ULNAR NERVE ENTRAPMENT AT THE WRIST

by

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MEDIAN nerve compression at the wrist is common and its treatment by decompression is usually successful. Ulnar nerve entrapment, on the other hand, is uncommon.

The ulnar nerve as it enters the hand passes through and divides in the canal of Guyon, into its deep (motor) and superficial (sensory) branch. This ulnar-carpal tunnel is bounded proximally and medially by the pisiform, distally and laterally by the hook of hamate, anteriorly by the volar carpal ligament and posteriorly by a thick carpal ligament overlying the pisotriquetrial articulation. Proximal to the wrist the ulnar nerve gives off its dorsal cutaneous branch.

Two cases of ulnar nerve entrapment at the wrist are described.

Case 1

A 35-year-old lorry driver complained of clumsiness, burning pain and weakness in his left hand. This had developed over several months and there was no history of injury. The pain affected the ring and little fingers.

Examination revealed wasting of the small muscles of the hand except the thenar muscles, and there was clawing and abduction of the little finger. There was no sensory loss but percussion of the ulnar nerve at the wrist caused tingling in the ring and little finger. Clinical and radiological examination were normal.

A clinical diagnosis of ulnar nerve compression at the wrist was made and this was confirmed by nerve conduction studies, which showed evidence of lower motor neuron denervation in the muscles supplied by the deep branch of the ulnar nerve and that the level of the lesion was at the wrist. Eight weeks later he complained that his hand had become very weak and he experienced altered sensation in his ring and little fingers.

At operation the ulnar nerve was explored in the ulnar carpal canal. Proximal to the bifurcation of the nerve there was a firm swelling. On careful dissection this proved to be a ganglion arising from the thenar aspect of the carpal joint, over which the ulnar nerve was stretched. The ganglion was excised.

There was immediate subjective improvement with relief of pain in the ring and little fingers. Four weeks later there was no sensory deficit and the patient felt his hand was stronger. After seven weeks EMG showed evidence of reinnervation of adductor pollicis with a response in the muscle on stimulation of the ulnar nerve above the wrist.

Case 2

A 48-year-old builder complained that over the course of a day his left little and ring fingers became flexed and he was unable to extend them actively.

Movement returned to the fingers but was followed by a generalised weakness in the hand for fine movements.

When first seen at out-patients there was motor weakness of the ulnar innervated muscles of the hand and marked wasting of the first dorsal interosseous muscle, but no sensory abnormality. There was no history of injury and examination of the rest of the arm was normal. A clinical diagnosis of entrapment of the deep branch of the ulnar nerve was made and confirmed by nerve conduction studies which showed a delay for the wrist latency value of the left ulnar nerve, particularly that of the funiculi to adductor pollicis.

At operation the deep branch of the ulnar nerve was found to be compressed by a tight band which arched over the nerve from the pisiform to the hook of hamate. There was no ganglion.

By the tenth post-operative day the patient noticed that his hand was regaining strength. Nerve conduction studies fourteen weeks after the operation showed a normal EMG picture in the adductor pollicis muscle and the wrist latency for the deep branch of the ulnar nerve had diminished from the previous 7.6m secs. to 4.8m secs. Clinically there was excellent functional recovery.

DISCUSSION

Occupation neuritis of the deep branch of the ulnar nerve was first reported by Hunt (1908). Seddon (1952) described carpal ganglion as a cause of paralysis of the deep branch of the ulnar nerve. The importance of recognising trauma as a precipitating factor in a nerve already being compressed by a ganglion was pointed out by Seddon (1952) and Vanderpool *et al* (1968). Hayes *et al* (1969) described a ligamentous band which bridges over the deep branch of the ulnar nerve from the pisiform to the hook of hamate, so rendering the deep branch more liable to compression by a ganglion. Jeffrey (1971) described compression of the deep branch by an anomalous muscle. McFarland *et al* (1971) reported a case of lipoma causing compression of the motor branch of the ulnar nerve.

Shea and McClain (1969) tabulated 136 cases of ulnar nerve compression at and below the wrist and listed them under 19 causative lesions. Ganglia were present in 39 cases and occupational neuritis in a further 32 cases, these two aetiological factors accounting for 52.2 per cent of the total number tabulated. These authors also introduced a useful classification of the entrapment syndrome. Type 1 causes motor and sensory symptoms due to a lesion proximal to the bifurcation of the ulnar nerve. Normal sensation on the medial side of the dorsum of the hand indicates that the dorsal cutaneous branch of the ulnar nerve is spared. In Type 2 sensation is normal but there is motor weakness due to pressure on the deep branch. In Type 3 there is involvement of the superficial branch alone, with sparing of the motor branch, causing sensory deficit on the volar surface of the hypothenar eminence and in the ring and little fingers.

These two reported cases are examples of Type 1 and Type 2 lesions.

SUMMARY

Two cases of ulnar nerve entrapment at the wrist are described. In the first case the main trunk of the nerve was compressed by a ganglion in the ulnar carpal canal, and in the second, the deep branch was entrapped by a tendinous band as it left the canal. In both cases there was a complete clinical recovery and this was confirmed by post-operative nerve conduction studies.

I wish to thank Mr. J. B. Pyper, F.R.C.S., Consultant Orthopaedic Surgeon, who kindly allowed me to see and operate on one of his patients (Case 2) and Dr. A. K. Irvine, M.R.C.P.I., Consultant in Physical Medicine, who carried out EMG studies for me.

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