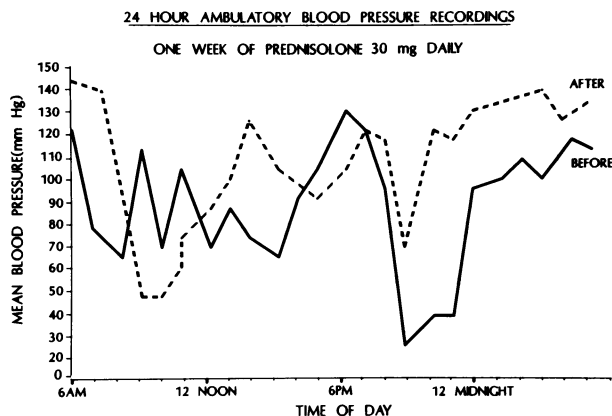


Figure 2. 1988 data whilst on therapy with NaCl 1800 mg three times a day, 9  $\alpha$ -hydrocortisone 100  $\mu$ g three times a day and dihydroergotamine 10 mg three times a day. Mean arterial pressures were calculated as diastolic pressure plus one third of the pulse pressure

### Discussion

Chronic syndromes of autonomic failure are difficult to classify but the patient fits into the category of idiopathic orthostatic hypotension or pure autonomic failure<sup>2</sup>. Since the patient dated much of his symptomatology to the lightning strike, we have examined the literature in that regard. A review<sup>3</sup> of lightning injuries highlights the fact that both central and peripheral nervous systems can be involved with a diversity in clinical syndromes that include spinal cord injuries and cranial and peripheral nerve palsies. Autonomic failure has not been described. When the symptomatology of telephone-mediated lightning strikes is compared to direct strikes<sup>4</sup>, the spectrum is similar although the frequency of certain symptoms differs. The principal pathological changes are peri-vascular widening and myelin degeneration in peripheral nerves<sup>5</sup>.

However, there is a low probability of a relationship between the strike and the patient's disorder, because peripheral nerve damage is usually isolated and affects large sensori-motor nerves whereas our patient had evidence for diffuse degeneration of his peripheral autonomic system. It would be easier to conceive central autonomic dysfunction arising from an isolated brainstem or hypothalamic lesion as a result of lightning injury. However, detailed autonomic

testing and the MRI scan did not support this. Also, some syncopal symptoms predated the lightning incident and his postural dizziness became severe only 5 years later. Nevertheless, the possibility remains that some as yet unexplained mechanism might have led to lightning injury exacerbating an underlying autonomic neuropathy.

Acute arrhythmia and myocardial damage including infarction<sup>6</sup> are well described after lightning strike but there are no reports of persisting arrhythmias. Focal myocardial damage could predispose to a subsequent arrhythmia, but since 10% of patients with hypertrophic cardiomyopathy have atrial fibrillation<sup>7</sup> this is the more likely underlying disorder. Cardiac sympathetic denervation appeared complete in this patient and would be expected to lead to up-regulation of myocardial  $\beta$ -adrenoreceptors. The resulting cardiac supersensitivity to circulating adrenaline might further predispose to arrhythmias. Part of the variation in his symptomatology over the years may have been due to episodes of unrecognized paroxysmal atrial fibrillation, since recent restoration and maintenance of sinus rhythm with amiodarone has been associated with marked symptomatic improvement even though significant orthostatic hypotension persists.

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## Hypothyroidism and renal impairment

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The symptoms of chronic renal failure can be very similar to those of myxoedema<sup>1</sup>, but any doubt about the true diagnosis is usually easily resolved when raised plasma urea

and creatinine levels confirm uraemia. Impairment of renal function can, however, be a manifestation of myxoedema. We recently encountered three patients who were referred because of suspected renal disease but who were found on further investigation to have hypothyroidism.

### Case reports

#### Case 1

A 36-year-old man was referred for nephrological advice because the plasma creatinine was found to be elevated during investigation of lethargy and muscle aches. On examination he had a husky voice, slow mentation, clubbing of the finger nails, and prolongation of ankle reflex relaxation. Urinalysis was normal. Investigations confirmed the raised plasma creatinine level but also showed hypothyroidism (Table 1). His symptoms rapidly improved after commencing thyroxine and plasma creatinine became normal again.

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