Pronator Syndrome and Other Nerve Compressions That Mimic Carpal Tunnel Syndrome

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The purpose of this clinical commentary is to provide a comprehensive review of compressive neuropathies that may mimic carpal tunnel syndrome, provide the clinician with information to differentially diagnose these median nerve compression sites, and provide an evidence-based opinion regarding conservative intervention techniques for the various compression syndromes. While rare in comparison to carpal tunnel syndrome, pronator syndrome and anterior interosseous nerve syndrome are proximal median nerve compressions that may be suspected if a patient with carpal tunnel syndrome fails to respond to conservative or surgical intervention. Differential diagnosis is based largely on the symptoms, patterns of paresthesia, and specific patterns of muscle weakness. Due to the relative rarity of pronator syndrome and anterior interosseous nerve syndrome, few controlled studies exist to determine the most effective treatment techniques. Based on sound anatomical and biomechanical considerations, anecdotal experience, and available research, however, treatment strategies for pronator syndrome and anterior interosseous nerve syndrome compression neuropathies can be divided into 4 major categories: (1) rest/immobilization, (2) modalities, (3) nerve gliding, and (4) nonconservative treatment. *J Orthop Sport Phys Ther 2004;34:601-609.*

Key Words: *anterior interosseous nerve syndrome, differential diagnosis, hand, median nerve, pronator syndrome*

Carpal tunnel syndrome (CTS) is a commonly diagnosed cause of median nerve compression.[20,23] Due to the increased awareness of CTS, it may often be over diagnosed, misdiagnosed, or simply assumed based on symptoms.[33] Median nerve neuropathies proximal to the carpal tunnel, however, should be suspected especially when the symptoms persist following conservative or surgical intervention aimed to the site of the carpal tunnel. Median nerve neuropathies can be due to diabetes, human immunodeficiency virus, nutritional deficiencies, and entrapment/compression of the nerve. Median nerve compression proximal to the carpal tunnel may be divided into 2 major categories: pronator syndrome (PS) and anterior interosseous nerve syndrome (AINS).

While relatively rare, median nerve compressions proximal to the wrist sometimes occur in isolation or in conjunction with other lesions. Gessini et al[13] reported the following distribution of compression syndromes on a series of 228 patients with median nerve entrapments: 201 patients (88.2%) with CTS, 21 patients (9.2%) with PS, 3 patients (1.3%) with AINS, 2 patients (0.9%) with lacertus fibrosus compression, 1 patient (0.5%) with Struthers' ligament compression. Consequently, due to the low incidence of PS and AINS, very little data exist which might aid the clinician in differentially diagnosing proximal sites of median nerve compression. Differential diagnosis is also complicated by the fact that electrodiagnostic testing is often inconclusive in cases of PS.[11,25,31,34] Therefore, a diagnosis of PS or AINS is based largely upon the clinician's understanding of median nerve anatomy, the potential sites of compression of the median nerve, and the characteristic signs and subjective complaints of pain and/or paresthesia.[25,34]

The purpose of this paper is to provide a review of compressive neuropathies that may mimic CTS, provide the clinician with information to differentially diagnose these compression sites, and provide evidence-based opinions regarding conservative intervention techniques for the various compression syndromes.
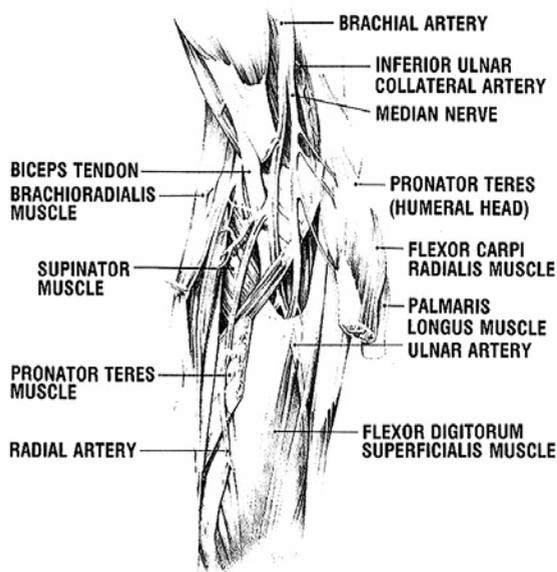
MEDIAN NERVE ANATOMY

The median nerve is formed by the lateral (C5 through C7) and

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FIGURE 1. Path of median nerve. At the elbow, from lateral to medial, are the biceps tendon, brachial artery, and the median nerve. The lacertus fibrosus is removed and the pronator teres is reflected, revealing the course of the median nerve as it passes deep to the flexor digitorum superficialis. Reprinted with permission from RH Elberman, ed. *Operative Nerve Repair and Reconstruction.* Philadelphia, PA: JB Lippincott; 1991.
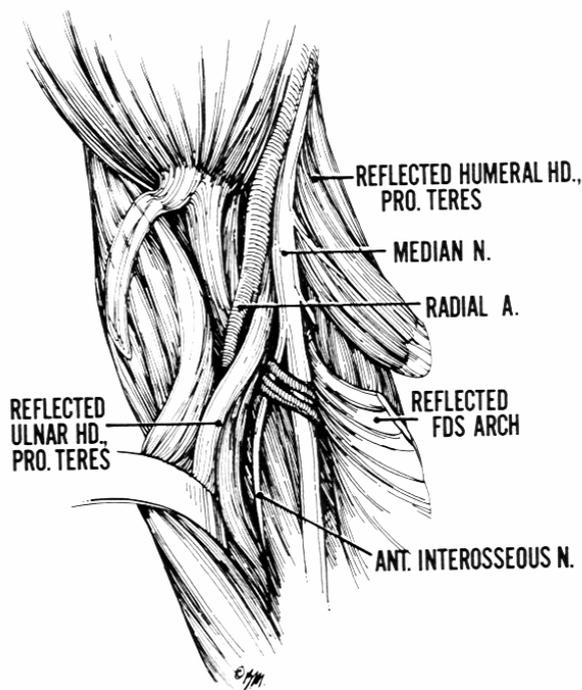
FIGURE 2. Anterior interosseous nerve. The anterior interosseous branch of the median nerve originates 5 to 8 cm distal to the medial epicondyle from the posterolateral aspect of the median nerve just distal to the proximal border of the superficial head of the pronator teres. Abbreviations: FDS, flexor digitorum superficialis; pro. teres, pronator teres. Illustration by Elizabeth Roselius, ©1993. Reprinted with permission from DP Green, ed. *Operative Hand Surgery.* New York, NY: Churchill Livingstone; 1993.

medial (C8 and T1) cords of the brachial plexus. At the elbow, from lateral to medial, are the biceps tendon, brachial artery, and the median nerve (Figure 1). The median nerve lies anterior to the brachialis and deep to the lacertus fibrosus.[11,31] The median nerve then passes between the superficial (humeral) head and deep (ulnar) head of the pronator teres in the proximal third of the forearm. At this point, the median nerve crosses to the lateral side of the ulnar artery, separated from the ulnar artery by the deep head of the pronator teres.[11] The course of the median nerve proceeds posterior to the fibrous arch formed by the 2 heads of the flexor digitorum superficialis (FDS), finally emerging in the distal third of the forearm along the lateral edge of the FDS.[31]

The first branches from the median nerve appear in the antecubital fossa and sequentially supply the pronator teres, flexor carpi radialis, palmaris longus, and the FDS.[28] The anterior interosseous branch of the median nerve originates 5 to 8 cm distal to the medial epicondyle from the posterolateral aspect of the median nerve just distal to the proximal border of the superficial head of the pronator teres[31] (Figure 2). The anterior interosseous nerve then accompanies the median nerve through the fibrous arch of the FDS and comes to lie anteriorly on the interosseous membrane, coursing distally with the anterior interosseous artery, where it supplies motor branches to the flexor pollicis longus (FPL), flexor digitorum profundus (FDP) of the index and long fingers, and the pronator quadratus (PQ), in which it terminates.[11]

Another branch of the median nerve that is significant to differential diagnosis is the palmar cutaneous branch. This branch arises from the lateral side of the median nerve 7 cm proximal to the distal wrist crease.[3] The nerve then runs deep to the antebrachial fascia, close to the medial border of the flexor carpi radialis, and then enters its own tunnel. The palmar cutaneous branch innervates the skin over the palm and proximal thenar area.

PRONATOR SYNDROME

PS was first described by Seyfferth in 1951.[21] It was originally thought that the median nerve was compressed between the 2 heads of the pronator teres or by the FDS.[14] Since Seyfferth's original description, PS has been expanded to encompass compression of the median nerve at the ligament of Struthers, lacertus fibrosus (bicipital aponeurosis), pronator teres, and the arch of the FDS.[31]

Ligament of Struthers Compression

The ligament of Struthers connects an anomalous bony spur on the humerus to the medial epicondyle[5]
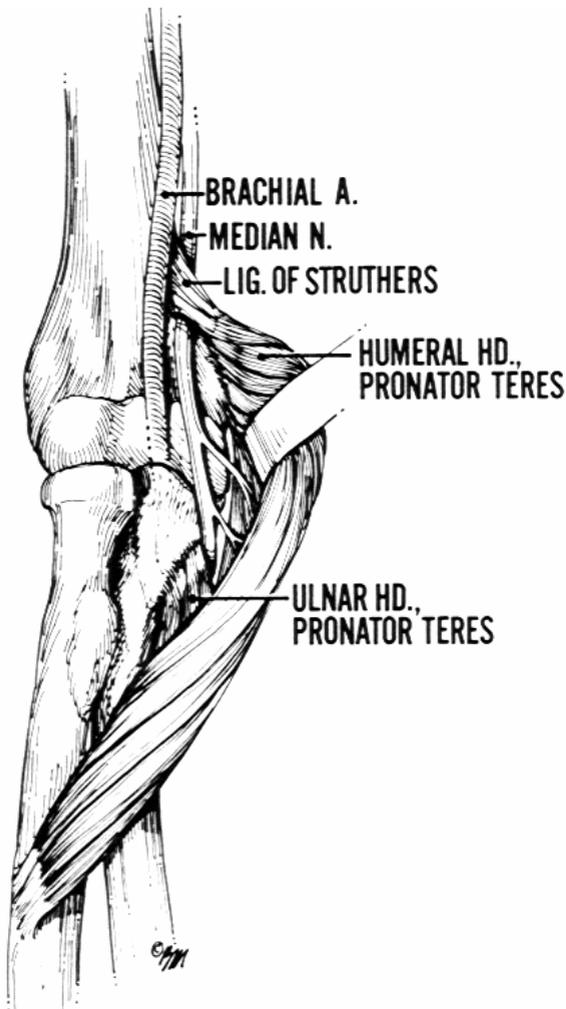
FIGURE 3. Ligament of Struthers. The ligament of Struthers connects an anomalous bony spur on the humerus to the medial epicondyle as the median nerves passes underneath. Illustration by Elizabeth Roselius, ©1993. Reprinted with permission from DP Green, ed. *Operative Hand Surgery.* New York, NY: Churchill Livingstone; 1993.

(Figure 3). The presence of the ligament of Struthers is considered rare and is reported to occur in only 0.7% to 2.7% of the population.[5,7,31,35] While presence of this ligament is rare, compression of the median nerve caused by the ligament of Struthers is even more rare at 0.5% of patients with median nerve compression.[13]
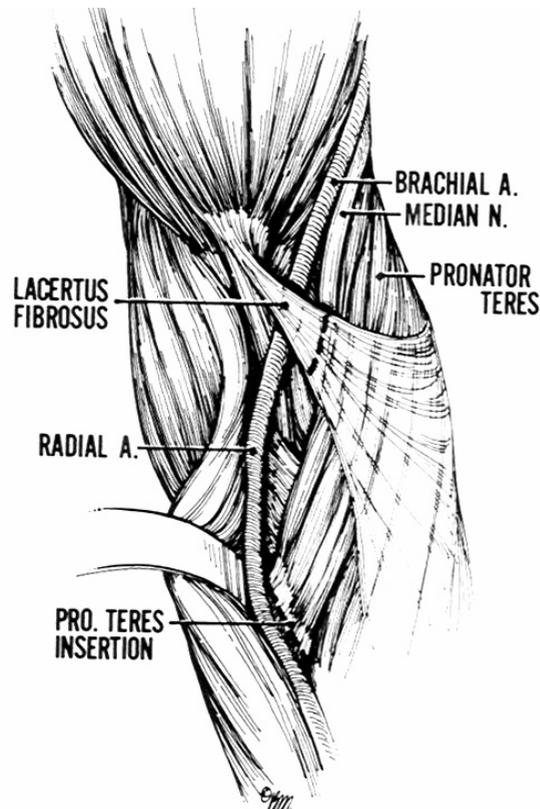
Lacertus Fibrosus Compression

The lacertus fibrosus, or bicipital aponeurosis, extends obliquely from the biceps tendon across the antecubital fossa to the fascia overlying the flexor forearm muscles[22] (Figure 4). Several case studies exist to document the lacertus fibrosus as a potential site of median nerve entrapment.[19,22,28] In general, median nerve entrapment by the lacertus fibrosus is also considered rare and has been reported to occur in only 0.9% of cases of median nerve compression.[13]

Pronator Teres Compression

The course of the median nerve in relation to the pronator teres is somewhat variable. In approximately 80% of cadaver dissections, the median nerve passes between the superficial and deep heads of the pronator teres.[11,17,31,32] When both heads of the pronator teres are present, the median nerve passes behind both heads in 4.6% of cases and passes through the deep head in 1.8% of cases.[32] The deep head of the pronator teres is absent in 21.7% of the population.[21] Consequently, the median nerve passes behind the superficial head with the deep head absent in 11% of cases of PS.[32] Despite this variability, most cases of PS are believed to be caused by fibrous bands that compress the median nerve as it passes between the 2 heads of the pronator teres.[25] The frequency of median nerve entrapment at the pronator teres has been reported at 9.2% of cases of median nerve compression.[13]

Flexor Digitorum Superficialis Compression

The FDS is often reported as having 2 heads, humeral and interosseous membrane, which are

FIGURE 4. Lacertus fibrosus. The lacertus fibrosus extends obliquely from the biceps tendon across the antecubital fossa to the fascia overlying the flexor forearm muscles, possibly creating compression of the median nerve. Illustration by Elizabeth Roselius, ©1993. Reprinted with permission from DP Green, ed. *Operative Hand Surgery.* New York, NY: Churchill Livingstone; 1993.

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joined together by a tendinous arch.[31] Dellon and MacKinnon[9] described a tough fibrous arch linking the 2 heads of the FDS, through which the median nerve could be potentially compressed. Compression of the median nerve at the FDS is considered the second most common cause of PS, following the pronator teres.[25]

CLINICAL PRESENTATION OF PRONATOR SYNDROME

Patients with pronator syndrome typically complain of pain in the proximal volar (anterior) aspect of the forearm.[11,14,15,25,35] This pain is commonly aggravated by activities such as repetitive pronation and supination.[11,25,31] PS has also been related to repetitive exertional grasping work, such as that performed by assembly line workers, carpenters, weightlifters, and tennis players.[15] PS usually presents in the fifth decade and is 4 times more common in women than men.[15,31] The onset of symptoms are insidious and there is usually a delay in diagnosis of 9 months to 2 years.[31]

In addition to complaints of pain, patients also typically complain of paresthesia in the thumb, index, and long fingers.[11,14,15,35] Patients may also complain of numbness in the palm consistent with the distribution of the palmar cutaneous branch of the median nerve.[25] Although these neurologic signs are present, these patients have a notable absence of nocturnal symptoms in contrast to patients with CTS.[14,25,31,34] Once the subjective complaints of pain and paresthesia are determined to be consistent with PS, objective tests can then be performed to further clarify the location of median nerve compression. As PS is considered rare, no sensitivity or specificity data exist in regard to objective tests for PS.

Three tests have been described to determine the site of proximal median nerve entrapment. These tests are based on creating maximal tension on the anatomical sites that can contribute to compression of the median nerve as it courses from the elbow to the wrist. The pronator teres is indicated as the source of compression by reproduction of symptoms with resisted pronation, with the forearm in neutral, as the elbow is gradually extended.[31] The lacertus fibrosus is implicated if a reproduction of symptoms occurs with resisted elbow flexion at 120° to 130° flexion with the forearm in maximal supination.[31] Finally, the FDS is implicated as the source of compression by reproduction of symptoms with resisted flexion of the proximal interphalangeal joint to the long finger.[25]

Another test to aid in the diagnosis of PS is the pronator compression test (Figure 5). The test is performed by placing pressure over the pronator muscle in both upper extremities. A positive test is indicated by reproduction of paresthesia in the lateral 3½ digits in 30 seconds or less, while the

FIGURE 5. Pronator compression test. The test is performed by placing pressure over the pronator muscle in both upper extremities. A positive test is indicated by reproduction of paresthesia in the lateral 3½ digits in 30 seconds or less, while the uninvolved limb remains asymptomatic.

uninvolved limb remains asymptomatic. While no sensitivity or specificity data exist regarding the pronator compression test, a study was performed by Gainor,[12] which indicated a positive compression test in all 10 patients with surgically confirmed pronator syndrome.

The clinician should also be aware of the possible presence of a "double crush" syndrome. Double crush syndrome refers to multiple asymptomatic nerve compression sites along the course of a nerve that create a symptomatic compressive neuropathy as the result of the cumulative compression sites.[8] Upton and McComas[8] theorized that neural function becomes impaired because single axons, compressed in one region, become more susceptible to damage at another site. For example, a patient may present with CTS, have the transverse carpal ligament surgically released, and experience minimal relief of symptoms. In this case, there may also be a concomitant compression of the median nerve at the pronator teres or at the cervical level that would explain the lack of complete symptomatic relief following surgery. When a double crush syndrome is present, differentiation of the possible sites of proximal median nerve compression can be difficult.

Other tests that may be useful include Phalen's test and Tinel's sign. If the patient has only a proximal median nerve compression, then the Phalen's test for CTS would be expected to be negative.[14,35] Tinel's sign over the pronator teres may be positive, but only if symptoms have existed for more than 4 months.[14]

Electrodiagnostic studies are somewhat controversial and are rarely diagnostic for PS.[11,25,31] Nerve conduction studies are not a sensitive indicator of median nerve entrapment at the elbow/forearm.[1,31] This is either due to the fact that these studies are within normal range at time of presentation,[1,34] or that patients with CTS may have a slowing of the median nerve conduction velocity for a variable

distance proximal to the wrist.[31] Electromyographic studies of median-nerve-innervated muscles are considered somewhat more reliable.[1,11] Electrodiagnostic studies, however, are useful when attempting to rule out the presence of CTS both as a primary diagnosis or coexisting with PS (double crush syndrome).[25,31]

ANTERIOR INTEROSSEOUS NERVE SYNDROME

AINS was first described by Kiloh and Nevin in 1952.[14] AINS is considered rare, as it accounts for fewer than 1% of all upper extremity neuropathies.[31] The anterior interosseous nerve is clinically considered a pure motor nerve,[35] although it does have terminal sensory branches at the distal radioulnar joint and the radiocarpal joint.[31]

There are several sites that may compress the anterior interosseous nerve, including the deep head of the pronator teres,[28] FDS,[16] Gantzer's muscle (accessory head of the FPL),[4] tendinous origin of the palmaris profundus,[24] and an accessory lacertus fibrosus.[28] Compression of the anterior interosseous nerve can also occur by direct physical compression of the nerve or the nerve's vascular supply by a blood vessel. These blood vessels that may act as sources of compression include ulnar recurrent vessels, an aberrant radial artery, an anomalous median artery, and the anterior interosseous vessels as they cross the anterior interosseous nerve.[24]

CLINICAL PRESENTATION OF ANTERIOR INTEROSSEOUS NERVE SYNDROME

As in PS, the patient often complains of pain in the proximal volar (anterior) forearm.[14] This pain tends to increase with repetitive forearm motion.[31] The patient may also complain of difficulty writing or picking up small objects,[29] due to weakness in the FPL, FDP of the index and long finger, and the PQ.[31] A key characteristic is the usual lack of paresthesia.[11,35]

Two types of AINS have been described: an incomplete and a complete syndrome.[16] Incomplete AINS is characterized by the loss of function of only the FPL or the FDP to the index finger.[28] The complete AINS is characterized by weakness of the FPL, FDP of the index and long fingers, and the PQ. In addition, the patient may present with a "classic attitude" of weak pinch when attempting to touch the tip of the thumb to the tip of the index finger[11,16,31,35] (Figure 6). This change in pinch is considered to be an indicator of late stages of AINS.[11]

In addition to manual muscle testing and observation of pinch, electrodiagnostic testing is useful in the diagnosis of AINS[11,29] in contrast to PS. Electromyographic testing confirms the diagnosis of AINS in 80% to 90% of cases.[14]
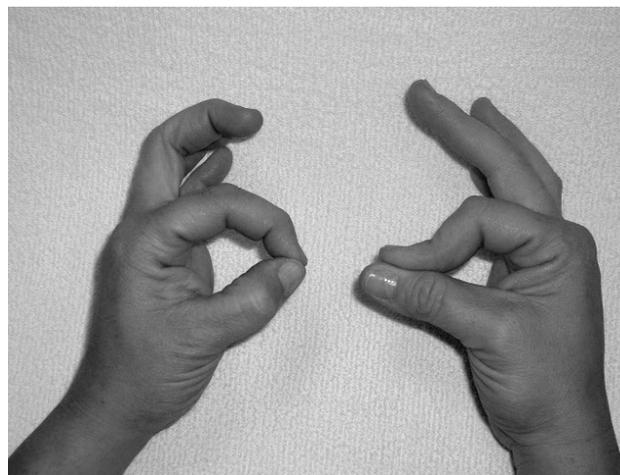
FIGURE 6. Anterior interosseous nerve syndrome change in pinch. Note the pinch on the right hand exhibits hyperextension at the distal interphalangeal joint due to weakness of the flexor digitorum profundus and the use of the lateral portion of the interphalangeal joint of the thumb due to weakness of the flexor pollicis longus.

DIFFERENTIAL DIAGNOSIS

Although PS and AINS are relatively rare in comparison to CTS, it is important to be able to differentially diagnose these syndromes to direct intervention appropriately (Table 1).

Paresthesia is absent in AINS and present in both PS and CTS. CTS involves paresthesia in the lateral 3½ digits, whereas PS involves paresthesia in the lateral 3½ digits and often in the distribution of the palmar cutaneous branch of the median nerve.[25] CTS often involves nocturnal symptoms, whereas PS has none.[14] The paresthesia in CTS may be reproduced by compression over the wrist or Phalen's test, whereas PS requires compression at the pronator teres to reproduce paresthesia. Tinel's sign is present at the wrist in 80% of cases of CTS and is present at the pronator teres in PS in less than 50% of cases.[14] One must also be aware that CTS and PS can exist simultaneously (double crush syndrome), which further complicates any attempt at differential diagnosis.

AINS is easily differentiated from CTS by the lack of paresthesia complaints. However, AINS can also be differentiated from CTS in that thenar atrophy occurs in later stages of CTS and does not in AINS.[25] AINS, particularly the incomplete syndrome, may also need to be differentiated from tendon ruptures of the FPL or the FDP. This can be done via tenodesis or direct electrical stimulation of the FDP or FPL.[31]

Although not covered extensively in this paper, even more proximal causes of median nerve pathology should also be ruled out. These causes include, but are not limited to, cervical radiculopathy, brachial plexopathy, and thoracic outlet syndrome.[25]

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TABLE 1. Differential diagnostic characteristics of carpal tunnel, pronator, and anterior interosseous syndromes.

	Paresthesia	Nocturnal Symptoms	Muscle Weakness/Atrophy	Tinel's	Phalen's	Direct Compression	Electrodiagnostic Tests
Carpal tunnel syndrome (CTS)	Lateral 3½ digits	Yes	Abductor pollicis brevis, opponens pollicis, flexor pollicis brevis	Positive at carpal tunnel	Positive	Positive at carpal tunnel	Diagnostic
Pronator syndrome (PS)	Lateral 3½ digits and palm	No	Not a traditional sign, but may involve abductor pollicis brevis, opponens pollicis, flexor pollicis brevis, flexor pollicis longus, flexor digitorum profundus of index and long fingers, pronator quadratus, and flexor carpi radialis	Positive at pronator teres <50%	Negative	Positive at pronator teres, negative at carpal tunnel	Rarely diagnostic
Anterior interosseous nerve syndrome (AINS)	None	No	Flexor pollicis longus, flexor digitorum profundus of index and long fingers, pronator quadratus	Negative	Negative	Negative	Diagnostic

INTERVENTION

With conservative treatment, 50% of patients with PS have been reported to recover in 4 months.[14] In addition, there are reports of improvement from 18 months to 2.5 years after conservative treatment.[31] As PS and AINS are considered rare, few controlled studies exist to determine the most effective intervention techniques. However, based on sound anatomical and biomechanical considerations, anecdotal experience, and available research, interventions can be divided into 4 major categories: (1) rest/immobilization, (2) modalities, (3) nerve gliding, and (4) nonconservative treatment.[14,25,31]

Rest/Immobilization

Perhaps the most important aspect of conservative care is instructing the patient to avoid aggravating activities such as repetitive pronation/supination and aggressive physical activities involving forceful grip (weightlifting, tennis).[16] To prevent an exacerbation of their symptoms, the clinician may fabricate a posterior elbow splint with the elbow at 90° flexion and the forearm in mid rotation.[14,25] This splint is typically worn for 2 weeks and only removed for gentle range-of-motion activities. We suggest that patients consciously avoid activities known to aggravate their symptoms for an additional 2 to 4 weeks after splint removal and gradually reintegrate these activities as symptoms allow.

Modalities

Some of the modalities typically used for treatment of nerve compression syndromes include ultrasound, electrical stimulation, and iontophoresis. Although research on the use of these modalities for PS and AINS does not exist, research on the use of these modalities for treatment of other nerve pathologies does exist and may be relevant to the treatment of individuals with PS and AINS. Ebenbichler et al[10] found that in cases of mild to moderate CTS, ultrasound reduced symptoms and improved nerve conduction velocity compared with a placebo group. The ultrasound treatment was applied with the following parameters: frequency, 1.0 MHz; intensity, 1.0 W/cm^2; duty cycle, 25%; duration, 15 minutes. Ten sessions were applied at a frequency of 5 times per week for 2 weeks, followed by 10 additional sessions (twice a week for 5 weeks). The clinician should be aware that although this treatment has been shown to be effective in the treatment of CTS, more research is needed to determine its effectiveness in the treatment of PS and AINS.

Electrical stimulation is thought to reduce pain and promote nerve healing, based on the work of Al-Majed et al,[2] who used a rat model with cut and reapproximated femoral nerves. Low-frequency electrical stimulation was applied continuously for 1 hour directly to the proximal end of the cut peripheral nerve and regeneration occurred in 3 weeks versus 8 to 10 weeks without electrical stimulation. These results must be interpreted with due caution, as a rat

model was used and the nerve pathology was different than a nerve compression syndrome. A chronically compressed nerve, however, may undergo Wallerian degeneration (degeneration of the axon from the point of injury/compression distally) and may benefit from electrical stimulation's ability to accelerate nerve healing.

Iontophoresis is commonly used to reduce inflammation and neurologic literature does suggest that inflammation may be the primary cause of PS and AINS.[31] In a study by Banta[6] on the use of iontophoresis and wrist splinting on patients with median neuropathy at the wrist (ie, CTS), 58% of patients demonstrated a positive response to use of iontophoresis and neutral wrist splinting. The exact mechanism by which iontophoresis affects nerve healing is unknown, although it does appear to have some limited benefits.

Based on the available information on modalities, we would recommend a trial of ultrasound over the pronator teres, utilizing parameters similar to Ebenbichler et al.[10] The data on the effectiveness of other modalities, such as electrical stimulation and iontophoresis, are less conclusive.
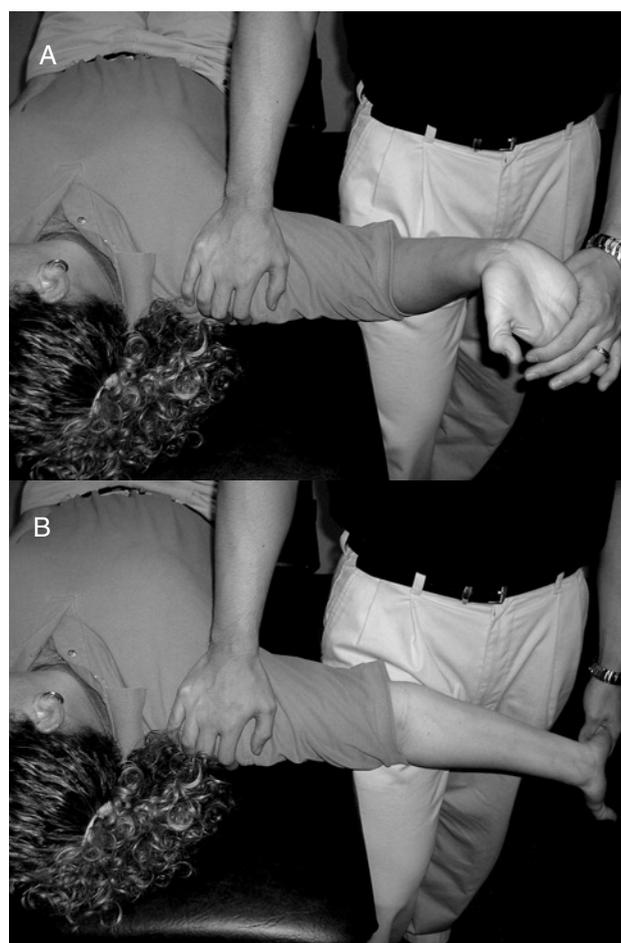
Nerve Gliding

Nerve gliding/mobilization (Figure 7) is a controversial treatment technique with study results ranging from positive to no effect. Rozmaryn et al[26] found that 43% of patients with CTS who performed a nerve-gliding home exercise program eventually underwent surgery versus 71.2% of patients treated with immobilization. Sweeny et al[30] reported that of 29 patients with mechanical allodynia (increased pain in response to a stimulus that is normally not painful) who performed a nerve-gliding program, 21% reported complete recovery, 45% reported improvement, and 34% reported no changes. In contrast to these studies, Scrimshaw and Maher[27] found no significant difference between neural mobilization and nonmobilization groups in 76 patients after spinal surgery. Although the results of these studies differ, the clinician should still be aware of the general physiological properties of peripheral nerves to perform neural mobilization exercises effectively. Attenuating a tensile load is not a characteristic function of nerve and, in fact, Liu et al[18] found that the ulnar nerve experienced histologic damage to axons and myelin sheaths at 4.2% elongation (12.7 kg/cm^2 stress) and tears in the perineurium at 6% elongation. Therefore, if a nerve compression exists, the clinician must consider whether the increased stress imparted to the nerve in an attempt to mobilize it is clinically beneficial or detrimental.

The clinician may also utilize soft tissue mobilization techniques to the area of suspected entrapment in an attempt to induce muscle relaxation and/or decrease muscle tension prior to nerve mobilization.

For example, with PS, the clinician can perform soft tissue mobilization to the pronator teres prior to performing median nerve mobilization. This may decrease the mechanical force imparted to the nerve at the area of entrapment and, therefore, decrease the probability of inducing histologic damage to the nerve during nerve mobilization exercises. To further induce muscle relaxation and/or decrease muscle tension prior to nerve mobilization, soft tissue techniques can be preceded with superficial heating modalities.

To mobilize the median nerve at the level of the pronator teres, the tension on the median nerve should be established proximally by side bending and rotating the cervical spine to the contralateral side and established distally by extending the wrist and fingers. The focus then is on gently mobilizing the median nerve by flexing and extending the elbow while supinating the forearm (Figure 7). Avoid any

FIGURE 7. Nerve gliding/mobilization. (A) To mobilize the median nerve at the level of the pronator teres, the tension on the median nerve should be established proximally by side bending and rotating the cervical spine to the contralateral side and established distally by extending the wrist and fingers. (B) The focus then is on gently mobilizing the median nerve by flexing and extending the elbow while supinating the forearm. Avoid any exacerbation of symptoms and do not proceed past the point of pain and symptom reproduction.

exacerbation of symptoms and do not proceed past the point of pain and symptom reproduction. As tolerance improves, the patient may be instructed in soft tissue and nerve mobilization techniques in a home exercise program. The home nerve mobilization technique is essentially identical to Figure 7; however, the patient performs the technique actively versus passively.

Other Options and Nonconservative Management

If conservative therapy is unsuccessful, cortisone injection into the region of the pronator teres has been suggested.[25,31] Generally, surgery is not indicated until after 8 to 12 weeks of conservative treatment with no significant change in symptoms. Surgical decompression of the median nerve involves decompression of all possible sites of nerve compression, as differentiation is difficult.[25] In general, most studies report 85% to 90% good to excellent outcomes following surgical decompression.[31]

CONCLUSION

PS and AINS are considered rare, especially in comparison to CTS. When conservative and, in some cases, surgical treatment of CTS are ineffective, other more proximal causes of median nerve compression must be considered. Differential diagnosis is sometimes difficult, as some symptoms are common to all 3 syndromes and electrodiagnostic studies (EMG and NCS) may be inconclusive in regards to PS. With an understanding of the relevant anatomy, clinical presentation, and objective tests, a differentiation between these nerve compression syndromes can at least be considered and investigated. Once a diagnosis of PS or AINS is established, intervention can be implemented. However, few controlled studies exist to establish the optimal intervention protocols. The literature does provide some data on effective treatment of peripheral nerve pathology in general; however, the direct mechanisms by which these treatments have their effect is unclear. Based on experience and available literature, the most effective conservative intervention program would include rest/immobilization, modalities, and gentle nerve-gliding/mobilization techniques.

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CLINICAL COMMENTARY