Palsy of the anterior interosseous nerve (AIN) was first described by Tinel in 1918 under the title 'Dissociated paralysis of the median nerve'. In 1948 Parsonage and Turner noted six examples of this syndrome in a review of 1.36 patients with neuralgic amyotrophy, and in 1952 Kiloh and Nevin reported two cases of the palsy as an isolated neuritis. In 1955 Lipscomb and Burleson described the condition in association with a supracondylar fracture and in 1965 Fearn and Goodfellow first observed that an entrapment neuropathy was responsible for some examples of the AIN.

The indications for operative treatment for spontaneous AIN palsy are controversial. When the cause of the palsy is an entrapment, the nerve should be explored early. If it is due to neuralgic amyotrophy or an isolated neuritis, surgery is not generally indicated and conservative treatment is followed. However, it is not easy to determine the appropriate method of management because there are no clinical findings or neurophysiological investigations to differentiate these two lesions. Recently, there have been several reports of cases in which an hourglass-like fascicular constriction was discovered in the main trunk of the median nerve after interfascicular neurolysis. The aetiology and the strategy for treating AIN palsy should therefore be reconsidered.

This review describes the anatomy, clinical features, the aetiology and the treatment of an hourglass-like constriction of the fascicles in spontaneous palsy.

Anatomy

The anterior interosseous nerve is the largest branch of the median nerve and arises 5 to 8 cm distal to the level of the lateral epicondyle, usually immediately distal to the superficial head of pronator teres. It was found to originate from the radial side of the median nerve in 61% of 31 cadaver arms and from the deep, posterior aspect in the remainder. It runs between the deep and superficial heads of pronator teres accompanying the median nerve and passes beneath the arcade of flexor digitorum superficialis to lie on the anterior interosseous membrane, terminating in the capsule of the wrist. Proximally, above its branching from the median nerve, the fasciculus destined to become the anterior interosseous nerve runs in the posterior part of the main trunk of the median nerve. According to Sunderland's detailed anatomical studies of the median nerve, fibres destined to become the anterior interosseous nerve can be isolated proximally for more than 10 cm, with some communicating fibres to the main trunk.

The anterior interosseous nerve supplies flexor pollicis longus (FPL), flexor digitorum profundus to the index (FDP1), flexor digitorum profundus to the middle finger (FDP2), and pronator quadratus. Sunderland stated that it was rare for FDP1 to be innervated other than by the median nerve, but the supply to the middle finger was variable. In 15% of limbs there may be a Marin-Gruber anastomosis between the ulnar and median or anterior interosseous nerves. There is no superficial sensory branch.

Clinical features

The incidence of the palsy is low and accounts for less than 1% of all compression syndromes in the upper limb. Between 1986 and 1990, we saw only 11 such patients out of 1011 with peripheral nerve palsy, but the lesion is now being diagnosed more commonly. In the ten subsequent years, 43 patients were referred to our clinic with the disorder which is four times as many as in the previous five years. This change is probably a result of the increased awareness of the condition by orthopaedic surgeons. Occasionally, bilateral cases are seen, and recurrent palsy has been reported.

Werner summarised the clinical features of 69 patients reported in the literature before 1985 and included four of his own. There were 38 males and 31 females with a mean age of 37.5 years (9 to 72). The right side was affected in 45 and the left in 24. Schantz and Riegels-Nielsen described
Pain is a common feature of anterior interosseous nerve entrapment neuropathy, and/or often preceding the motor symptoms. Neuritis from local trauma...surgery, or other manifestations of muscular-skeletal or systemic diseases were often recorded immediately preceding the onset of paralysis. Patients usually experienced pain in the region of the elbow before the onset of the palsy. Miller-Breslow, Terrono and Millender followed ten limbs in nine patients; all described an acute spontaneous painful episode lasting for a mean of 11 days. Seror reported that pain was recorded in 85% of the 117 cases which were collected from the literature and in ten of his 14 patients. In the experience of the author, 39 of 43 patients (99%) had pain at onset; eight complained of pain from the shoulder girdle to the elbow, four in the upper arm, three in the upper arm and forearm, 20 in the elbow and four in the forearm. Rask stated that pain may be the earliest symptom of this entrapment neuropathy. However, Wong and Dellow reported that the important point in the history for distinguishing a bursal neuritis from local compression is pain in the upper arm, elbow, and/or forearm often preceding the motor symptoms. Pain is a common feature of anterior interosseous nerve palsy, but it is not a predictive sign for differentiation from an inflammatory origin.

The typical symptom of the palsy is the inability to form an 'O' with the thumb and index finger (Fig. 1). Since and FDP1 are paralysed, the patient is unable to flex the interphalangeal joint of the thumb and the distal interphalangeal joint of the index finger. Pronator quadratus, also paralysed, but its weakness is not noticed by the patient, and manual muscle testing of this muscle is difficult to judge correctly, even when carried out with the elbow acute flexion.

The FPL and FDP1 are not always paralysed simultaneously. Werner reported that both were paralysed in patients, the FPL only in 25 and the FDP1 only in ten. Sood and Burke's report of 16 patients, only FPL was paralysed in five and FDP1 in two; none had paralysis of FII. In the author's series, both FPL and FDP1 were paralysed in 19 patients, only FPL in 11 and only FDP1 in nine. In patients both FPL and FDP1 were active but weak. Patients had weakness of FDP2. Sometimes the m. pronator teres is paralysed, but its weakness is not noticed by the patient, and manual muscle testing of this muscle is difficult to judge correctly, even when carried out with the elbow acute flexion.

The reported causes are listed in Table I and are divided into two categories, traumatic and non-traumatic/spontaneous. Palsy of the anterior interosseous nerve has b described in association with neuralgic amyotrophy, isolated neuritis, and entrapment neuropathy. The nerve is susceptible to entrapment by soft tissue and by vascular bony structures. According to Spinner, it is vulnerable to injury or compression by the following:

(i) a tendinous origin of the deep head of pronator teres

![Fig. 1](image-url)

Photograph showing the inability to form an 'O' with the thumb and the index finger in palsy of the left anterior interosseous nerve.

Table 1. The aetiology of anterior interosseous nerve palsy

<table>
<thead>
<tr>
<th>Category</th>
<th>Examples</th>
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<tbody>
<tr>
<td>Traumatic</td>
<td>Penetrating injuries</td>
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<tr>
<td></td>
<td>Fracture</td>
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<td></td>
<td>Supracondylar fracture of the humerus</td>
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<td></td>
<td>Forearm fractures</td>
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<td></td>
<td>Venepuncture</td>
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<td></td>
<td>Cast fixation</td>
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<td></td>
<td>Open reduction and fixation of fractures</td>
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<tr>
<td>Spontaneous</td>
<td>Entrapment neuropathy</td>
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<td></td>
<td>Muscular and fibrous abnormalities</td>
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<tr>
<td></td>
<td>Gantzer's muscle</td>
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<td>Enlarged bicipital bursa</td>
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<td></td>
<td>Vascular abnormalities</td>
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<td></td>
<td>Volkmann's ischaemic contracture</td>
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<tr>
<td></td>
<td>Neuralgic amyotrophy</td>
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<tr>
<td></td>
<td>Isolated neuritis</td>
</tr>
<tr>
<td></td>
<td>Unknown</td>
</tr>
</tbody>
</table>

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(ii) a tendinous origin of flexor digitorum sublimis to the middle finger;
(iii) thrombosis of the ulnar collateral vessels which cross it;
(iv) accessory muscles and tendons from flexor digitorum sublimis;
(v) an accessory head of FPL (Gantzer's muscle);  
(vi) an aberrant radial artery;
(vii) a tendinous origin of palmaris longus or flexor carpi radialis brevis; and
(viii) an enlarged bicipital bursa.

Collins and Weber 29 considered entrapment to be by far the most common cause and Hill, Howard and Huffer 30 observed it in 24 of 28 cases of incomplete palsy. Schantz and Riegels-Nielson 18 found evidence of nerve compression in nine of 15 patients. Werner 17 reported that fibrous bands within pronator teres were seen in 52 patients, but that an indentation in the nerve or neuromata were found in 14. In the eight cases which were studied by Sood and Burke, 15 there was no demonstrable abnormality in four and a further two patients were deemed to have anatomical structures with a potential for causing compression but with no evidence of any abnormality of the nerve itself. Between 1969 and 1985, the author encountered 31 cases of non-traumatic palsy and performed an exploratory operation in ten. 23 The operative findings showed that the palsy was due to compression of the nerve by a fibrous band in one patient, while in the other nine the nerve was slightly swollen, scarred, hardened, or even normal. During the last ten years, I have explored the nerve in a further 23 patients. Four median nerves showed slight swelling, hardening, or adhesion to the surrounding tissue at the elbow, but the other 19 were normal in appearance with no entrapment. Fearn and Goodfellow 28 described a case of entrapment neuropathy resulting from a crescentic fibrous band. The illustration in their paper showed a band which compressed both the median and anterior interosseous nerves. Generally, the latter runs in a posteromedial direction and is located deeper than the median nerve. However, the patients of Fearn and Goodfellow 5 had only anterior interosseous nerve palsy without motor and sensory disturbance of the median nerve, which raises the question as to why it alone was involved.

Vichare 31 also found it difficult to explain why a band should involve the anterior interosseous nerve alone, leaving the adjacent and larger median nerve unaffected. Fearn and Goodfellow 28 suggested that it is necessary - to be wary of ascribing paralysis in the distribution of a peripheral nerve to a hypothetical 'neuritis' and that the search for a mechanical cause may be rewarding. The converse is also true, since the cause should not necessarily be attributed to entrapment even if there is a fibrous band on the nerve since an hourglass-like constriction is very often seen in the fascicles destined to the anterior interosseous nerve in the median nerve above the elbow. This will be discussed later.

Overall, we conclude that entrapment neuropathy is one of the causes of this palsy, but its incidence is low and the most common cause is a so-called neuritis or neuralgic amyotrophy.

Treatment

The recognition of an anatomical cause for the problem has initiated a debate about the management of this palsy. Theoretically, conservative treatment is recommended for neuralgic amyotrophy and isolated neuritis, whereas decompression is advised for the AIN syndrome. However, there are no clinical signs and symptoms to differentiate these two lesions, in the specialty literature for orthopaedics, neurosurgery, hand surgery and plastic surgery, 46 of 100 reported cases (46%) were explored surgically, but of those reported in the neurology journals, only four of 32 patients (12.5%) underwent surgical exploration. Although the results of both types of treatment were almost the same. 19

Spinder 32 reported that patients who have spontaneous paralysis of the AIN should initially be treated conservatively, because many have a satisfactory return of function and no recurrence, but if there are no signs of clinical or electromyographical improvement in six to eight weeks, exploration is indicated. Nigst and Dick 12 recommended operative treatment in patients in whom there was no perceptible improvement after conservative treatment for eight weeks, since surgical decompression reduces the time needed for recovery. Hill et al. 26 recommended that exploration and external neurolysis be undertaken when there is no clinical and/or electromyographical improvement by 12 weeks after onset.

However, several authors advise conservative treatment. Sero 24 concluded that surgery should not be considered for a year because late spontaneous recovery is sometimes seen after this time. Futami et al. 32 stated that conservative treatment is advisable in most cases because useful recovery can be expected within ten months on average. Surgical intervention may be required only in rare cases which do not respond to conservative treatment after more than two years. Miller-Breslow et al. 20 treated ten patients and believed the condition to be a neuritis. They concluded that surgical decompression may not hasten recovery. Tsukahara et al. 33 treated 12 hands from 11 patients conservatively. All paralyzed muscles recovered to more than MRC grade 3, but it took a long time for adequate recovery when signs of improvement were not detected within six months after onset. Sood and Burke 15 explored eight patients and obtained good results in seven and a poor outcome in one. They also treated 11 patients conservatively, with good results in eight, a fair result in one and poor results in two. A comparison of the results of operative and non-operative treatment revealed a similar outcome and they therefore concluded that it is likely that the condition results from a multifocal neuritis, which often resolves spontaneously. Nakano et al. 13 described two patients who presented with a bilateral palsy with separate times of onset for each side. The first side was treated surgically, and the second con-
The centre of the photograph is the elbow flexion crease, and the left side is the arm. The left median nerve was slightly adhered to the surrounding tissues, but there was no compression on the nerve.

Two hourglass-like fascicular constrictions were revealed in one fascicle 0 and 20 mm above the elbow flexion crease after interfascicular neurectomy in 23 patients who did not show any recovery three months after onset and who agreed to surgical exploration. There were 12 men and 11 women, with a mean age of 43.2 years (23 to 64). The mean interval between onset and the operation was 5.5 months (3 to 18). No patient showed any clear physical cause such as manual work or sports activity. The median and anterior interosseous nerves were explored from the proximal one-third of the forearm to 5 to 10 cm above the elbow using an operating microscope. No external compression was found anywhere along the course of the nerves. By interfascicular neurolysis, an hourglass-like fascicular constriction (Fig. 2a and 3) was discovered in the fascicles of the anterior interosseous nerve within the median nerve between 2 to 7.5 cm above the elbow in 22 patients. The lesion was located above the elbow, a site which had not usually been explored in previous cases. This constriction had been reported only five patients in the literature. However, we found this lesion in almost all cases of palsy which did not have evidence of external compression. All except one patient had pain in the elbow, and paralysis was evident between one and 42 days after the onset of pain. In some cases, the cause had previously been attributed to isolated neuritis when there were no findings of entrapment neuropathy at exploration, but our findings suggest that the basic abnormality is this hourglass-like fascicular constriction.

In 21 of 22 patients the lesion was treated only by interfascicular neurolysis and all regained good function. However, we do not know whether this recovery was spontaneous or due to the neurolysis. We compared the results of 15 patients who had had interfascicular neurolysis and those of 11 with conservative treatment who were followed for more than two years. There were no significant differences in age, gender, the affected side, or the period from onset between the two groups. All patients who had had an interfascicular neurolysis obtained more than grade 3 power in flexor pollicis longus and/or flexor digitorum
rum profundus, but recovery did not occur in two patients treated conservatively. The muscle power after interfascicular neurolysis was significantly better than after conservative treatment at more than 24 months from onset, but there were no differences in the time from onset to recovery in the two groups. It seems that nerve regeneration can be expected without interfascicular neurolysis, but after this operation more fibres will regenerate. It is therefore recommended that exploration of the nerve be offered to patients who do not show any signs of recovery by three months after onset. External neurolysis alone is not adequate and interfascicular neurolysis should be performed to detect any lesion. However, this is a small retrospective study and a prospective, randomised investigation is required to establish a sound conclusion.

Regardless of the cause and management of the palsy, if motor function does not recover, tendon transfers will restore function satisfactorily. The brachioradialis is a good substitute for restoring flexion of the interphalangeal joint of the thumb. The transfer of the tendon of flexor digitorum profundus of the ring or middle finger to that of the index finger at the wrist can provide satisfactory flexion of the distal phalanx of the index finger. Schantz and Riegels-Nielson recommend delay in the use of tendon transfer until one year after the onset of palsy.

### Hourglass-like fascicular constriction

This condition was first reported in palsy of the posterior interosseous nerve. It was first described in the anterior interosseous nerve by Englert in 1976 and again by Haussmann and Kendel in 1981, Nakamura et al in 1991 and Nagano et al in 1996.

The aetiology remains unknown. Haussmann et al and Nakamura et al have suggested that it may be the result of mechanical torsion by rolling of the fascicles during flexion-extension of the elbow or pronation-supination of the forearm. Hosi et al felt that the constriction did not have a mechanical origin, because in their patient one lesion was found in each of two fascicles, which could not be explained by pronation-supination of the forearm. They suggested that the lesion may be due to an inflammatory response after infection or an autoimmune response. In our series, the constriction was seen in the fascicles of the main trunk of the median nerve and two or more constrictions coexisted. We also encountered a patient who had a constriction in the anterior interosseous nerve and in other motor branches of the median nerve. Another had palsies of both the anterior and posterior interosseous nerves simultaneously and both nerves showed this constriction.

We consider that the lesion has a different mechanical basis and suggest that the initial cause is an inflammation of the nerve, producing oedema and consequent adhesions in the fasciculus. The subsequent traction force produced by flexion and extension of the elbow pulls more strongly on the fasciculus forming the anterior interosseous nerve than on those which constitute the main trunk of the median nerve, since the traction force is thought to be stronger on the shorter segment, causing the fascicles to become constricted. Tazaki et al performed an experimental study on the median nerve of the rabbit. After the injection of saline into the fasciculus, the nerve was swollen, lost its flexibility and kinked sharply upon passive elbow flexion. Repeated saline injection and movement of the elbow caused local torsion at the kinking point in the fasciculus. They concluded that the cause of fascicular constrictive neuropathy may be oedema and consequent loss of flexibility of the fasciculus with movement of the elbow. Further study is necessary to clarify the pathogenesis of this constriction.

For treatment Haussmann and Nakamura resected the constriction and performed nerve grafting. We have also carried out nerve grafting in one patient because the constriction was so severe that the fasciculus appeared to be completely ruptured. However, recovery after interfascicular neurolysis has generally been good. Therefore, we recommend only interfascicular neurolysis, and believe that nerve grafting is unnecessary.

### References