drove badly and ascribed this to not being able to see the kerb. As yet he has not given up driving because he thinks that it is up to the doctors at the eye hospital to tell him not to drive if they think that he is unsafe.

Comment

Our experience suggests that glaucoma may sometimes account for driving difficulties and raises again the difficult question of the duty of medical practitioners to warn their patients if their eyesight has deteriorated below the level consistent with safety on the road.

Patients over the age of 70, who want their driving licence renewed, need to apply with a certificate indicating that they have reached the required visual standard; this is taken as 6/15 on the Snellen chart. These elderly patients may, however, have glaucoma, as this is a relatively common disease in this age group (a prevalence of 1.1% was found among patients over the age of 65 in Wales²), and the presence of a visual field loss should not be overlooked.

¹ Cross AG. *Medical aspects of fitness to drive*. London: Medical Commission of Accident Prevention, 1976.

² Hollows FC, Graham PA. Intraocular pressure, glaucoma and glaucoma suspects in a defined population. *Br J Ophthalmol* 1966;**50**:570-86.

(Accepted 11 June 1982)

University Surgical Unit, University of Southampton SO9 5NH

J M MACKEAN, BA, research fellow

A R ELKINGTON, FRCS, senior lecturer in ophthalmology

Ulnar nerve palsy and walking frames

Distal ulnar nerve palsies have been described after repeated trauma to the heel of the hand in operators of pneumatic drills, carpenters using wood planes, secretaries using staplers and returning typewriter carriages,¹ and lathe workers using a chuck key. It has also been noted in motorists closing car doors and after prolonged bicycle riding.² We report two cases after the use of a walking frame.

Case reports

CASE 1

A 72-year-old woman fractured her left femur in October 1973 and a Thompson's prosthesis was inserted. In January 1980 a right Thompson's prosthesis was inserted after a fracture of her right femur and the patient has used a walking frame since then. In January 1981 she was admitted to the geriatric unit because of immobility. On examination she had claw hands with bilateral wasting and profound weakness of the interosseous and hypothenar muscles. No sensory loss was shown. The patient admitted to increasing difficulty using her hands since walking with a frame. Callosities over both hypothenar eminences corresponded to the sites of pressure while gripping her frame. Bilateral ulnar nerve palsies were diagnosed.

Ulnar nerve conduction velocity studies (see table) showed a prolonged terminal latency to abductor digiti minimi, considerably prolonged terminal latency to the first dorsal interosseous muscle, and a small amplitude or absent sensory nerve action potential from the fourth and fifth digits. Electromyography showed fibrillations at rest and no action potentials on volition in both first dorsal interosseous muscles, polyphasic long-duration action potentials, and reduced interference pattern in both abductor digiti minimi.

CASE 2

A 66-year-old woman presented in January 1981 with callosities on both hypothenar eminences, associated with ulnar claw-hand deformities, more pronounced on the left. She had had a scirrhous adenocarcinoma of the right breast treated by local radiotherapy in June 1978. In September 1979 she developed a pathological fracture of the left ilium and local recurrence of the carcinoma with axillary tethering, causing a swollen and painful right arm. At this stage she started to use a walking frame.

In March 1980 she complained of pain and weakness in the left leg, and on examination was found to have bilateral L5/S1 motor and sensory deficits due to a combination of osteoarthritis and vertebral metastases. From June

Results of ulnar nerve conduction velocity studies in two patients

	Normal	Right	Left
Case 1			
Ulnar nerve:			
Motor			
Terminal latency { to abductor digiti minimi (ms)	<4.0	5.8	4.5
to first dorsal interosseous muscle (ms)	<5.5	10.2	9.2
Palmar conduction time from first dorsal interosseous to abductor digiti minimi muscle (ms)	<1.5	4.4	4.7
Sensory (fifth digit)			
Amplitude (μV)	>5	2	Absent
Conduction velocity (m/s)	>45	39	Absent
Median nerve:			
Sensory (second digit)			
Terminal latency (ms)	>4.4	Not recorded	3.0
Conduction velocity (m/s)	>45		59
Amplitude (μV)	>5		20
Case 2			
Ulnar nerve:			
Motor			
Terminal latency { to abductor digiti minimi (ms)	<4.0	3.3	3.9
to first dorsal interosseous muscle (ms)	<5.5	5.1	7.3
Palmar conduction time from first dorsal interosseous to abductor digiti minimi muscle (ms)	<1.5	1.8	3.4
Sensory (fifth digit)			
Amplitude (μV)	>5	2.5	<1.0
Conduction velocity (m/s)	>45	43	37
Median nerve:			
Motor			
Terminal latency (ms)	<4.5	Not recorded	4.2
Conduction velocity (m/s)	>45		57
Sensory (first digit)			
Amplitude (μV)	>5		11
Conduction velocity (m/s)	>45		46

to August 1980 she received three courses of chemotherapy (vincristine, methotrexate, and chlorambucil), with a total dose of 4.5 mg vincristine.

Ulnar nerve conduction velocity studies (see table) showed a prolonged terminal latency to the first dorsal interosseous muscle, with normal or borderline terminal latency to abductor digiti minimi and decreased amplitude of the sensory nerve action potential from the fifth digits. Electromyography showed extensive denervation, with fibrillations and discrete motor-unit activity, in the first dorsal interosseous muscle and mild denervation in the abductor digiti minimi.

Comment

The deep branch of the ulnar nerve is particularly vulnerable to pressure against the hook of the hamate. We believe that this is the first report of ulnar nerve palsies secondary to chronic pressure from a walking frame. Both patients had bilateral palsies with severe disease of the deep terminal branch and lesser disease of the superficial (sensory) branch. Neither patient had clinical or electrical evidence of median nerve neuropathy. Case 1 had not received any drugs known to cause neuropathies. Case 2 had finished a short course of vincristine with no neurological complications some five months before any sign of ulnar nerve palsy. The L5/S1 signs were present before chemotherapy and were attributed to degenerative and metastatic changes in the lumbar spine.

Both walking frames were standard, correctly used, and had hard plastic hand-grips that have since been padded. It remains to be seen whether the patients will recover.

We should like to thank Dr V Brezinova and Dr E M Sedgwick of the Wessex Neurological Centre for performing the neurophysiological studies, and Dr H D H Eastwood and Dr V L Hall for permission to report the cases under their care.

¹ Dyck PJ, Thomas PK, Lambert EH. *Peripheral neuropathy*. Philadelphia: W B Saunders, 1975:699-700.

² Noth J, Dietz V, Mauritz KH. Cyclists' palsy. *J Neurol Sci* 1980;**47**: 111-6.

(Accepted 11 June 1982)

Department of Geriatric Medicine, Southampton General Hospital, Southampton SO9 4XY

R I REID, MB, MRCP, senior registrar

Wessex Radiotherapy Centre, Royal South Hants Hospital, Southampton SO9 4PE

M A ASHBY, MB, MRCP, senior house officer