PERIPHERAL COMPRESSION LESIONS OF THE ULNAR NERVE

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This paper reviews sixty-one cases of ulnar nerve lesion due to extrinsic pressure encountered in the past eight years and draws attention to the diverse mechanical hazards to which the ulnar nerve is vulnerable. The clinical features that may be of value in diagnosis are described.

ANATOMY

The anatomical course of the ulnar nerve renders it particularly vulnerable at the elbow and at the wrist. At both sites the nerve or its terminal branches pass through narrow canals in which small space-occupying lesions may exert pressure.

As the nerve crosses the elbow it passes from the extensor aspect of the arm into the flexor mass of the forearm, running between the two heads of origin of the flexor carpi ulnaris muscle. The two heads are bridged by an aponeurotic band overlying the nerve and confining it in an elliptical fibro-osseous canal bordered laterally by the elbow joint and its transverse ligament and medially by the flexor carpi ulnaris aponeurosis. With flexion of the elbow this "cubital tunnel" decreases in volume, and the aponeurosis becomes stretched. Eighteen cadaveric specimens were dissected and the distance measured between the ulnar and humeral attachments of the aponeurosis. It was observed that this distance increased by five millimetres for each 45 degrees of flexion, elongation being 40 per cent from full extension to 135 degrees of flexion (Figs. 1 and 2).

The main ulnar trunk and artery enter the hand by passing through a triangular space (Guyon's tunnel) which is bordered medially by the flexor carpi ulnaris tendon and pisiform bone, anteriorly by the anterior carpal ligament and posteriorly by the transverse carpal ligament.
(Fig. 3). Distal to this tunnel, under cover of the palmaris brevis muscle, the nerve divides into superficial and deep branches, the superficial branch continuing under cover of the palmaris brevis muscle to supply it and also to provide cutaneous innervation for the anterior aspect of the hand. The deep palmar branch passes posteriorly lateral to the pisiform bone through a tunnel bounded proximally by the piso-hamate ligament and distally by the arched origins of the abductor, flexor and opponens digiti minimi which arise from the pisiform, tendon of flexor carpi ulnaris and the hook of the hamate. Therefore at the wrist the ulnar nerve is confined in two narrow canals—proximally in Guyon's tunnel, and distally as the deep palmar branch passes between the origins of the small muscles to the little finger and the piso-hamate ligament.

The intraneural arrangement of the nerve bundles is also relevant to a study of the effects of compressive lesions. Figure 4 shows the arrangement of nerve bundles within the ulnar nerve at elbow and wrist levels. It is noteworthy that at the elbow most of the nerve fibres destined for flexor carpi ulnaris and flexor digitorum profundus lie deeply in the nerve, whereas the motor fibres for the intrinsic muscles of the hand and the sensory fibres run more superficially. Theoretically, therefore, compression of the nerve on its superficial aspect would affect the forearm muscles least, whereas compression arising in the floor of the ulnar groove would produce early involvement of the forearm muscles. Similarly at the level of the wrist

![Diagram](image)

**LEVEL OF MEDIAL EPICONDYLE**

- Motor to intrinsic muscles of hand
- Motor to F.C.U.
- Sensory to F.C.U.
- Motor to F.C.U. & F.D.P.
- Sensory to Radial & Ulnar

**LEVEL OF PISIFORM**

- Sensory
- Motor to intrinsic muscles of hand

**FIG. 4**

Intraneural relationships of nerve bundles within the ulnar nerve at elbow and wrist levels (adapted from Sunderland 1945). (F.C.U. = Flexor carpi ulnaris; F.D.P. = Flexor digitorum profundus.)

The motor fibres to the intrinsic muscles are located posteriorly and the sensory fibres anteriorly within the nerve. If the compressing agent presented on the posterior aspect of the nerve, motor weakness might precede sensory loss. However, the varying physiological susceptibilities of motor and sensory nerve fibres also play a role in determining the clinical effects of nerve compression and may override the purely anatomical considerations (Brooks 1963).

**COMPRESSION AT THE ELBOW**

There were forty cases of ulnar nerve compression at the elbow, thirty in men and ten in women, the age range being from seventeen to eighty-one years. Multiple causes of compression frequently combined to involve the nerve. In the largest group of eighteen cases compression by the anatomical confines of the cubital tunnel (cubital tunnel syndrome) alone appeared to be responsible (Fig. 5), but in this group there were five cases with slight
osteoarthritis of the elbow which may have contributed. Of the eighteen cases sixteen occurred in the dominant arm. The significance of elbow flexion in producing this lesion was shown by one patient who developed a nerve lesion during the period of attachment of a pectoral skin flap to the hand. In a further sixteen cases severe osteoarthritis was present. Fifteen of them gave a history of previous injury, the latent period between the injury and the onset of neuritis ranging from ten to fifty years, the average being thirty years. In only five cases was

Figure 5—A typical example of cubital tunnel compression. Beneath the split aponeurosis (held by haemostats) the ulnar nerve is narrowed, while proximal to this point the nerve is thickened (Case 26). Figure 6—Another case. Although the radiographs showed osteoarthritis, at operation the compressing lesion was found to be the pendunculated ganglion shown here which arose from the joint and passed distally with the nerve deep to flexor carpi ulnaris, at which level the nerve was narrowed (Case 15).

A girl of 17, the youngest in the series, had compression of the ulnar nerve by the accessory muscle anconeus epitrochlearis, which arises from the medial border of the olecranon and the tendon of triceps; it crosses over the ulnar nerve as it lies in the ulnar groove and is inserted into the medial epicondyle. Figure 7 shows the muscle overlying the dissector placed in the ulnar groove. In Figure 8 the muscle has been reflected to reveal the constricted and hyperaemic nerve.

the precise nature of the injury identifiable; three had ununited fractures of the medial epicondyle and two of the lateral condyle. In the remainder secondary degenerative change obscured the original injury. In only four of the osteoarthritic cases was the neuritis directly attributable to irregularities of the ulnar groove arising from the marginal osteophytes. Capsular thickening in the medial wall of the cubital tunnel or an increase in the carrying angle (which increases the tension of the aponeurotic roof of the cubital tunnel) appeared to be the significant factor in most cases. In this group also were three cases of ganglia arising
from the osteoarthritic joint capsule compressing the nerve within the cubital tunnel (Fig. 6). There was a further case of ganglion compression at this site without osteoarthritis. Subluxation of the ulnar nerve over the medial epicondyle was the cause of nerve involvement in one case in which osteoarthritis was also present. In two cases the accessory muscle anconeus epitrochlearis (Le Double 1897) which bridges the ulnar groove was the cause of compression (Figs. 7 and 8). In three cases no pathology was recognised at operation but all recovered after simple decompression of the cubital tunnel and these may have been examples of the cubital tunnel syndrome.

**CLINICAL FEATURES**

The clinical presentation showed a striking uniformity. Paraesthesiae within the distribution of the ulnar nerve was usually the earliest complaint and sometimes preceded subjective motor weakness by months or years. Objective motor weakness and wasting of the intrinsic muscles were usually more marked than the sensory changes and often had developed earlier than the patient had realised.

The sensory symptoms were rarely distressing or disabling and lacked the painful nocturnal character of median nerve compression. The exact distribution of the sensory changes was of great significance, for if the dorsal cutaneous branch of the nerve was involved, this clearly established a proximal lesion of the nerve, whereas if it was spared the compression was more likely to be at the wrist.

The long flexor muscles, flexor carpi ulnaris and flexor digitorum profundus, were unaffected in twenty-four of the forty cases of ulnar nerve compression at the elbow, usually when the major compressing agent was the superficial aponeurosis. When the source of compression lay deep to the nerve, these muscles were usually involved, a fact which may be explained by the intraneural arrangement of the nerve bundles (Fig. 4).

In one case of cubital tunnel syndrome motor weakness was restricted to the distribution of the deep palmar branch, the hypothenar and the long flexor muscles being spared, but the associated sensory disturbance enabled the correct diagnosis of a lesion at the elbow to be made before operation.

**TREATMENT**

When the cause of the compression was in the cubital tunnel alone, treatment consisted of excising the aponeurotic roof of the tunnel, and this simple procedure was also used when mild osteoarthritis existed with cubital tunnel compression. Compression by a ganglion or an accessory muscle was relieved by their excision. Anterior transposition was reserved for the cases with more severe osteoarthritis and for the case of recurrent subluxation of the nerve.

**RESULTS**

Rapid subjective improvement was noticed in most cases, often immediately after operation. Objective motor and sensory recovery usually occurred in a few months after the operation, the longest period before recovery being eighteen months. Thirty-six cases with history of symptoms from two months to seven years before operation showed worthwhile recovery; only two had no benefit from the operation but two other patients were untraced. A number of cases with complete ulnar paralysis and sensory loss known to have been present for many years were not included in this series because there was little chance of useful recovery with operation.

**ILLUSTRATIVE CASES**

**Cubital tunnel compression associated with mild osteoarthritis**

**Case 26**—A fifty-seven-year-old joiner gave no history of injury to his elbow. One year before admission he experienced gradual onset of paraesthesiae in the ulnar distribution of his right hand. Nine months before admission he had noted progressive weakness of his hand and loss of fine movements. On examination he had marked weakness and wasting of the interossei although the flexor carpi ulnaris

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appeared normal. There was reduced pinprick sensation in the ulnar distribution of his hand. Radiographs showed mild osteoarthrosis of the elbow. At operation compression by flexor carpi ulnaris aponeurosis was found and released (Fig. 5). Immediately after operation the paraesthesiae had disappeared and within four months he was neurologically normal.

**Ganglion compression of ulnar nerve at elbow**

Case 15—A sixty-eight-year-old male gardener had for one year before admission noted weakness and wasting of the left hand with no sensory symptoms. He gave no history of trauma. For the same period he had suffered occasional pain in the left elbow. On physical examination there was evidence of clawing of the hand with weakness and wasting of the musculature innervated by the ulnar nerve. There was decreased pinprick sensation in the ulnar distribution of the hand. A 20 degrees flexion contracture was present at the elbow. Radiographs of the elbow showed osteoarthritic changes. At operation a ganglion one centimetre in diameter was found arising from the elbow joint, passing distally with the ulnar nerve compressing it against the aponeurosis of the flexor carpi ulnaris (Fig. 6). The ganglion was excised and the aponeurosis removed. After five months sensation was normal. At one year the hypothenar muscles had recovered but the remaining intrinsic muscles were still paralysed.

Case 14—A sixty-four-year-old housewife noticed swelling of her right elbow one year before admission. There was no history of injury. Approximately five months before admission she had noted weakness of the grip and paraesthesia in the fourth and fifth fingers of the right hand. Examination revealed weakness of all the ulnar innervated musculature with decreased pinprick sensation in the ulnar distribution of her fingers and palm. The elbow lacked 15 degrees of extension and radiographs showed osteoarthrosis. There was a palpable mass in the line of the ulnar nerve. At operation an elongated ganglion eight centimetres in length was found arising from the joint which extended proximally and distally in intimate association with the ulnar nerve. The ganglion was removed and the nerve was not transposed. She had complete recovery after eight months.

**COMPRESSION AT THE WRIST**

Twenty-one cases of ulnar nerve compression occurred at the wrist. As with the lesions located at the elbow the compressing agent usually combined with the anatomical fixation of the nerve at the wrist to produce its effect. The following conditions were encountered: ganglia (thirteen), occupational trauma (three), acute trauma (two), narrowed canal of deep palmar branch (one), osteoarthritis of inferior radio-ulnar joint (one), accessory ossicle at wrist (one).

**CLINICAL FEATURES**

Ganglia produced the largest number of lesions at this level and were divided into proximal, arising near the radio-ulnar or piso-triquetral joints, and distal, arising from the distal carpus deeply in the palm.
The seven proximal ganglia, sometimes palpable, were associated with both motor and sensory symptoms, although the clinical picture varied, depending upon the exact relationship between the nerve and the ganglion; thus either motor or sensory symptoms and signs predominated in individual cases (Fig. 9). The sensory disturbance spared the dorsal cutaneous branch which arises more proximally in the forearm. The motor involvement affected the hypothenar as well as the interosseous muscles and clawing of the medial two fingers was usually present.

The five distal ganglia were all impalpable. They exerted pressure only on the deep branch as it passed through the narrow tunnel into the deeper layers of the palm, producing a pure motor weakness of this branch but sparing the hypothenar muscles and the sensory nerves (Fig. 10). Clawing of the fingers occurred only once in this group and was restricted to the ring finger. Several showed an abducted position of the fifth finger from the unopposed action of the abductor of the little finger (Fig. 11). One case showed first the clinical features of a distal ganglion and later of a proximal lesion because of the rapid enlargement and migration of the ganglion.

Two cases of carpal ganglia were associated with osteoarthritis of the wrist. Several of the cases with ganglion compression gave a history of injury to the base of the hypothenar area including one pneumatic drill operator. Three other cases of occupational injury were seen; they were caused by carpentry, the use of a vibrating buffer and of a floor polisher. These cases were not explored, and cleared up after cessation of the activity concerned.

Two other cases were seen after acute injury; one case each was seen with a narrowed deep palmar branch canal, with osteoarthritis of the distal radio-ulnar joint and with compression by an accessory carpal ossicle (Figs. 12 and 13).

### Treatment and Results

Exploration was done on all cases, except those in which there was a clear history of acute or chronic trauma, which were observed initially for a period of four to six weeks and operated upon only if recovery did not take place during this period.

After removal of the compressing agent recovery took place within a few months in all but one case.
ILLUSTRATIVE CASES

Compression by a proximal ganglion with sensory and motor involvement

Case 42—A woman of forty-three years complained of weakness of the ring and little fingers with inability to extend them fully, associated with local tenderness over the pisiform bone of one month's duration. A "stinging" pain was felt in the ring and little finger-tips and the grip was markedly impaired, the patient being unable to hold objects firmly. The little finger tended to "stick out" in the abducted position when washing the hands. Examination revealed the typical main-en-griffe deformity due to ulnar nerve palsy with severe sensory loss in the ulnar cutaneous distribution and local tenderness on pressure over the pisiform bone. Severe intrinsic and hypothenar muscle wasting was evident. Radiographs of the cervical spine and wrist were normal. At operation the right ulnar nerve was exposed at the wrist and found to be compressed by a large underlying ganglion adjacent to the pisiform-triquetral joint (Fig. 9). Immediate relief of pain occurred after removing the ganglion and she returned to work as a darnier ten weeks later. Three months later motor recovery was almost complete.

Compression by distal ganglion producing a deep palmar branch lesion

Case 52—A fifty-four-year-old electrician struck the palm of his left hand while pulling a heavy cable four months before admission, with the onset of pain in the hypothenar area. There was gradual decrease of pain over the ensuing weeks but subsequently he noted weakness in his grip. Physical examination revealed weakness and atrophy of the intrinsic muscles of the hand with the hypothenar muscles being spared. Sensation was intact. There was no palpable mass and radiographs of the hand, elbow and neck were normal. At operation a ganglion measuring 1.5 centimetres in diameter was removed from the carpus. It was found to be pressing on the deep palmar branch just distal to its passage beneath the origins of the small muscles to the little finger (Fig. 10). The ganglion was excised and three months later neurological examination was normal. Such cases illustrate the need of surgical exploration even when occupational injury seems to be the cause, if symptoms do not rapidly subside after a change of occupation.

Compression of the ulnar nerve by accessory ossicle at wrist

Case 60—A thirty-three-year-old clerk had been aware of a swelling in the ulnar border of her left wrist for twenty years, which she had first noticed after a "sprain." Three months before coming to hospital she noticed weakness and wasting of the hand. Examination revealed a hard mobile bone-like swelling three by two centimetres in diameter just distal to the pisiform. There was complete ulnar intrinsic paralysis and hypoesthesia. Clawing of the median two fingers was present. Radiographs confirmed the swelling to be bone (Fig. 12) and at operation it was found to be loosely connected to soft tissues lateral to the piso-hamate ligament overlying and compressing the ulnar nerve at that level (Fig. 13). The ossicle lay outside the wrist joint and was not within muscle. Its origin was quite obscure.

DISCUSSION

In recent years involvement of most large peripheral nerves from mechanical compression in a narrow anatomical canal has now been reported.

Some lesions such as median nerve compression within the carpal tunnel are common and well known. Mechanical neuritis of the ulnar nerve, historically one of the first to be recognised, is much less common and more likely to escape recognition because of the more insidious and less compelling character of its symptoms. Although ulnar nerve damage has been described at most levels in the upper extremity in association with fractures or other injuries, such lesions present little problem in diagnosis because of their close association with injury and these have not been included in this series.

Of the sixty-one cases of more insidious onset reported here, forty occurred at the elbow joint level and twenty-one at the wrist. The anatomical relationships discussed in this paper were important factors in the production of the neuritis, whatever additional pathology might be present.

Ulnar nerve damage at the elbow—The first type of mechanical ulnar neuritis to be recognised was the tardy palsy following fracture or osteoarthritis of the elbow. Panas (1878) reported three cases of slowly progressive ulnar nerve lesions associated with bony abnormality at the elbow (old fracture, osteoarthritis, shallow ulnar groove). He believed the pathogenesis was repeated trauma causing hyperaemia, oedema and eventually infiltration of fibrous tissue.
Mouchet (1898) reported seven cases following fracture of the lateral humeral condyle in childhood with subsequent cubitus valgus and progressive ulnar palsy after a latent period of several years. He considered that traction produced interstitial neuritis and a fusiform neuroma at the elbow.

By the early 1920's three main pathological factors were recognised: 1) increased valgus deformity after injury, leading to traction neuritis as described by Mouchet (1898), Brickner (1924) and Miller (1924); 2) irregularity of the ulnar groove from osteoarthritis without previous trauma, or injury to the medial epicondyle causing a friction neuritis (Shelden 1921); 3) subluxation of the ulnar nerve in its groove due to lax or weak ligament either congenital or acquired, or a shallow groove causing ulnar palsy by repetitive motion of the nerve over the medial epicondyle (Platt 1926). Other mechanisms were occasionally described, including scars in the region of the medial epicondyle, occupational injuries, ganglia, a variety of tumours and accessory muscles.

Recently the identification of the "cubital tunnel syndrome" (Osborne 1957, Feindel and Stratford 1958) has caused a reappraisal of the traditional theories of mechanical injury to the ulnar nerve. In the cubital tunnel syndrome the nerve is compressed between the aponeurosis joining the heads of origin of flexor carpi ulnaris and the floor of the tunnel formed by the medial ligament of the elbow joint, especially the transverse ligament. During flexion the space of the tunnel is diminished both by tightening of the aponeurosis and bulging of the joint. This was the primary pathology in nearly half of the lesions at the elbow reported in this paper.

The cubital tunnel syndrome presents more commonly in men, usually affects the dominant hand and seldom causes pain or nocturnal distress in contrast to the carpal tunnel syndrome. These differences may be attributed to the increased importance of occupational activity in the production of cubital tunnel syndrome, whereas fluid retention during pregnancy and the menopause may be operative in the carpal tunnel syndrome. Osteoarthritis of the elbow or deformity such as cubitus valgus or fracture of the medial epicondyle could all reduce the space available in the cubital tunnel, and many of the cases described in this paper have shown how often these factors combine to produce the nerve damage. Rheumatoid arthritis may operate in the same way (Pulkki and Vainio 1962) but this condition was not encountered in this series.

The experience reported in this paper suggests that anterior transposition of the ulnar nerve may be applied more selectively than has sometimes been recommended, as the lesser procedure of local excision of the aponeurosis has resulted in recovery of nerve function in this series even in the presence of osteoarthritis of the joint. When there is clear evidence of narrowing of the nerve at its entrance to the cubital tunnel local decompression only is required whatever the associated pathology.

Recurrent subluxation of the ulnar nerve has assumed less significance since the observation that it occurs in 16 per cent of the population (Childress 1956) and rarely causes symptoms unless there is some additional factor such as trauma. This series supports this concept, for in only one case was recurrent subluxation regarded as the main cause of neuritis. An additional symptomatic case of subluxation developed after an earlier extensive mobilisation of the ulnar nerve for cubital tunnel syndrome, and since this experience we have not deroofed the ulnar groove when local decompression in the cubital tunnel has demonstrated a significant lesion. If more extensive exploration of the nerve is for any reason found necessary then a formal anterior transposition should be carried out.

Ulnar nerve lesions at the wrist—Since Brooks (1952) and Seddon (1952) emphasised the importance of ganglia as compressing agents at the wrist, some thirty-five cases have now been described including the eleven reported in this paper (Jenkins 1952; Boyes 1955; Mallett and Zilkha 1955; Blunden 1958; Ebeling, Gilliatt and Thomas 1960; Toshima and Kimata 1961; Richmond 1963; Dupont, Cloutier, Prévost and Dion 1965).
As noted by Brooks (1963), the symptoms may vary according to the exact location of the ganglion in relation to the nerve and according to the degree of compression. These lesions tend to fall into two general patterns: 1) ganglion lying proximal to the piso-hamate ligament with both sensory and motor involvement; and 2) ganglion arising deeply in the palm distal to the piso-hamate ligament with affection of the interossei but sparing of sensation and of the hypothenar muscles.

Occupational neuritis of the deep branch of the ulnar nerve may occur from prolonged repetitive pressure on the hypothenar aspect of the palm. From 1908 to 1914 Hunt reported the first seven cases in the English literature, and many others have since been recognised (Harris 1929, Worster-Drought 1929, Russell and Whitty 1947, Bakke and Wolff 1948, Magee 1955, Moffat 1964). It is important to recognise trauma as an additional insult to a nerve already threatened by a ganglion as confirmed in two cases in this series and in four cases recorded by Seddon (1952). Any case which does not recover within a few weeks of injury deserves exploration.

Osteoarthritis is occasionally the cause of compression at the wrist. One case is reported of involvement of the ulnar nerve with osteoarthritis of the pisiform-triquetral joint (Jenkins 1951) and one case of osteoarthritis of the inferior radio-ulnar joint is also contained in this series. Occasional cases have been described with scarring of this area involving the ulnar nerve, and they tend to occur within weeks or months of an injury (Cameron 1954, Howard 1961, Stein and Morgan 1962, Dupont et al. 1965, Fenning 1965).

Differential Diagnosis

Although various neurological affections of the cord or of the brachial plexus may suggest ulnar nerve involvement, a careful clinical examination will usually demonstrate that the paralysis or sensory disturbance in these conditions does not exactly conform with the peripheral distribution of the ulnar nerve. Radiographs of the cervical spine and lung apices as well as of the elbow and the wrist are advisable before reaching the diagnosis of peripheral compression syndrome.

Delay in nerve conduction has been found of value by others (Simpson 1956, Gilliatt and Sears 1958, Ebeling et al. 1960, Gilliatt and Thomas 1960) in establishing the site of nerve involvement and although no difficulty in this series was encountered in establishing the correct level by routine clinical examination alone, this procedure may prove useful if there is any doubt.

Summary

1. Sixty-one cases of compression of the ulnar nerve are reported, forty at the elbow and twenty-one at the wrist. Although contributory factors may include deformity, osteoarthritis, injury, ganglia and other tumours, the narrow anatomical confines of the nerve at these two levels are noteworthy and alone may produce nerve compression.
2. Careful clinical examination will usually determine the level of involvement if not the exact pathology. Surgical exploration is indicated both as a diagnostic and therapeutic procedure in most cases.
3. Following removal of the compressing agent rapid recovery occurred in most cases.

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