a role for complement similar to that in Thai children.³ The prevalence of dengue due to different serotypes can thus be expected to produce an increased incidence of dengue shock syndrome and haemorrhagic fever in the Caribbean.

¹ *Lancet*, 1976, 2, 239.

² Ventura, A K, and Hewitt, C M, *American Journal of Tropical Medicine and Hygiene*, 1970, 19, 712.

³ Bokisch, V A, *et al*, *New England Journal of Medicine*, 1973, 289, 996.

⁴ Halstead, S B, and O'Rourke, E J, *Journal of Experimental Medicine*, 1977, 140, 201.

⁵ Halstead, B, *Yale Journal of Biology and Medicine*, 1970, 42, 350.

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Department of Medicine, University of the West Indies, Kingston, Jamaica

H S FRASER, PHD, MRCP, consultant physician and lecturer

W A WILSON, MD, MRCP, consultant physician and lecturer

E J THOMAS, BSC, research assistant

J G P SISSONS, MB, MRCP, consultant physician and lecturer

Skateboard injuries

Though the popularity of skateboarding is increasing, only three series of injuries to skateboarders have been described,¹⁻³ two in the USA and one in Britain. The sport's current position may be judged by the \$25 000 prize money that a recent professional competition offered. The potential for injury may be realised by looking at the speeds and feats of agility that skateboarders attain. The world records include long jump over 18 five-gallon oil drums, a distance of 4.4 metres, high jump 150 cm, and downhill speed 91.7 kph.

The sport's popularity is difficult to judge in the United Kingdom. It has just been recognised by the Sports Council and a national association formed. Sales of skateboards are still increasing. One major sports shop in Sheffield sold 450 boards in October 1977 compared with 48 in October 1976. To find out what injuries have been severe enough to bring sufferers to hospital I have surveyed all casualty attendances resulting from skateboard falls from 1 August to 30 November 1977.

Patients and results

This period of review included August and September, when the weather encouraged skateboarding, and October and November, less encouraging months. Sheffield has four casualty departments serving a population of about 500 000. Most children up to age 15 attend the Children's Hospital casualty department, which is always open and whose experience has been reported.⁴ Very few children attend the Royal Infirmary casualty department, where my survey was conducted.

During the period under review 36 patients with skateboard injuries attended the department, compared with none in 1976. The patients' ages ranged from 7 to 37 years with 26 aged between 11 and 15; 6 between 16 and 19; 2 aged 7; 1 aged 10; and 1 aged 37. A total of 17 patients had fractures, including ulna and/or radius (8); scaphoid (1); clavicle (2); metatarsal (3); tibia and/or fibula (2); or hip (1). Grazes, sprains, and bruises accounted for the other 20 attendances including three patients with head injuries who had no complications. Only one patient was admitted (separation of acetal epiphysis). No abdominal injuries were recorded, and there were no deaths.

Discussion

Published reports suggest that there are an appreciable number of non-trivial skateboard injuries and that these are increasing, though most people have no residual effects. The injuries I found in this series are mild compared with those noted in a study in Hawaii.²

What can be done to reduce the number or severity of injuries? The provision of skateboard parks would help since here attendants can insist on wearing protective gear, provide training, and outlaw dangerous practices. These will also help to keep skateboarders off roads and pavements, where they are in danger from traffic and a menace to pedestrians. Protective wear is important, and helmets are increasingly being worn. Those developed for canoeing (£5.00) are

better than nothing, but those used in ice hockey are eminently suitable, being very substantial yet light and of an attractive design—but they are not cheap (£12.00). In Britain availability seems to be the main problem: a delivery of 120 of a slightly modified but cheaper ice hockey helmet sold out in two days at a Sheffield sports shop. Wearing knee and elbow pads, gloves, jackets, and long trousers all helps to reduce grazing.

The limited data of this study show that two areas where fractures frequently occur are not protected: the wrists and forearms and the ankles and feet. There are two problems in designing suitable protective wear for these areas. Firstly, the mobility of the joints must be maintained to allow agile movement of the skateboarder. Secondly, the protective wear must be lax enough to avoid it acting as a shock transmitter, merely moving the damage further along the structural chain. Suggested protection for the wrist and forearm ranges from simple support of the Tubigrip type to a rigid polypropylene imitation of a half-arm plaster-of-Paris. For the ankle and foot boots similar to those used in ice skating may provide protection at the expense of the fine control of balance that is an essential of this exhilarating but dangerous sport.

I thank Mr N R M Kay, the casualty officers, nurses, and administrative staff for their encouragement and help.

¹ Jacobs, R A, and Keller, E L, *Pediatrics*, 1977, 59, 939.

² Atienza, F, and Sia, C, *Pediatrics*, 1976, 57, 793.

³ Illingworth, C, *et al*, *British Medical Journal*, 1977, 2, 1636.

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Casualty Department, Royal Infirmary, Sheffield

IAN KEMM, MB, CHB, preregistration house officer

Sauna bath burn

Some of the hazards of sauna bathing are well recognised, and have been described in a publication by Taggart *et al*.¹ Nevertheless, a burn caused by exposure to the hot air of the sauna alone without the patient's coming into contact with the heating element has not been reported.

Case report

A 62-year-old man entered a sauna at 1330. After 15 min he poured water over the coils of the heating element to give humidity and increase the temperature. He found this unpleasant and wanted to get out of the sauna, feeling faint and weak, but was unable to open the door. He sat down on the bench to see out his allocated time of half an hour. Shortly after his time was up he was found sitting in the sauna by the attendants. As the door was opened he felt a severe pain from the impact of the cold air. He managed to walk out with help. At that time he was described as red all over.

On admission to hospital he was fully conscious. He had generalised erythema of the whole body except the lower abdomen and the perineal region. There were areas of blistering estimated at 13% of the total body surface over both shoulders and upper arm, upper part of the back, and outer aspects of both thighs. There were small spots of deep burn on the nostrils, both ears, and the lips. His blood pressure was 180/100 mm Hg, and body temperature 36°C; the haemoglobin concentration was 17.0 g/dl and the packed cell volume 0.52.

The patient had a history of hypertension and was taking digoxin, while an electrocardiogram showed atrial ectopic beats with right bundle branch block. The usual intravenous infusion of plasma was set up, but in view of the history of cardiac disease the rate of infusion was kept cautiously low and the patient's condition carefully monitored. The blood pressure, pulse, and urinary output remained perfectly adequate and the infusion was stopped after 18 h. The erythematous areas rapidly settled and returned to normal after 48 h. All the areas of the burn, which initially had been superficial skin loss with blistering, gradually underwent patchy deterioration, so that as some of the blistered areas gradually healed others progressed to full-thickness necrosis. Ten days after the accident the deep areas of burn were shaved and grafted. It was noted at operation that many of the strips of deep burn followed the pattern of thrombosed superficial veins. There were also similar areas of superficial vein thrombosis seen in the thigh split skin graft donor sites. By 19 days most of the grafts had healed but in a few areas further deep necrosis occurred with blistering of the grafts, requiring further grafts.

Comment

The sauna is a potentially dangerous place,¹ many fatalities having been reported.² 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.³ In the guinea-pig ear skin temperatures above 50° C for 60 s cause appreciable damage.⁴ 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.¹

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 sauna¹—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.¹

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

² *The Times*, 13 April 1972.

³ Lawrence, J C, personal communication.

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

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Department of Plastic Surgery, Bridge of Earn Hospital, Perth and Dundee Royal Infirmary, Dundee

ARTHUR MCG MORRIS, MA, FRCS, consultant plastic surgeon

SANJEEVA RAI, MB, FRCS, senior house officer in plastic surgery

Bilateral aneuralgic amyotrophy

Neuralgic amyotrophy is the generic term suggested by Parsonage and Turner¹ 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.²

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.³

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 (MRC⁴ 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%^{1,3} of reported series and may be bilateral in 15–30%.^{1,3,5} Lesions of the cranial nerves, lumbosacral plexus, autonomic system, individual peripheral nerves,³ and spinal cord¹ are also documented and sub-clinical disease of other parts of the nervous system may occur even in a classical presentation.³ 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.³ The pathogenesis is unknown, although postinfection or immunisation cases may have an allergic basis.² 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.

¹ Parsonage, M J, and Turner, J W A, *Lancet*, 1948, 2, 973.

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

³ Bradley, W G, et al, *Brain*, 1975, 98, 381.

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

⁵ Spillane, J D, *Lancet*, 1943, 2, 532.

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Regional Neurological Centre, Newcastle General Hospital, Newcastle upon Tyne NE4 6BE

R J M LANE, MB, MRCP, research associate and honorary registrar

J A DEWAR, MA, BM, registrar

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 . . ."