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 136 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 superior border of 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 from the main trunk of the median nerve as far proximal as the brachial plexus, and clinical experience has shown that the fasciculus 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) and occasionally 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 clinical experience has shown that the fasciculus can be isolated proximally for more than 10 cm, with some communicating fibres to the main trunk.

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 increase 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
21 cases in 20 patients, of whom 14 were male and six were female, with 15 affected on the right and six the left side. Sood and Burke\textsuperscript{15} reported 16 patients, nine men and seven women, for whom the age at presentation ranged from 32 to 75 years. The author of this review encountered 43 patients between 1992 and 2002. There were 24 males and 19 females, with the right side affected in 20 and the left in 23. The mean age at onset was 42.7 years (14 to 73). From these observations, it is clear that there were no significant differences in gender or in the side affected and that most patients were between 30 and 60 years of age.

Possible predisposing factors such as an influenza-like illness, venepuncture for major abdominal surgery, a history of trauma, inoculations, or other manifestations of musculoskeletal or systemic diseases were often recorded immediately preceding the onset of paralysis.\textsuperscript{14,15,19}

Patients usually experienced pain in the region of the elbow before the onset of the palsy. Miller-Breslow, Terrono and Millender\textsuperscript{20} followed ten limbs in nine patients; all described an acute spontaneous painful episode lasting for a mean of 11 days. Seror\textsuperscript{21} 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 (89\%) 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\textsuperscript{22} stated that pain may be the earliest symptom of this entrapment neuropathy. However, Wong and Dellon\textsuperscript{19} reported that the important point in the history for distinguishing a brachial 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 differentiating an inflammatory from a mechanical origin.

The typical symptom of the palsy is the inability to form an ‘O’ with the thumb and index finger (Fig. 1). Since FPL and FDP1 are paralysed, the patient is not able to flex the interphalangeal joint of the thumb and the distal interphalangeal joint of the index finger. Pronator quadratus is 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 in acute flexion.

The FPL and FDP1 are not always paralysed simultaneously. Werner\textsuperscript{17} reported that both were paralysed in 34 patients, the FPL only in 25 and the FDP1 only in ten. In Sood and Burke’s report\textsuperscript{15} of 16 patients, only FPL was paralysed in five and FDP1 in two; none had paralysis of FDP2. In the author’s series, both FPL and FDP1 were paralysed in 19 patients, only FPL in 11 and only FDP1 in nine. In four patients both FPL and FDP1 were active but weak. Eight patients had weakness of FDP2. Sometimes the motor branches to pronator teres, flexor carpi radialis and/or palmaris longus are also involved.\textsuperscript{23,24} In my 43 patients, pronator teres was paralysed in 12, flexor carpi radialis in 11, and palmaris longus in 12.

\textbf{Aetiology}

The reported causes are listed in Table I and are divided into two categories, traumatic and non-traumatic/spontaneous.\textsuperscript{25,26}

Palsy of the anterior interosseous nerve has been described in association with neuralgic amyotrophy,\textsuperscript{2,27} isolated neuritis,\textsuperscript{2} and entrapment neuropathy.\textsuperscript{4} The nerve is susceptible to entrapment by soft tissue and by vascular and bony structures. According to Spinner,\textsuperscript{28} it is vulnerable to injury or compression by the following:

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

\begin{table}[h]
\centering
\caption{The aetiology of anterior interosseous nerve palsy}
\begin{tabular}{|l|}
\hline
Traumatic \\
Penetrating injuries \\
Fracture \\
Supracondylar fracture of the humerus \\
Forearm fractures \\
Venepuncture \\
Cast fixation \\
Open reduction and fixation of fractures \\
Spontaneous \\
Entrapment neuropathy \\
Muscular and fibrous abnormalities \\
Gantzer’s muscle \\
Enlarged bicipital bursa \\
Vascular abnormalities \\
Volkmann’s ischaemic contracture \\
Neuralgic amyotrophy \\
Isolated neuritis \\
Unknown \\
\hline
\end{tabular}
\end{table}

\begin{figure}[h]
\centering
\includegraphics[width=0.5\textwidth]{fig1.png}
\caption{Photograph showing the inability to form an ‘O’ with the thumb and the index finger in palsy of the left anterior interosseous nerve.}
\end{figure}
(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);28
(vi) an aberrant radial artery;
(vii) a tendinous origin of palmaris longus or flexor carpi radialis brevis; and
(viii) an enlarged bicapital bursa.

Collins and Weber29 considered entrapment to be by far the most common cause and Hill, Howard and Huffer30 observed it in 24 of 28 cases of incomplete palsy. Schantz and Riegels-Nielson18 found evidence of nerve compression in nine of 15 patients. Werner17 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 Goodfellow5 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 posteroradial direction and is located deeper than the median nerve. However, the patients of Fearn and Goodfellow5 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. Vichare31 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 Goodfellow5 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

Spinner9 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 electromyographic improvement in six to eight weeks, exploration is indicated. Nigst and Dick12 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 al30 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. Seror21 concluded that surgery should not be considered for a year because late spontaneous recovery is sometimes seen after this time. Futami et al32 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 al20 treated ten patients and believed the condition to be a neuritis. They concluded that surgical decompression may not hasten recovery. Tsukahara et al33 treated 12 hands from 11 patients conservatively. All paralysed 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 Burke15 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 al13 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-
servatively. No entrapment was found in either patient. All four recovered. Operation did not hasten the return of function, and therefore they concluded that surgery should not necessarily be done unless there is compelling evidence of an entrapment or a significant injury such as a penetrating wound.

After our experience in managing the 31 cases of non-traumatic AIN palsy noted above, we felt\(^{23}\) that the incidence of entrapment neuropathy was very low, and therefore recommended a policy of ‘wait and see’. However, in 1992, we encountered a patient with tenderness at the distal part of the arm. Exploration of the median nerve showed it to be partially swollen at the lower part of the arm. Interfascicular neurolysis of this area revealed an hourglass-like constriction in the fasciculus destined to become the anterior interosseous nerve. We suspected that the same lesion may have existed in the nine patients who had no evidence of external compression. Since then, we have performed interfascicular neurolysis in 23 patients who did not show any recovery by three months after onset and who agreed to surgical exploration. There were 12 men and 11 women, with a mean age at operation of 43.2 years (23 to 64). The mean interval between onset and the operation was 5.5 months (3 to 10). 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 (Figs 2 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 in only five patients in the literature.\(^{7,8,34-36}\) However, we have 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 such 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.\(^{37}\) 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 MRC grade 3 power in flexor pollicis longus and/or flexor digito-
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-Nielsen 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 Engle 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. Tajiri et al performed an experimental study on the median nerve in 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 et al 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


