Anterior interosseous nerve syndrome (Kiloh-Nevin Syndrome) is the triad of weakness of the flexor pollicis longus, the flexor digitorum profundus of the index finger, and the pronator quadratus. It is a manifestation of neuropathy affecting either the anterior interosseous nerve itself (anterior interosseous neuropathy) or its fascicles more proximally within the median nerve or brachial plexus (pseudo–anterior interosseous neuropathy). Anterior interosseous neuropathy in the presence of normal anatomic variation of the anterior interosseous nerve must be distinguished clinically from pseudo–anterior interosseous neuropathy, which can present with telltale signs in addition to the signature weaknesses of anterior interosseous nerve syndrome. A history of penetrating injury mitigates toward early exploration and nerve repair. A history of sudden onset and rapid progression, particularly when accompanied by a prodrome of pain and fatigue, suggests the presence of a focal neuritis, which typically resolves completely within 6 to 12 months without surgical intervention. If no improvement is noted within 6 to 12 months or if the neurologic condition worsens, surgical exploration may be warranted. In the presence of untreatable injury to the anterior interosseous nerve, with permanent muscular atrophy, functional tendon transfers of the flexor digitorum superficialis of the ring or middle finger or of the brachioradialis may be helpful.

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band, thrombophlebitis, and antecubital vein catheterization or phlebotomy.

From the numerous reports supposing a broad range of causes—inflammatory, compressive, post-traumatic—it becomes clear that AIN syndrome refers not to a single pathologic entity, but to a common clinical manifestation of several different pathologies. These may or may not affect the AIN itself.

Much of the controversy surrounding the management of anterior interosseous nerve syndrome arises from subtle but important semantic differences between the terms anterior interosseous nerve syndrome and anterior interosseous neuropathy, which, unfortunately, have tended even among experienced hand surgeons to be used rather interchangeably. Many types of pathologies may result in the signature triad of palsies representing the syndrome.

Strictly speaking, anterior interosseous syndrome refers to that constellation of signs and symptoms referable to weakness of the pronator quadratus, the flexor pollicis longus, and the flexor digitorum profundus to the index finger. Although the AIN supplies sensory fibers to the radiocarpal, midcarpal, and carpometacarpal joints, AIN syndrome by definition refers to a purely motor constellation of signs and symptoms.

Although AIN syndrome is strictly motor, it may be associated with additional extrasyndromic signs and symptoms. Additional findings may suggest either that the underlying pathology resides outside of the AIN itself (median nerve or brachial plexus) or that aberrant anatomic features exist distal to the pathologic lesion in the AIN.

Among the many potential underlying pathologies manifesting as AIN syndrome are abnormalities either within or proximal to the AIN. Thus, causes of AIN syndrome are appropriately divided into 2 broad categories. Anterior interosseous neuropathies include those compression neuropathies, neuritides, congenital anomalies, and anatomic lesions and discontinuities of the AIN itself. Pseudo—anterior interosseous neuropathies, on the other hand, represent pathologies affecting more proximal anatomic sites, but involve nerve fascicles contributing to the anterior interosseous nerve more distally. Parsonage-Turner syndrome, in which AIN syndrome is associated with weakness of the parascapular muscles, is a classic example of the pseudo—anterior interosseous neuropathies.

ANATOMY

Just after coursing between the two heads of the pronator teres muscle, the median nerve gives rise to the AIN from its radial aspect. This take-off of the AIN from the median nerve occurs 5 to 8 cm distal to the lateral epicondyle and 22.4 to 23.4 cm proximal to the radial styloid. Coursing beneath the fibrous arch of the flexor digitorum superficialis muscle, the AIN then enters the flexor digitorum profundus muscle belly an average of 30% the forearm length distal to the medial epicondyle. The nerve then courses distally on the volar surface of the interosseous membrane. Approximately 4 cm distal to its takeoff from the median nerve, the AIN gives rise to motor branches to the flexor pollicis longus, the flexor digitorum profundus to the index finger, and, variably, the flexor digitorum profundus to the middle finger. It then supplies a motor branch to the pronator quadratus before terminating as sensory branches to the radiocarpal, midcarpal, and carpometacarpal joints.

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fibrous bands arising from the superficial head of the pronator teres, a nerve running deep to both heads of the pronator teres, and a double lacertus fibrosus.

**Clinical Presentation**

The only consistent finding in true AIN syndrome by definition is paresis or paralysis of the pronator quadratus, the flexor pollicis longus, and the flexor digitorum of the index finger. In addition, Hill et al have reported a series of 33 patients with incomplete AIN syndromes who presented with weakness of either the flexor pollicis longus or flexor digitorum profundus indicis only. Weakness may initially be noted as difficulty, fatigue, or clumsiness with writing or with fine pinch activities, such as sewing. Deterioration of handwriting has been described as a classic presentation in a series of patients with AIN syndrome.22

Although there is no consistent history of onset of neuropathy, there are several different patterns of onset that provide important clues for determining etiology. A history of trauma obviously suggests either mechanical disruption of the nerve, injury to the nerve, or compression neuropathy. Injuries associated with anterior interosseous neuropathy include penetrating trauma, blunt injury, and traction injury. The association between fractures of the supracondylar humerus and proximal forearm6–8 have classic associations with anterior interosseous syndrome.

With anterior interosseous or pseudo–anterior interosseous neuritides, onset of neurologic symptoms is typically sudden and rapidly progressive. Patients often relate an antecedent prodrome of proximal volar forearm or shoulder pain, and these patients are generally suspected of exhibiting an inflammatory neuropathy. Pain will often be of sudden onset and may be related to minor trauma. The prodrome may consist of systemic complaints, including generalized fatigue and fever. Hepatitis has been reported to be associated with acute brachial neuritis.23

**Examination**

AIN syndrome is suggested by the resting repose of the hand, which will exhibit an unnatural extension of the distal interphalangeal (DIP) joint of the index finger and interphalangeal (IP) joint of the thumb, compared with the gentle flexion arcade of the remaining fingers. The metacarpophalangeal joint of the thumb may compensate by assuming hyperflexion.

The signature finding on physical examination is weakness of the flexor pollicis longus, flexor digitorum profundus indicis, and pronator quadratus. Weakness of the flexor pollicis longus and flexor digitorum profundus to the index finger is indicated by an inability to make the “OK” sign. Rather, the DIP joint of the index finger and the IP joint of the thumb are hyperextended during attempted tip-to-tip pinch. The area of contact between the thumb and index finger is a flatter, broader area found more proximally. The “OK” sign on the affected hand has more the appearance of a Playboy bunny (Fig 1). In addition, Spinner has described a sign that, for all his contributions to our understanding of anterior interosseous syndrome, should rightly bear his name: upon making a fist, the tips of the index finger and thumb remain conspicuously excluded (Fig 2).

Examination of the pronator quadratus is difficult and unreliable. Theoretically, contributions to resisted forearm pronation from the pronator quadratus and from the pronator teres are distinguishable. With the elbow bent at 90°, the patient is asked to forcibly pronate the forearm, against the resistance of the examiner. Flexion of the elbow at 90° delivers the pronator teres, which originates from the medial humerus, out of its optimal sarcomere length on the Starling length:tension curve, thus isolating contributions from the pronator quadratus distally. However,

**Table 1**

<table>
<thead>
<tr>
<th>Anatomic Features Predisposing Toward Anterior Interosseous Compression Neuropathy</th>
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<tbody>
<tr>
<td>Tendinous origin of the deep head of the pronator teres</td>
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<tr>
<td>Tendinous origin of the flexor superficialis to the long finger</td>
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<tr>
<td>Tendinous origin of variant muscles (palmaris profundus, flexor carpi radialis brevis)</td>
</tr>
<tr>
<td>Thrombosis of crossing ulnar collateral vessels</td>
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<tr>
<td>Accessory muscle and tendon from the flexor digitorum superficialis to the flexor pollicis longus</td>
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<tr>
<td>Gantzer’s muscle (accessory head of the flexor pollicis longus)</td>
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<tr>
<td>Aberrant radial artery</td>
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<tr>
<td>Enlarged bicipital bursa near the origin of the AIN</td>
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</table>

Data from Spinner.24
Stewart finds this maneuver to be unreliable and states rather that “if weakness of pronation is detected, the pronator teres is involved; this indicates a lesion of the main trunk of the median nerve or an anatomic anomaly with the pronator teres being innervated by the AIN.”

The AIN provides no sensory fibers to the skin. Therefore, an abnormal sensibility testing result definitively rules out an anterior interosseous neuropathy. Abnormal sensibility in the median distribution in the presence of an anterior interosseous syndrome strongly suggests a proximal median compression neuropathy involving fascicles of the AIN.

Abnormalities of other typically median nerve functions, such as flexor digitorum superficialis activity, do not necessarily exclude a diagnosis of anterior interosseous neuropathy. As stated above, the pronator teres may be aberrantly innervated by the AIN, as may the flexor digitorum superficialis.

Careful objective testing of the parascapular muscles can reveal subtle weakness of the shoulder girdle, a finding that also would argue strongly against a diagnosis of anterior interosseous neuropathy. Additional testing to exclude Parsonage-Turner syndrome, acute brachial neuritides, or other proximal pseudo—anterior interosseous neuropathies is clinically prudent in any patient with anterior interosseous syndrome.

**Variants**

In the presence of anterior interosseous syndrome, additional findings or symptoms can provide important clues to determining whether the pathology resides within or proximal to the AIN. This is an important distinction clinically, because certain neuropathies proximal to the anterior interosseous nerve—for example, acute brachial neuropathy—tend to be self-limited. On the other hand, because of certain well-described variations in forearm innervation, specific clinical findings in addition to anterior interosseous syndrome itself are completely consistent with a true anterior interosseous neuropathy and do not imply pathology more proximally.

A meticulous physical examination and a knowledge of important anatomic variants can allow the clinician to distinguish a pseudo—anterior interosseous neuropathy from a true anterior interosseous neuropathy. Some additional features known to be associated

![FIGURE 1. Playboy bunny sign. Classic repose of a left hand affected by AIN syndrome. Note the extension of the index finger DIP joint and thumb IP joint. The contact point between the thumb and index finger has migrated more proximally and has become a broad surface. Instead of the “OK” sign formed by the unaffected right side, the thumb and index finger of the left hand form the elongated nose of a Playboy bunny, and the gently flexed middle and ring fingers form floppy bunny ears.](image)

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![FIGURE 2. Spinner's sign. The patient is asked to make a fist. Typically the tips of the small, ring, and middle fingers are able to achieve flexion to the distal palmar crease. However, the tips of the index finger is conspicuously excluded. The thumb remains straight. This patient has a partial AIN syndrome, because he has sufficient strength of the flexor digitorum profundus of the index finger to cause some active flexion of its DIP joint.](image)
with anterior interosseous neuropathy stand in notable contradistinction to specific findings that exclude its diagnosis (Fig 3).

**Weakness of the Deep Flexor of the Middle Finger**

According to Sunderland’s classic studies on upper extremity innervation, “the portion of the flexor digitorum profundus serving the index finger is the only part of this muscle that is exclusively and constantly supplied by the median nerve.” The deep flexors to the small and ring fingers are consistently innervated by the ulnar nerve. Deep flexors to the ring and middle fingers receive variable contributions from the AIN and ulnar nerve. Recently, Bhadra and coworkers performed cadaver dissections confirming this: in only 5% of forearms did the AIN supply the deep flexor to the index finger alone. In 75% of forearms, the AIN supplied the index and middle fingers, and the ulnar nerve supplied the deep flexors to the small, ring, and middle fingers. In 20% of forearms, the AIN exclusively innervated the deep flexors to the index and middle fingers. Consequently, with a lesion of the anterior interosseous nerve, there may be some weakness of the flexor digitorum profundus to the middle finger, as well as to the deep flexors to the thumb and index finger.

**Martin-Gruber Communications**

Martin-Gruber communications between the ulnar and median nerves in the proximal forearm are present in approximately 15% of upper limbs. Motor fibers targeted for typically ulnarly innervated muscles of the hand (most notably the adductor pollicis, abductor digiti quinti, and first dorsal interosseous, as well as the second and third dorsal interossei) may be carried temporarily in the median nerve. Therefore, when afflicted with either a neuritis or compression neuropathy of the AIN, patients with a Martin-Gruber communicating branch may exhibit weakness or paralysis of these intrinsic muscles in addition to signature weakness of the flexor pollicis longus, pronator quadratus, and flexor digitorum profundus of the index finger. Conversely, in the rare patient with ulnar innervation of the flexor digitorum profundus of the index finger, an anterior interosseous neuropathy may manifest as isolated weakness of thumb IP flexion and forearm pronation.

**Involvement of the Flexor Digitorum Superficialis**

In approximately 30% of forearms, the flexor digitorum superficialis is supplied by a separate branch deriving from the AIN. Therefore, weakness of flexion of all of the proximal IP joints (with blockade of profundus contributions) is possible with an anterior interosseous neuropathy. Even with anterior interosseus neuropathy versus pseudoneuropathy. Findings in addition to the classic triad may indicate that the lesion resides proximal to the AIN. However, certain anatomic variants allow for additional features in the presence of a true anterior interosseous neuropathy.

**Anterior Interosseous Nerve Syndrome**

**Anterior Interosseous Neuropathy**
- Lesion of the AIN
- Weakness of FPL, PQ, FDP index
- May have weakness of intrinsics, middle finger FDP, and even FDS
- Normal sensibility
- Normal shoulder girdle

**Pseudo-Anterior Interosseous Neuropathy**
- Lesion of fibers that ultimately constitute the AIN
  - Weakness of FPL, PQ, FDP index (may be only finding)
  - May have weakness of intrinsics, middle finger FDP, and even FDS
  - +/- Weakness of shoulder girdle
  - +/- Weakness of thenar muscles
  - +/- Weakness of other muscles
  - +/- Abnormal sensibility (median)
ous innervation of the superficial flexors, however, functionally significant weakness of all fingers is extremely rare because of the continued activity of deep flexors to the small, ring, and—generally—middle fingers.

Shoulder Girdle Weakness

Parsonage and Turner\(^3\) were the first to describe anterior interosseous syndrome arising from a diffuse neuritis, presumably of anterior horn cells. In such cases, weakness of the deep flexors to the thumb and index finger is associated with varying degrees of weakness of the scapular muscles (Parsonage-Turner syndrome) and usually is preceded or accompanied by pain.\(^23\)

Etiologies

The key to treatment of anterior interosseous syndrome is the recognition of a variety of potential anatomic, inflammatory, infectious, posttraumatic, and compressive causes (Table 2). These may affect either the AIN itself (anterior interosseous neuropathy) or the median nerve or brachial plexus more proximally (pseudo—anterior interosseous neuropathy).\(^13,25,28\) Borchardt and Wjasmsenski\(^28\) described a similar clinical weakness of the deep flexors of the thumb and index finger after penetrating wounds of the upper forearm. Kiloh and Nevin\(^2\) reported 2 cases of acute interstitial neuritis of the AIN, manifesting with a similar pattern of thumb/index long flexor weakness, identifying an inflammatory etiology of anterior interosseous syndrome. Parsonage and Turner\(^3\) described an acute brachial plexus neuritis manifesting as AIN syndrome in association with weakness of the shoulder girdle.

Gunther and colleagues\(^18\) have aptly shown that, at the level of the elbow, motor fibers of the median nerve destined to become the AIN lie posteriorly with respect to the main nerve trunk. Hence, post-traumatic median neuropathy after supracondylar humerus fractures or proximal radius fractures may result in a pattern of clinical weakness representative of a pseudo—anterior interosseous neuropathy. In one series of supracondylar humerus fractures associated with anterior interosseous syndrome, Spinner\(^24\) reviewed patterns of fracture displacement. He concluded that prerequisites for nerve injury were a distal point of fixation of the anterior interosseous nerve and posterior displacement of the distal fragment. In such cases, the median nerve slipped between the fracture fragments; isolated injury to the AIN itself was not noted.

Differential Diagnosis

Anterior interosseous neuropathy must also be distinguished from tendon rupture. The latter is easily excluded from the diagnosis by careful examination for tenodesis: with intact flexor tendons, passive wrist extension should effect passive thumb and finger IP joint flexion. In addition, tendon rupture should be suspected in patients with rheumatoid arthritis and Kienböck disease.\(^14\) In the former instance, attritional rupture may occur as a result of volar carpal subluxation. In the latter case, proximal carpal pathology may lead similarly to attritional tendon rupture. Tendon rupture secondary to scaphoid nonunion also has been described.\(^29,30\) In the above instances of secondary tendon rupture, limitation of passive extension of the wrist due to underlying wrist pathology may make it difficult to exclude tendon rupture on the basis of an observed tenodesis effect.

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**TABLE 2**

<table>
<thead>
<tr>
<th>Causes of Anterior Interosseous Syndrome</th>
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<tr>
<td><strong>Anterior interosseous neuropathies</strong></td>
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<tr>
<td>Direct injury to nerve</td>
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<td>Midshaft radius fracture</td>
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<tr>
<td>Compression</td>
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<tr>
<td>Fibrous bands (pronator, FDS)</td>
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<tr>
<td>Enlarged median artery</td>
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<td>Hematoma</td>
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<tr>
<td>Trauma</td>
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<tr>
<td>Coagulopathy</td>
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<tr>
<td>Tumor</td>
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<tr>
<td>Idiopathic inflammatory anterior interosseous neuropathy</td>
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<tr>
<td>Pseudo–anterior interosseous neuropathies</td>
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<tr>
<td>Supracondylar humerus fracture</td>
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<tr>
<td>Proximal radius fracture</td>
</tr>
<tr>
<td>Antebrachial venipuncture or catheterization</td>
</tr>
<tr>
<td>Inflammatory</td>
</tr>
<tr>
<td>Acute brachial neuropathy</td>
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<tr>
<td>Parsonage-Turner syndrome</td>
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</tbody>
</table>

Abbreviation: FDS, flexor digitorum superficialis. Data from Spinner.\(^24\)
Electrodiagnostic Studies

Hill et al. have described 33 cases of incomplete AIN syndrome in which either the flexor pollicis longus or the flexor digitorum profundus to the index finger was paretic or paralyzed. Because of the above types of variations in forearm muscle innervation and because it is possible to exhibit an incomplete anterior interosseous neuropathy, it is prudent to sample electromyographically all muscles typically innervated by the AIN. Electrodiagnostic studies at a minimum should include electromyography of the flexor pollicis longus, pronator quadratus, and flexor digitorum profundus indicis. Because the pronator teres is typically innervated by the median nerve, electromyographic testing of the pronator teres should logically distinguish anterior interosseous neuropathy from proximal compression of the median nerve affecting fascicles of the AIN. However, Ashworth et al. have described a case in which compression of the AIN by a fibrous band arising from the deep head of the pronator teres resulted in weakness and denervation of the pronator teres. On exploration, the AIN was found to innervate the pronator teres.

Electromyographic studies may be most helpful after a history of trauma, particularly blunt injury. Complete lesions are probably most amenable to immediate exploration, whereas surgical exploration of incomplete lesions may be deferred for several months. As with most other neuropathies, the presence of positive sharp waves or fibrillation potentials indicate nerve degeneration and may provide an indication for surgical exploration.

North and Kaul suggest that any patient suspected of having AIN syndrome should receive electromyographic studies of the shoulder girdle to rule out neuralgic amyotrophy. Among patients manifesting signs and symptoms suggestive of anterior interosseous-like neuropathy, Parsonage-Turner syndrome is found with relatively high frequency.

Treatment

Review of the literature shows considerable controversy surrounding the treatment of AIN syndrome. Sunderland reported that when fibrous bands constricting the AIN were found at operation, resection of these bands followed by anterior interosseous neurolysis resulted in motor recovery in every case. Stern discussed 3 cases of AIN syndrome due to proximal median compression neuropathy. These proved to be uniformly unresponsive to conservative management, but rapid and long-term recovery occurred in all cases managed surgically. On the other hand, Miller-Breslow et al. reported their results of nonoperative management in 10 patients with spontaneous AIN paralysis. Eight patients recovered fully within 1 year. However, all of their patients reported a prodrome of forearm pain and probably suffered from anterior interosseous neuritis rather than compression neuropathy. Hence, controversy relating the surgical versus nonoperative treatment of AIN syndrome may in large part be due to the broad range of pathologies resulting in a common triad of findings.

Therefore, the probability and extent of motor recovery from AIN syndrome is predicated on the establishment of a correct and precise diagnosis. One must recognize that AIN syndrome represents a constellation of findings and does not refer to a single distinct pathology. A thorough and directed neurologic examination in combination with a thorough history and well-considered electrodiagnostic study will distinguish pseudo-anterior interosseous neuropathies from focal lesions of the AIN itself.

Once an accurate diagnosis is established, etiologic factors must guide therapy. A history of penetrating trauma suggests mechanical disruption or compression of the nerve and mitigates strongly toward surgical exploration and nerve decompression or repair. In the presence of blunt trauma, management of anterior interosseous palsy is less straightforward. Electromyography suggestive of a complete lesion may mandate early surgical exploration. Partial injuries may be given an opportunity to recover spontaneously. If no improvement is noted either clinically or electromyographically after 6 to 12 weeks, surgical exploration may be warranted. If positive sharp waves or fibrillation potentials are present on electromyography, surgical exploration and neurolysis may be indicated.

Spontaneous or rapid onset of weakness is suggestive of an anterior interosseous neuritis or pseudo-anterior interosseous neuritis. In such cases, neurologic symptoms are typically preceded by proximal anterior forearm or shoulder pain and tenderness. Pain generally has subsided by the time weakness of pinch
A history of fatigue, fever, myalgia, or other prodromic systemic symptoms also strongly suggests an inflammatory process, which may resolve spontaneously or with systemic steroids. However, to date there have been no persuasive studies showing that the use of systemic steroids has any effect on the rate, extent, or probability of motor recovery from AIN syndrome. As a general rule, Spinner treatments spontaneously paralysis of the AIN nonsurgically initially but recommends surgical exploration within 12 weeks if no clinical or electromyographic improvement is evident. However, spontaneous recovery after 12 months is well-documented, and some have considered waiting at least this long for spontaneous recovery before proceeding with surgical exploration. Miller-Breslow et al followed up 10 patients with spontaneous anterior interosseous nerve paralysis, all of whom related an initial history of pain. Eight patients recovered fully within 1 year. On the basis of their experience, Miller-Breslow et al advocated nonsurgical management extending beyond 1 year.

Should the AIN fail to recover, should it be unreconstructible, or should there be irreversible muscular atrophy after a prolonged period of denervation, tendon transfers offer an acceptable means of functional reconstruction of the thumb and index finger. In such cases, Spinner advocates transfer of a slip of flexor digitorum superficialis tendon from the ring or long finger to the tendon of either the flexor pollicis longus or the flexor digitorum profundus indicis distally. Obviously, this is possible only if the median nerve itself, with its innervation of the flexor digitorum superficialis, is unaffected. Transfers of the brachioradialis to the flexor pollicis longus or the extensor carpi radialis longus to the flexor digitorum profundus of the index finger are also acceptable alternatives for functional restoration.

**SUMMARY**

AIN syndrome consists of a triad of clinical findings: weakness of the pronator quadratus, flexor pollicis longus, and flexor digitorum profundus of the index finger. It is important to recognize that this clinical constellation of findings does not refer to a single distinct pathology. An understanding of common variants of forearm innervation combined with a thorough physical examination for accompanying features can provide important clues as to whether pathology resides within (anterior interosseous neuropathy) or proximal to (pseudo–anterior interosseous neuropathy) the AIN. Proper treatment is predicated on a precise and accurate diagnosis.

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