

**Comment**

The sauna is a potentially dangerous place,<sup>1</sup> many fatalities having been reported.<sup>2</sup> The temperature is thermostatically controlled over a range of 60° to 120° C, and is so high that any metal fittings would burn the skin on contact. Even the seats have to be made of a dry wood such as aspen. Seats with a higher sap content would be too hot to sit on. Door locks are not fitted and a simple spring catch keeps the door closed. The temperature in this sauna was set at 80° C but this can be increased by applying water from a ladle on to the hot coals. It seems surprising that burns in a sauna bath are so rare, but this is perhaps because people getting into difficulties in a sauna usually die.

Lawrence has shown that skin contact with a copper pipe filled with hot water becomes uncomfortable when the skin temperature reaches 43° C.<sup>3</sup> In the guinea-pig ear skin temperatures above 50° C for 60 s cause appreciable damage.<sup>4</sup> The low specific heat of air means that the human body in a sauna can normally withstand a temperature of 120° C, because the skin is cooled by evaporation of sweat and the circulation of blood through the skin. In the sauna the body temperature rises only by 1° C every 10 min.<sup>1</sup>

We can only speculate on the factors contributing to the burn in this case. Nevertheless, it seems likely that the tendency to a decreased blood pressure caused by the sauna together with a low cardiac output—possibly as a result of the cardiac effects of the sauna<sup>1</sup>—impaired the skin circulation. This would cause increased warming of the skin, an effect more pronounced on the outer and upper parts of the body exposed to greater heat. The return of hot venous blood in the superficial veins would give areas of even greater increase of skin temperature causing the appearances similar to an acute erythema abigne and superficial thrombosis, which was present in this patient. The increased temperature of moist air from the water applied to the heating coals would exacerbate this effect.

We conclude that the prudent sauna bather should check the exit before entering a sauna, should not go in alone, should take frequent cool showers to lower the skin temperature, and should not prolong a sauna bath past the point where he feels that he has had enough.<sup>1</sup>

<sup>1</sup> Taggart, P, Parkinson, P, and Carruthers, M, *British Medical Journal*, 1972, **3**, 71.

<sup>2</sup> *The Times*, 13 April 1972.

<sup>3</sup> Lawrence, J C, personal communication.

<sup>4</sup> Cruickshank, C N D, and Hersley, B, *Annals of Surgery*, 1960, **151**, 419.

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## Bilateral aneuralgic amyotrophy

Neuralgic amyotrophy is the generic term suggested by Parsonage and Turner<sup>1</sup> for the syndrome of acute pain, weakness, and variable muscle wasting and sensory loss around the shoulder girdle in the absence of trauma or structural cause. Classically pain and tenderness begin near the insertion of deltoid, at the top of the shoulder or around the scapula and are often severe. They are followed by weakness and wasting of one or more muscles, usually innervated from the C5/C6 nerve roots, and sometimes sensory loss, most commonly in the circumflex nerve distribution. In about half the reported cases specific or non-specific infections or vaccination may have a role in pathogenesis.<sup>2</sup>

We describe the unusual presentation of acute, bilateral, painless brachial neuropathy without sensory loss, but with evidence of early, mild disease of the spinal cord, in the absence of family history, obvious precipitant, or history of previous attack.<sup>3</sup>

**Case history**

A 41-year-old driver developed acute painless weakness of both arms. His shoulders and upper arms were mainly affected and the weakness was slightly

more pronounced on the right than the left. He had been fit and well before this. Half an hour later he noticed weakness of his right leg, which resolved within an hour. He had no changes in sensation or sphincter disturbance but noticed a transient erythematous rash over his right shoulder at the onset of his symptoms. He was admitted to another hospital, where he was found to be afebrile with definite proximal arm weakness without fasciculation. Reflexes were brisk throughout and clonus was elicited at the right ankle, but plantar responses were flexor.

When seen a month later, he had weakness (MRC<sup>4</sup> grade 2/3) and wasting of deltoids, spinati, biceps, and triceps on both sides with a little weakness (grade 4) in the forearms and intrinsic hand muscles, without wasting. No sensory loss or scapula winging was found. Biceps and triceps reflexes were preserved and all other tendon reflexes were slightly increased. The legs showed no weakness or wasting and he was otherwise normal.

The results of routine blood tests, and chest and cervical spine x-ray films were normal, as were CSF total protein, globulin, and cell count; a Metrizamide myelogram showed good root sleeve filling. Antibodies to influenza A and *Mycoplasma pneumoniae* were detected at titres of 1/40, which were unchanged a month later. Polio titres were consistent with previous immunisation. No antibodies were detected in the CSF.

The results of nerve conduction studies of the ulnar, median, and lateral popliteal nerves were normal. Erb's point stimulation showed increased latencies at the deltoid and triceps, electromyographic findings being reported as consistent with denervation atrophy in C5/6 innervated muscles due to a patchy, mixed axonal, and demyelinating neuropathy of the brachial plexus bilaterally.

**Discussion**

This case is unusual in the acuteness of onset, the painless, asymmetrical, bilateral signs in the arms, and the subsequent symptoms in the leg with mild bilateral pyramidal signs. The distal arm muscles were also affected, indicating spread of damage beyond the C5/C6 innervated muscles.

It is well recognised, however, that the classical picture is subject to considerable modification. Painless attacks occur in 6–50%<sup>1,3</sup> of reported series and may be bilateral in 15–30%.<sup>1,3,5</sup> Lesions of the cranial nerves, lumbosacral plexus, autonomic system, individual peripheral nerves,<sup>3</sup> and spinal cord<sup>1</sup> are also documented and sub-clinical disease of other parts of the nervous system may occur even in a classical presentation.<sup>3</sup> Atypical features are commoner in the familial recurrent forms, which may be associated with hypotelorism. The pathological feature here is a characteristic tomaculous neuropathy, while in the more classic, sporadic variety, it is patchy demyelination, with axonal damage in more severely damaged nerve fibres.<sup>3</sup> The pathogenesis is unknown, although postinfection or immunisation cases may have an allergic basis.<sup>2</sup> The prognosis depends on the extent of muscle wasting, but in general recovery occurs after 18 months or more and may be helped by physiotherapy.

We thank Dr Peter Hudgson for permission to report this case under his care and Dr David Barwick for the electromyographic and nerve conduction studies.

<sup>1</sup> Parsonage, M J, and Turner, J W A, *Lancet*, 1948, **2**, 973.

<sup>2</sup> Walton, J N, in *Brain's Diseases of the Nervous System*, 8th edn, ed J N Walton. London, OUP, 1977.

<sup>3</sup> Bradley, W G, et al, *Brain*, 1975, **98**, 381.

<sup>4</sup> *Aids to the Investigation of Peripheral Nerve Injuries*, 2nd edn. London, HMSO, 1943.

<sup>5</sup> Spillane, J D, *Lancet*, 1943, **2**, 532.

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**Correction****An atypical case of Cornelia de Lange's syndrome**

We regret that the title of the paper by Dr Jane Brylewski (25 March, p 756) was incorrectly printed, and should have read: "An atypical case . . ."