Compressive neuropathy of the median nerve in the arm and forearm can be a difficult diagnostic and therapeutic challenge. It must be distinguished from cervical radiculitis, the thoracic outlet syndrome, brachial plexus neuritis, overuse of the muscles of the forearm, and carpal tunnel syndrome. In 1951, Seyffarth described a group of seventeen patients with symptoms that he considered diagnostic of the pronator teres syndrome. Preoperatively and postoperatively, all of the patients were examined by one or two of us (C. R. H. and R. L. L.). Twenty-nine patients were female and ten were male; their ages ranged from fourteen to seventy-eight years. The criteria for diagnosis were symptoms and signs of the median neuropathy localized to the forearm and hand: aching discomfort and easy fatigability of the muscles of the forearm brought on by cyclic stress, and indentation and tenderness of the pronator teres muscle. With repetitive pronation the patients also had numbness in the distribution of the median nerve in the hand; the numbness seldom was well localized, although it tended to involve the index finger and thumb and first was noticed after the discomfort became evident. Nocturnal awakening because of pain as well as numbness in the hand in the morning, commonly associated with the carpal tunnel syndrome, typically was absent. The duration of symptoms averaged twenty-three months (range, one to 120 months). The symptoms usually began insidiously, but occasionally after a specific muscle sprain or episode of activity their onset was rapid and dramatic. The symptoms frequently began during activities that required repetitive grasping or pronation, or both (for example, prolonged hammering, scraping dishes, ladling food, or practicing tennis serves).

We are reporting a study of thirty-nine patients in whom we diagnosed the pronator teres syndrome and attempted to identify the factors by which one can differentiate this disorder from other lesions.

Clinical Material

From 1972 to 1979, thirty-nine patients seen at the Mayo Clinic were diagnosed as having the pronator teres syndrome. Preoperatively and postoperatively, all of the patients were examined by one or two of us (C. R. H. and R. L. L.). Twenty-nine patients were female and ten were male; their ages ranged from fourteen to seventy-eight years. The criteria for diagnosis were symptoms and signs of a median neuropathy localized to the forearm and hand: aching discomfort and easy fatigability of the muscles of the forearm brought on by cyclic stress, and indentation and tenderness of the pronator teres muscle. With repetitive pronation the patients also had numbness in the distribution of the median nerve in the hand; the numbness seldom was well localized, although it tended to involve the index finger and thumb and first was noticed after the discomfort became evident. Nocturnal awakening because of pain as well as numbness in the hand in the morning, commonly associated with the carpal tunnel syndrome, typically was absent. The duration of symptoms averaged twenty-three months (range, one to 120 months). The symptoms usually began insidiously, but occasionally after a specific muscle sprain or episode of activity their onset was rapid and dramatic. The symptoms frequently began during activities that required repetitive grasping or pronation, or both (for example, prolonged hammering, scraping dishes, ladling food, or practicing tennis serves).

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under the taut origin of the flexor digitorum superficialis. We had no patients with post-traumatic syndromes in our series. The lack of specificity of the symptoms and the subtlety of the physical findings in several patients led to suspicion of either functional neurosis or conversion hysteria. Seven patients had had misdiagnoses that had led to carpal tunnel release, and one of them had had an ulnar transposition operation without relief of symptoms. The previous operations were done between three and twenty-eight months before our initial examination.

The physical findings in the seven patients who were not operated on and in the thirty-two who were operated on differed only in degree. The outstanding positive finding was tenderness in the proximal part of the pronator teres muscle, which was noted in thirty-seven patients. In the other two patients, this symptom may have been present but it was not noted. We found tenderness in the median nerve in the antecubital space in thirty-one patients, while tenderness was noted distally less frequently. Thirty patients experienced paresthesias in the distribution of the median nerve and the hand when the forearm was pronated forcibly against resistance. A positive Tinel sign at the proximal edge of the pronator teres was elicited in twenty patients. Fourteen patients suffered exacerbation of the paresthesias when the flexors of the long finger were contracted against resistance. In seventeen patients, the pronator muscle was observed to be unusually firm compared with the contralateral muscle. A depression in the contour of the forearm superficial to the lacertus fibrosus was obvious in three patients. Mild thenar atrophy was present in only three patients, and the Tinel test was rarely positive at the wrist, although nineteen patients had a positive Phalen sign at the wrist. Hypohidrosis in the distribution of the median nerve, which is common in the carpal tunnel syndrome, was not found in any of our patients.

Electrical Studies

Electromyography was done on forty forearms in thirty-eight patients. One patient had had an electromyographic study at another institution six months before an unsuccessful carpal-tunnel release, and the test was not repeated. All patients had bilateral measurements in the ulnar and median motor and sensory nerves. We measured the compound muscle and nerve-action potentials (amplitude and latency) from the thenar muscles and index finger during stimulation of the median nerve at the wrist and proximal to the pronator teres muscle. Nine of the thirty-eight patients studied electromyographically had measurements of segmental conduction velocity across the pronator muscle by needle-electrode stimulation; in an additional sixteen studies, the sensory-nerve conduction velocity was measured in the median nerve of the forearm, including the segment through the pronator teres. Electromyograms were made of several muscles that are innervated by the median nerve. A slowing of conduction velocity of more than ten meters per second or a block of conduction in the motor or sensory fibers in the median nerve at the level of the pronator teres, and electromyographic abnormalities limited to muscles innervated by the median nerve, were considered definitive electrophysiological findings for the pronator teres syndrome. Intraoperative recordings from the median nerve were made in ten forearms in eight patients in an attempt to identify a more localized abnormality than could be detected with routine recordings, and to record any electrophysiological changes in the nerve after decompression. Direct intraoperative measurements of mixed nerve-action potentials were made from the exposed nerve both proximal and distal to the pronator teres before and after surgical release of compression on the median nerve. Nerve-action potentials were stimulated and recorded using hand-held silver-hook pairs of electrodes with the nerve elevated away from surrounding tissue. Stimulation was applied distally in the forearm while recording was done proximal to the pronator teres muscle. Recordings were made before, immediately after, and fifteen to twenty minutes after surgical decompression of the nerve. Distances along the nerve between the stimulating and the recording electrodes were measured with a millimeter ruler to allow calculation of velocities from the latencies of the initial-positive peak. Amplitudes were measured from peak to peak.

Treatment

Seven patients considered that their symptoms were not severe enough to justify an operation. An explanation of the probable cause for the symptoms and steps that could be taken to minimize them, including job modifications, were given. Surgical exploration was performed in thirty-two patients (thirty-six forearms). In all thirty-two patients, cutaneous sensory nerves of the arm and forearm were identified and protected. We inspected the lacertus fibrosus, assessed its thickness, and determined whether it indented the antebrachial fascia over the pronator, especially during passive pronation and extension. Then the course of the median nerve was noted, and it was freed along the radial border. The relationship of the nerve to the pronator teres was assessed. Intramuscular tendinous bands within or under the pronator and fascial constricting bands between the superficial and deep heads of the muscle were released from the accompanying radial artery, as were vascular lesions or penetrating vessels. The anterior interosseous nerve was identified and protected, as was the arch of the flexor digitorum superficialis. If there was a tight falciiform edge, it was split carefully to expose the median nerve as it penetrated deep to the muscle; all motor branches to the muscle were protected.

Postoperative care consisted of immobilization of the arm in a padded plaster splint for two or three weeks, followed by mobilization of the elbow and strengthening of the muscles with activities added as tolerated. We advocated avoidance of strenuous work for six weeks. Occasionally a patient needed supervised physical therapy because of difficulty in mobilizing or strengthening the elbow.
Results

Evaluation of positive intraoperative findings depended on the surgeon's experience and judgment, and probably improved during the course of the study. In several patients more than one abnormality was found. Sixteen patients (sixteen forearms) had a hypertrophied pronator muscle or a tendinous band in the pronator capable of constricting the median nerve; fifteen had a thickened lacertus fibrosus that appeared to indent the flexor muscle mass; and twelve patients had a taut fibrous arch of the flexor digitorum superficialis. In six patients, the median nerve passed posterior to the ulnar head of the pronator. One patient with bilateral symptoms had a large pronator muscle with an extended proximal origin in each forearm, as well as tight ligamentous bands extending from the medial intermuscular septum to the pronator fascia. These bands simulated the ligament of Struthers and they compressed the nerve at the point of entry to the pronator. One patient had a large vascular structure penetrating through the nerve and another had a bursa on the insertion of the biceps that measured two centimeters in diameter and compressed the nerve. In two patients, no abnormalities were recognized.

The seven patients who were not operated on were followed for five to seventy-two months (average, twenty-eight months): two improved, four were unchanged, and one was worse. The improvement in the two patients was attributed to job modifications that allowed occasional rest periods and less repetitive use of the hands, but neither patient was always free of symptoms. The four patients whose condition was unchanged had adapted their jobs or activities to minimize their symptoms. At the time of writing, the one patient (a dentist) who was worse was considering undergoing an operation.

Postoperative follow-up in the thirty-two patients ranged from three to eighty-eight months and averaged eighteen months. We defined an excellent result as no residual pain, paresthesias, or weakness and no hypohidrosis, muscle atrophy, or diminished sensibility. A good result meant that most symptoms were relieved and the patient could pursue all previously limited activities in spite of the fact that a mild weakness, minimum pain, and some paresthesias of a non-disabling degree often were present. A fair result meant that the patient was able to work at least part-time but had residual episodes of disability.

The results were excellent in eight forearms, good in twenty, and fair in five. The condition of three patients was unchanged. One patient whose condition improved from fair to good had anomalous muscles and a small median nerve. Postoperatively, she had some weakness of the finger flexors and paresthesia in the hand. These gradually resolved during an eight-month period, but some weakness during pronation persisted. Another patient had a good result in the right arm but only a fair result in the left arm. The most common lesions in the patients with good or excellent results were a taut lacertus fibrosus, an intramuscular tendinous abnormality about the median nerve, and tightness at the arch of the flexor digitorum superficialis. The patients with fair or unchanged ratings usually had less conspicuous lesions, although in all three patients who had unchanged ratings at least one anatomical abnormality was identified. Two of these three patients, however, later required care for emotional disorders, and in the third an undiagnosed neural disease appeared to have developed.

Electrodiagnosis

Among the results from forty standard electrical studies, the mean values for amplitudes and velocities of the median motor and sensory nerves in the entire group were abnormal (velocities of 58.0 ± 1.1 and 62.1 ± 1.0 meters per second, respectively), but only twelve (30 per cent) of the forearms showed values outside the normal range. Of these, only six had evidence of a proximal lesion in the median nerve on standard studies. The changes were elicited primarily by needle-electrode examination.

Two (22 per cent) of the nine patients who had measurements of segmental motor-nerve conduction velocity showed local slowing or a block at the pronator teres. None had dispersion of evoked responses. Evidence of segmental compression of the sensory part of the median nerve during its course through the pronator teres was sought in twenty-five patients and was elicited in four (16 per cent). Four other patients showed other, nondiagnostic abnormalities.

A total of nine patients had electromyographic abnormalities suggestive of damage to the median nerve proximal to the wrist: five had abnormalities on the needle-electrode examination of muscles, four had delays in sensory conduction (all mild), and two had abnormalities on motor conduction studies (Table I).

<p>| TABLE I |
|-----------------|-----------------|-----------------|-----------------|-----------------|-----------------|</p>
<table>
<thead>
<tr>
<th>Electrographic Findings</th>
<th>Patients Not Operated On (No.)</th>
<th>Good</th>
<th>Fair</th>
<th>Poor</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total of forearms*</td>
<td>7</td>
<td>24</td>
<td>6</td>
<td>3</td>
<td>40</td>
</tr>
<tr>
<td>Normal</td>
<td>7</td>
<td>12</td>
<td>4</td>
<td>2</td>
<td>25</td>
</tr>
<tr>
<td>Pronator teres syndrome</td>
<td>0</td>
<td>1</td>
<td>1</td>
<td>0</td>
<td>2</td>
</tr>
<tr>
<td>Proximal median-nerve damage</td>
<td>0</td>
<td>8</td>
<td>1</td>
<td>0</td>
<td>9</td>
</tr>
<tr>
<td>Carpal tunnel syndrome</td>
<td>0</td>
<td>5</td>
<td>1</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>* Some patients had two types of abnormality.</td>
<td></td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

Only two patients had definite electrophysiological evidence of pronator teres syndrome. Seven patients had electromyographic evidence diagnostic of carpal tunnel syndrome (Table I), and one of these also had evidence of changes in a more proximal part of the nerve. One patient had unusual findings: dense fibrillation potentials in the pronator teres muscle.

Ten patients had intraoperative electrophysiological studies. On standard electromyographic studies in these...
patients, five of the recordings were normal, one showed slow sensory conduction velocity, three showed proximal changes on needle-electrode examination, and four had evidence of the carpal tunnel syndrome. None showed clear-cut electrophysiological evidence of neuropathy of the median nerve at the pronator muscle. Conduction velocities recorded intraoperatively were within the expected normal range in nine of the ten patients (fifty-six to sixty-eight meters per second), while one patient had slow conduction (forty-eight meters per second). Three of the ten patients showed an improvement of five meters per second or more after decompression, and seven had conduction velocities that were unchanged.

Measurements of amplitudes of nerve-action potentials ranged widely, but three increased 50 per cent or more after decompression, and none decreased by more than 20 per cent. All ten patients had good to excellent results after the operation.

Subsequent Operations

Six patients had additional operations after the initial so-called index operation for the pronator teres syndrome. The first patient was essentially symptom-free for three years, and then the symptoms recurred. Re-exploration revealed that the arch of the flexor digitorum superficialis was markedly tight, a finding that was not observed at the first operation forty-four months earlier. Her condition was improved on follow-up examination, six months after the second operation. The second patient had no significant relief from the original operation. Re-exploration eight months later disclosed that the tendon from the ulnar head of the pronator teres lying beneath the nerve acted as a sharp-edged, compressive structure when the forearm was pronated. This tendon was released, and the symptoms were alleviated. The third patient had two operations for carpal tunnel release after the index procedure on the pronator teres. The first release was performed on the left side six months after the index operation, and the second was done on the right side sixteen months after the pronator release. Symptoms of the pronator teres syndrome had been relieved by the index operation. The patient and the examiner both considered that the symptoms of the carpal tunnel syndrome differed from the earlier symptoms. The fourth patient underwent exploration of the carpal tunnel seventeen months after the release of the pronator teres. The median nerve was normal at the wrist, but a functional anastomosis was found between the flexor pollicis longus and the flexor digitorum profundus tendon of the index finger (Linburg's syndrome). This anastomosis was released and there was subsequent resolution of the symptoms. In the fifth patient, a symptomatic bilateral ulnar neuropathy developed six years after she had an excellent result from the operation on the pronator teres. She had anterior transposition of both ulnar nerves ninety-one months after the pronator release. Five years after the index operation the sixth patient underwent fusion of the sixth and seventh cervical vertebrae for symptoms of cervical radiculitis, unrelated to the pronator teres syndrome.

Discussion

The pronator teres syndrome can be produced by one or more abnormalities that frequently are subtle and require judgments that are not quantified easily. Constriction of the nerve is different from that seen in the carpal tunnel syndrome. Of the thirty-six median nerves that we explored, sixteen were constricted by fascial or tendinous structures in the pronator teres. The lacertus fibrosus appeared to be a cause of constriction in fifteen forearms, and the superficialis arch was taut in another thirteen. The incidence of the apparent causes of this syndrome in our series parallels the experience of Johnson et al.

Seyffarth described seventeen patients who had weakness of opposition and hyperesthesia of the ulnar border of the thumb and radial border of the index finger. This combination of symptoms was distinctly unusual in our experience. Injections of procaine hydrochloride into the pronator teres muscle produced partial or complete relief in a number of his patients at the time of follow-up. Seyffarth believed that the "pressure of work" from writing or from the tight gripping and turning of tools resulted in a hard, painful pronator muscle and caused the syndrome.

Hypertrophy of the muscles of the volar part of the forearm related to work appears to be a factor in the etiology of the pronator teres syndrome. The pronator muscle may be overdeveloped during repetitive activity, so that the lesion represents a dynamic compartment syndrome. The muscular induration seen on the symptomatic forearm and the indentation of the flexor pronator mass by the lacertus in some of our patients suggest that there is muscular hypertrophy within a fascial compartment. A recent example of this in our patients was a school-bus driver who had sprained his thumb. The cast that was applied necessitated his gripping the steering wheel with only the fingers. This led to a rather acute pronator teres syndrome in a few days, which subsided with rest. Recent work has suggested that the pressures in the proximal muscle compartment should be measured to assess the role of increased pressures in the etiology of the pronator teres syndrome. It is possible that fasciotomy in the proximal part of the forearm may be a significant factor in the favorable response to treatment.

Spinner noted acute syndromes in patients who were undergoing renal dialysis and anticoagulant therapy, and others have described patients with the syndrome due to trauma, but neither of these causes for the syndrome was evident in our patients. Spinner thought that resisted flexion and supination of the elbow seemed to significantly exacerbate symptoms caused by a tight lacertus fibrosus. We noted intraoperatively that the lacertus appears to compress the pronator teres during passive pronation, apparently because the tendon of the biceps moves distally, drawing the origin of the lacertus with it.

Of ten patients in the series of Bell and Goldner, in four who had cerebral palsy symptoms of the pronator teres a supinator hemiplegia under our p...
teres syndrome developed when the arm was placed in a supination cast for pronation contracture of the forearm. Tendon-lengthening in another patient with spastic hemiparesis apparently pulled the median nerve tautly under the superficialis, a situation similar to that in one of our patients.

In 1939, Beaton and Anson delineated the anatomical variations of the pronator teres, with special attention to the route of the median nerve. They found that the nerve passed between both heads of the pronator muscle in 82.5 per cent of 240 arms, below a solitary humeral head in 8.8 per cent, and below both heads in 6.3 per cent. In 2.5 per cent of the arms the nerve pierced the humeral head.

Spinner discussed the case of one patient in whom the median nerve ran posterior to both heads of the pronator. Six of our patients appeared to have this finding. We did not try to reroute the median nerve subcutaneously by rerouting the pronator teres, as suggested by Spinner.

Solnitzky believed that the variability of symptoms and physical findings in the pronator teres syndrome was explained by changes in patterns of rest and activity. He thought that the variability in the sensory and motor signs was due to the functional groupings of fibers within the nerve and the location of the compressing agent relative to the nerve. Our experience seems to support this concept. For example, compression of the median nerve that was caused by a tight lacertus fibrosus in our patients resulted in pain that spread diffusely in the volar aspect of the forearm, while compression by a tight superficialis arch caused more localized pain.

Diagnosis of the pronator teres syndrome seldom is easy. This point is illustrated by the cases of the seven patients in our group who had had unsuccessful operations between three and twenty-eight months before our initial examination.

Five patients required a subsequent operation after release of the pronator teres that initially gave good or excellent results. Aside from the possibility of error in the original diagnosis or the existence of some uncorrected lesion, a plausible reason for the second operation in one patient was that an unrelated ulnar neuropathy might have developed. In three patients a carpal tunnel syndrome may have coexisted with or developed after the pronator teres syndrome, perhaps in a nerve rendered more susceptible to compression in the carpal tunnel. Inadequate release of the compressing structures at the initial exploration may have accounted for the recurrence of symptoms in the fifth patient. Although a constricting fascial band and the superficialis arch were reported to be divided at the first operation, at re-exploration the superficialis arch again was found to be tight.

Lister compared the diagnosis of the pronator teres syndrome with that of the carpal tunnel syndrome. He described features common to both but noted that the pronator teres syndrome was not associated with a positive Phalen test. Our experience does not confirm his assertion. Fifty per cent of our patients had a positive Phalen test preoperatively, as do a moderate number of patients who are asymptomatic postoperatively.

Two electromyographic studies of the pronator teres syndrome have been reported. Buchthal et al. found only minimum abnormalities in eleven patients. Three patients had abnormal nerve-conduction studies, while all eleven had abnormal needle electromyograms. Morris and Peters noted abnormal motor conduction in six of eight involved arms in patients who had significant motor and sensory deficits on neurological examination.

The results of electrophysiological testing in our patients were similar to those reported by Buchthal et al. The motor and sensory conduction velocities and amplitudes for the median nerve were significantly although mildly reduced, particularly in patients with more severe symptoms. In most patients, therefore, the nerve was not affected severely enough to result in conduction values outside the normal range, and the mildness of the abnormality was consistent with the minimum neural deficit. The findings were probably due to the intermittent compression of the nerve and possibly to a selective involvement of the small pain fibers. The nerve-conduction findings and the frequency of abnormal studies on needle examination in this disorder resemble the signs of traumatic neuropathies more than those of chronic compressive neuropathies. Chronic compressive neuropathies typically are characterized by either local slowing of conduction or a conduction block due to segmental demyelination and axonal narrowing, while traumatic neuropathies more commonly result in axonal loss with low-amplitude responses. This type of pathological change also is consistent with our inability to localize the damage electrophysiologically in most patients.

The large number of normal electromyographic findings in our patients indicates that electromyography cannot exclude the diagnosis of the pronator teres syndrome, nor can it predict the outcome of operation. The entirely normal electromyographic findings in the patients who were not operated on may have influenced this group of patients not to have an operation, and that fact may be important in the evaluation of electromyographic tests. However, in patients with suspected pronator teres syndrome, electrophysiological testing was of value for two specific purposes: (1) it distinguished other peripheral nerve disorders that present with similar symptoms, and (2) it showed that a non-localizable median-nerve lesion was present in 15 to 20 per cent of the patients with the syndrome, and in a smaller percentage it accurately localized the lesion.

Identification and localization were enhanced by all three types of electrophysiological study — motor and sensory conductions across the pronator and needle electromyograms of the forearm and hand muscles. As no one study was superior to the other two, all are needed in testing for the pronator teres syndrome. Precise localization in a few patients was possible with conduction studies across short segments but it was not of value unless an abnormal-
The results of the intraoperative electrophysiological recordings were similar to those of preoperative studies: they showed only mild abnormalities that did not identify or localize the damage any better than did standard studies. However, after decompression of the nerve the change in conduction velocity and in amplitude of the nerve-action potentials measured intraoperatively in some patients was evidence that there was an improvement in nerve function.

Some of the patients who improved after operation had clear-cut electromyographic evidence of other disorders — carpal tunnel syndrome in seven patients and an upper-arm median neuropathy in one other patient. This implies that the pronator teres syndrome may coexist with other median neuropathies or that these other disorders may improve after decompression of the proximal median nerve.

The fact that increased latencies in conduction in the median nerve were found across the carpal canal in some of these patients requires some explanation. The symptoms in these patients appeared to arise primarily in the proximal part of the forearm, where tenderness of the pronator and the nerve proximal to the pronator area was evident. These symptoms could be worsened more by resisted pronation and contraction of the flexor digitorum superficialis than by the Phalen test. Most of this small group of patients had complaints of fatigue and pain in the forearm, with paresthesias in the fingers which usually followed the onset of the pain. Generally, these patients did not have nocturnal paresthesias. Three of them had had previous carpal-tunnel decompression without relief of their primary symptoms, and two additional patients had not responded to injection of cortisone into the carpal canal. As mentioned, there may be a relationship between the pronator teres syndrome and the carpal tunnel syndrome because of increased susceptibility of the nerve to a compression neuropathy when the syndromes coexist. The finding of an increased latency in the conduction time of the median nerve in the carpal tunnel could be explained in that way.

It is interesting to speculate why anterior interosseus-nerve palsy is not seen with the pronator teres syndrome when the sites of the lesions are so near to one another in the area between the ulnar head of the pronator and the origin of the flexor digitorum superficialis. In the seven-year period of this study, only two such patients with the Kiloh-Nevin syndrome were seen in our institution, and in them direct pressure of the ulnar head of the pronator teres muscle was seen on the anterior interosseus nerve just below its origin.

References