axillary perspiration. Half the patients reported compensatory sweating, mainly on the trunk, abdomen, and thighs, as in our patient. Two patients complained of sweating of the face elicited by the taste or smell of food. No patients developed a permanent Horner's syndrome, which has been reported as a long-term complication in up to 20% of patients undergoing upper dorsal sympathectomy[8]. Fifty-five of the 59 patients in the Kux study reported that they were highly satisfied with the results of the procedure.

Endoscopic sympathectomy is well tolerated, carries a low morbidity, and appears to be the operative procedure of choice for patients with severe palmar hyperhidrosis.

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Envenomation by the box-jellyfish - an unusual cause of ulnar nerve palsy

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Keywords: jellyfish; envenomation; neurotoxicity

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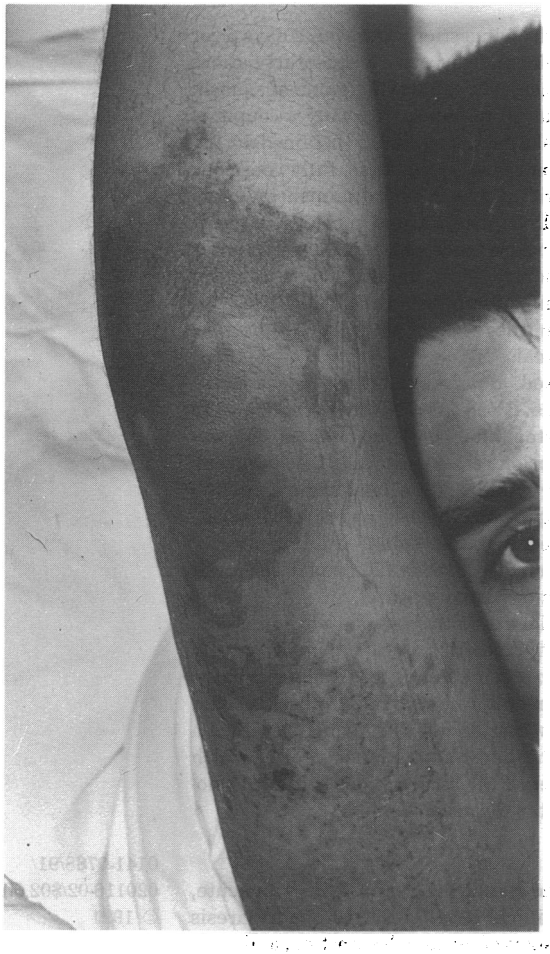

Figure 1. Skin changes over the right elbow, following envenomation by Chironex fleckeri, *10 days previously*

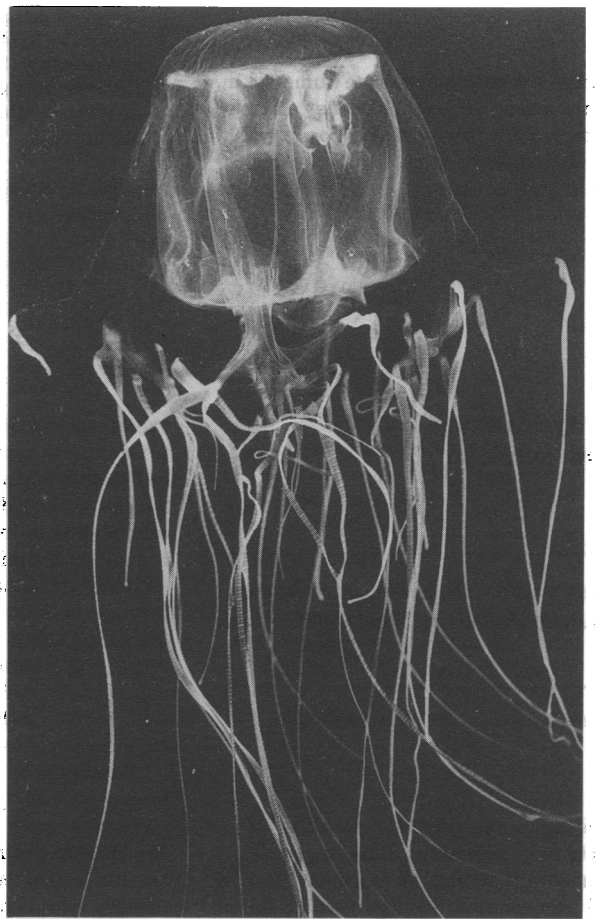

Figure 2. Chironex fleckeri *(box-jellyfish)*

Whilst systemic neurotoxicity is not uncommon following envenomation by the box-jellyfish, the isolated involvement of a single peripheral nerve has not been previously described, despite the frequency with which jellyfish stings occur worldwide. We describe such a case.

Case report

A 21-year-old British student was on a hockey tour in Thailand. Whilst swimming backstroke in the sea off Phuket he experienced sudden burning pain across his back and right arm. On return to the shore he developed wheezing and generalized oedema, and was noted to have livid wheals on his back and elbow. These subsequently developed the classical ladder-rung pattern of *Chironex fleckeri* (box-jellyfish) stings[1].

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He received no first aid until he arrived at a local hospital where he was treated for progressive anaphylaxis and circulatory collapse. After several days his systemic illness improved and he was left with deep discolouration over the dorsum of his right elbow and symptoms of a right ulnar nerve palsy. Ten days later he was transferred to our care.

Examination showed signs of intrinsic weakness of the right hand, with an incomplete sensory palsy in the ulnar nerve distribution. The skin overlying the elbow was deeply pigmented, and appeared close to necrosis (Figure 1). He was otherwise well with no systemic signs.

Nerve conductance studies showed reduced sweating in the ulnar side of the palm and a raised sensory threshold in the ulnar nerve territory. Electromyography showed conduction velocity diminished across the elbow, consistent with a lesion at that level.

Surgical exploration of the ulnar nerve at the elbow was carried out in case the delay was due to compression within the ulnar tunnel, however this was not confirmed. The nerve was oedematous and the adjacent muscle ischaemic. Despite its appearance, the skin healed without necrosis and four months later the ulnar nerve palsy was recovering.

Discussion

Few jellyfish have stings which can penetrate human skin, however members of the family Chirodropidae may all cause systemic envenomation. *Chironex fleckeri* (box-jellyfish, Figure 2) has been responsible for many deaths in tropical waters, particularly around Australia[2].

Envenomation may cause severe cutaneous pain and whealing, dermonecrosis, cardiac toxicity, pulmonary oedema and vasomotor failure. These may result in death unless treatment is prompt[3]. Peripheral neurotoxicity has been demonstrated in animal studies[4], but has not been reported in man. These effects result from multiple toxins found both in the discharging nematocysts and in the tentacles themselves. Rapid removal of the tentacles using vinegar, or dry sand reduces the local and systemic action of these toxins[5].

The absence of first aid in this case allowed high local concentrations of toxins to occur over the dorsum of the elbow. This resulted in pre-necrosis of the skin and underlying tissues. We postulate that the superficial position of the ulnar nerve at the elbow explains the development of the localised nerve injury.

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Guillain-Barré syndrome following severe exertion

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Keywords: Guillain-Barré syndrome; risk factor; exercise

The Guillain-Barré syndrome (GBS), consists of a progressive, symmetrical, ascending flaccid paralysis; areflexia; minimal or absent sensory impairment; and an albumino-cytological dissociation in the cerebro-spinal fluid[1]. Clinical and laboratory studies indicate that the syndrome is a result of auto-immune sensitization to neuritogenic peptides (eg the P_2 component of myelin basic protein)[2], initiated in areas with a deficient blood-nerve barrier[3], and ultimately resulting in demyelination. More than 30% of GBS cases have identifiable risk factors - infections, vaccinations, toxins, surgery, etc[2]. This report identifies unaccustomed severe exertion as a possible risk factor.

Case report

A previously healthy 22-year-old man was admitted with a 2-day history of initial muscle cramps, weakness and a sensation of 'numbness' in both lower limbs, which rapidly evolved within 24 h of admission to a symmetrical weakness of all four limbs with some perceived difficulty in breathing. Swallowing, visual and sphincter functions were normal. There was no history of recent febrile illness, rashes, sore throat, vaccination or drug ingestion. However, 2 days prior to the onset of clinical illness, this normally sedentary patient had engaged in a particularly exhausting game of squash, which had lasted about 90 minutes, with only a couple of minutes rest at intervals. No injuries were sustained during the game. Examination revealed an obese, fully conscious adult, afebrile, anicteric, areflexic, with diminished muscle power in both lower (MRC grade 2/5) and upper (MRC grade 3/5) limbs, a mild calf muscle tenderness, and absent plantar responses. Cranial nerves were intact. Pulse rate on admission was 136/min, regular rhythm; blood pressure was 150/95 mmHg in the supine position. Respiratory examination was normal.

Investigations showed a normal plasma biochemistry profile, with euglycemia. Blood smears were normal, and no pathogens were isolated after prolonged blood culture. Erythrocyte sedimentation rate was 4 mm/h (Westergren); and serological tests for syphilis, Widal agglutinin titres and urinary porphobilinogen tests were negative. Lumbar puncture demonstrated a cerebrospinal fluid protein of 200 mg/dl, glucose 2.8 mmol/l, chloride 120 mmol/l, cell count 1/mm^3, and no growth on tissue culture.

The patient's vital signs stabilized soon after admission, and he was started on hydrocortisone 100 mg intravenously as a bolus, followed with oral prednisolone 20 mg thrice daily. Clinical progress was monitored with daily muscle power/tidal volume/peak flow rate assessment, which revealed a clinical nadir on the third post-admission day, after which recovery was sustained. Full recovery, with late physiotherapy, was noted after 6 weeks.

Discussion

The patient showed the classic features of GBS - an acute, rapidly progressive, clinical onset of flaccid quadriparesis without any objective signs of sensory dysfunction, and early recovery without relapse. There was no clear precipitating factor, except for an isolated bout of severe unaccustomed

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