Mouse-directed computers and ulnar sensory neuropathy

While computers can be affected by "viruses", their users may be prone to pressure neuropathy. We recently examined two patients who presented with an isolated ulnar palmar-cutaneous-branch sensory neuropathy resulting from daily use of a mouse-directed personal computer (PC).

Both males, aged 23 (patient 1) and 34 (patient 2), presented with a two week and one month history, respectively, of paresthesia in the ulnar region of the right hand. Physical examination demonstrated sensory loss in the area of the palmar cutaneous branch of the right ulnar nerve. No weakness or atrophy was observed in these otherwise healthy young men.

Sensory nerve conduction studies of the palmar-cutaneous-branch of the right ulnar nerve revealed a 0.7 and 0.6 ms-longer latency compared with the left, in the first and second patient, respectively. The amplitude of the sensory action potential in the first patient was 12 μV at the affected side compared with 19 μV at the contralateral unaffected nerve. In the second patient these values were 14 μV and 21 μV, respectively. The dorsal cutaneous branch of the right ulnar nerve was normal in both patients, as well as needle electromyography and motor conduction studies of both median and ulnar nerves. Clinical and electrophysiological findings suggested an isolated right ulnar palmar-cutaneous-branch sensory neuropathy. After they abandoned the use of this steering device, recovery occurred progressively. Sensory nerve conduction studies of this sensory branch of the ulnar nerve were within normal limits six months later.

Both patients were using their PC for almost one year and had no predisposing factors for ulnar nerve damage. Their history clearly indicated that the region proximal to the wrist crease of the right arm was intermittently compressed while using the mouse (figure). The palmar sensory branch of the ulnar nerve arises proximal to the wrist crease and supplies sensory innervation to the proximal ulnar aspect of the palm. The deep palmar (motor) branch of the ulnar nerve can be damaged by recurrent pressure. However, lesion of the palmar sensory branch is very uncommon, especially when occurring from an occupational nature. This type of neuropathy should be recognised in patients using mouse-directed computers and can simply be prevented by using the keyboard. In addition, this case is not reported to discourage the use of this type of steering device, but to limit its use when any sensory disturbance occurs in the proximal ulnar aspect of the palm.

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Oedema associated with the interruption of preganglionic sympathetic tract

Although oedema and swelling of the legs are common in patients with acute transverse myelopathy, the mechanism is poorly understood. We report a patient with multiple sclerosis who showed a marked pitting oedema in the lower limbs following acute transverse myelopathy, which was associated with the interruption of preganglionic sympathetic fibres in the spinal cord.

The patient was a 46 year old female who suffered from the first attack of gait disturbance in April 1989. Sensory impairment below the level of T8, and increased knee and ankle jerks with extensor plantar responses and dysuria were noted. Protein and IgG levels in the CSF were elevated to 71-0 and 17-9 mg/dl, respectively and two oligoclonal IgG bands were detected. No oedema was observed in the legs or feet. Sym pathetic skin response (SSR) was studied according to a previously reported method.1

The patient lay in a supine position in a warm, quiet room. The skin temperature of the limbs was kept above 31°C. She was asked to open her eyes and not to fall asleep. Standard electromyographic disc electrodes were attached to the palm (G1) and dorsum of the hand (G2) as well as to the sole (G1) and dorsum of the foot (G2) bilaterally. Different types of stimuli were used to evoke the SSR. The endogenous stimulus was deep inspiration. Exogenous stimulus consisted of constant voltage of 50-150 V in single 200μs square pulses, applied to both supraorbital nerves. More than 10 electrical stimuli were administered at irregular intervals greater

Figure 1  Symathetic skin response of the patient. The plantar responses disappeared with the appearance of oedema in the legs in May 1990 (B). In December 1990 the oedema in the legs subsided and the plantar responses reappeared (C).
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