Review Article

Pronator Syndrome and Anterior Interosseous Nerve Syndrome

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Abstract

Dysfunction of the median nerve at the elbow or proximal forearm can characterize two distinct clinical entities: pronator syndrome (PS) or anterior interosseous nerve (AIN) syndrome. PS is characterized by vague volar forearm pain, with median nerve paresthesias and minimal motor findings. AIN syndrome is a pure motor palsy of any or all of the muscles innervated by that nerve: the flexor pollicis longus, the flexor digitorum profundus of the index and middle fingers, and the pronator quadratus. The sites of anatomic compression are essentially the same for both disorders. Typically, the findings of electrodiagnostic studies are normal in patients with PS and abnormal in those with AIN syndrome. PS is a controversial diagnosis and is typically treated nonsurgically. AIN syndrome is increasingly thought to be neuritis and it often resolves spontaneously following prolonged observation. Surgical indications for nerve decompression include persistent symptoms for >6 months in patients with PS or for a minimum of 12 months with no signs of motor improvement in those with AIN syndrome.

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Pronator syndrome (PS) and anterior interosseous nerve (AIN) syndrome are proximal median neuropathies with different presentations. PS is characterized by vague volar forearm pain associated with median nerve paresthesias, whereas AIN syndrome is a motor palsy of any or all of the muscles innervated by that nerve. Surgical decompression of the median nerve or the AIN in the forearm is rarely indicated; a prolonged nonsurgical approach is warranted in most cases.

Anatomy

The median nerve receives contributions from the roots of the fifth, sixth, seventh, and eighth cervical nerves as well as the first thoracic nerve. The medial and lateral cords of the brachial plexus converge to form the median nerve, which begins anterolateral to the brachial artery. The median nerve then crosses from lateral to medial over the brachial artery and courses between the biceps brachii and brachialis muscles. Typically, the nerve has no muscle branches above the elbow; however, there may be a variable branch innervating the pronator teres (PT) muscle. The median nerve then passes deep to the ligament of Struthers (if present) and continues into the antecubital region (Figure 1). The ligament of Struthers is an anatomic variant that extends from a small, supracondylar process on the humeral shaft to the medial epicondyle of the humerus; it is present in 1% to 2% of the population.[1-3]

As the median nerve traverses the elbow, it passes under the bicipital

aponeurosis (lacertus fibrosus) and into the antecubital fossa (Figure 2). The nerve remains medial to the biceps tendon and brachial artery and volar to the brachialis muscle and its insertion. The median nerve then travels between the two heads of the PT muscle and deep to the proximal fibrous arch of the flexor digitorum superficialis (FDS), continuing distally through the forearm between the FDS and flexor digitorum profundus (FDP) (Figure 3).

Distal to the elbow, the median nerve provides motor innervation to the PT, FDS, palmaris longus, and flexor carpi radialis (FCR). The nerve gives off two main branches (the AIN and the palmar cutaneous branch of the median nerve [PCBMN]) before continuing through the carpal tunnel at the wrist. The AIN arises just inferior to the elbow from the dorsoradial aspect of the median nerve. The branch lies approximately 4 cm distal to the medial epicondyle and 5 to 8 cm distal to the lateral epicondyle (Figure 4). The AIN runs distally along the FDP, passing between the FDP and the flexor pollicis longus (FPL) and then coursing along the anterior interosseous membrane with the anterior interosseous artery. Distally, the AIN innervates the pronator quadratus (PQ) muscle before terminating in the wrist capsule, providing innervation to the wrist joint. The PCBMN branches from the radial aspect of the median nerve approximately 5 cm proximal to the distal flexion crease at the wrist and provides sensory innervation to the thenar aspect of the palm.[4]

Figure 1

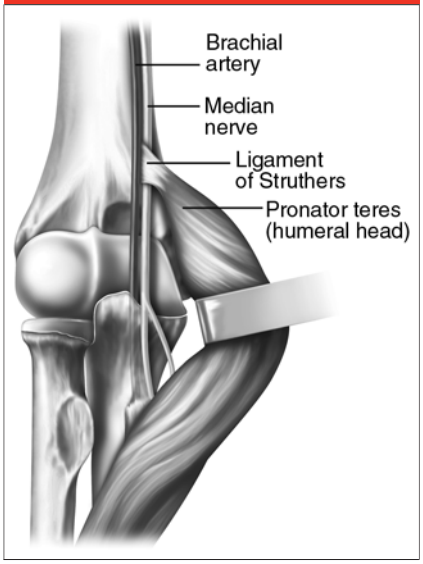


Illustration demonstrating the course of the median nerve superior to the elbow joint as the nerve travels medial to the brachial artery. The artery may be superficial to the ligament of Struthers (as shown here) or may accompany the median nerve deep to it.

Figure 2

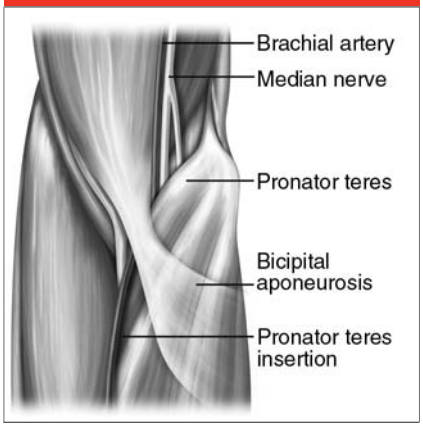


Illustration demonstrating the course of the median nerve as it enters the forearm. The nerve runs deep to the bicipital aponeurosis and medial to both the biceps tendon and brachial artery.

Figure 3

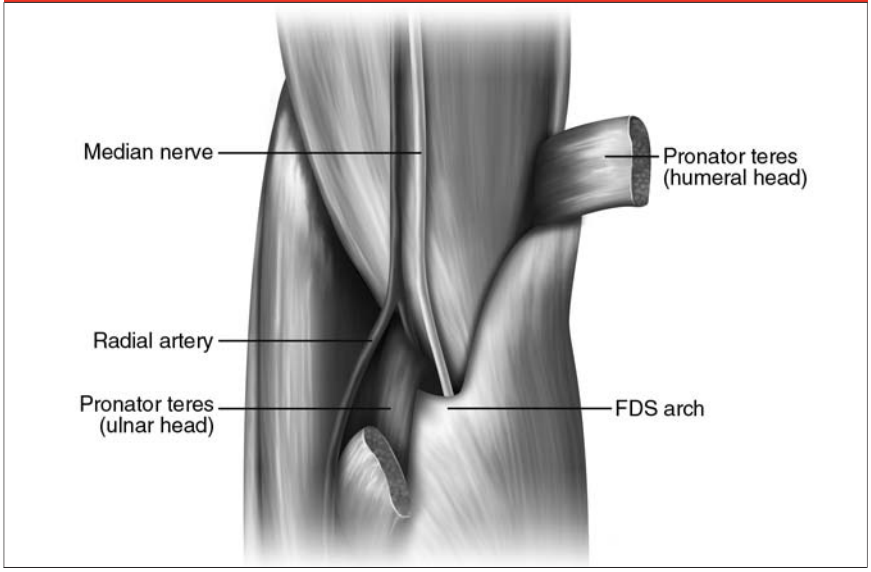


Illustration demonstrating the course of the median nerve as it passes beneath the proximal fibrous arch of the flexor digitorum superficialis (FDS) in the forearm.

Pronator Syndrome

Originally described by Seyffarth[5] in 1951, classic PS refers to compression of the median nerve as it passes between the two heads of the PT muscle or at the proximal arch of the FDS. Despite its name, this syndrome is a proximal median nerve dysfunction that may also result from compression of the nerve at the ligament of Struthers, the bicipital aponeurosis (lacertus fibrosus), or an accessory head of the FPL (ie, the Gantzer muscle).[6-10] Entrapment at any of these anatomic sites may produce the constellation of symptoms that characterize PS. Because the PS diagnosis is rare, its incidence and prevalence has not been firmly established.

Clinical Manifestations

Patients with PS may initially present with some of the same symptoms associated with carpal tunnel syndrome (CTS). Patients with PS or CTS may report numbness or paresthesias in the radial three and one half digits and pain in the forearm and volar wrist.[1,6,10-12] Because both syndromes are the result of median nerve dysfunction, the distal symptoms are similar.

PS can be distinguished from CTS by the presence of numbness, paresthesias, or both in the distribution of the PCBMN and the absence of findings with provocative testing for CTS. The PCBMN branches off the median nerve proximal to the carpal tunnel and traverses superficial to the transverse carpal ligament; thus, it is not affected by compression within the carpal tunnel.[4] Patients with PS may report decreased sensation in the PCBMN distribution over the thenar eminence.

Figure 4

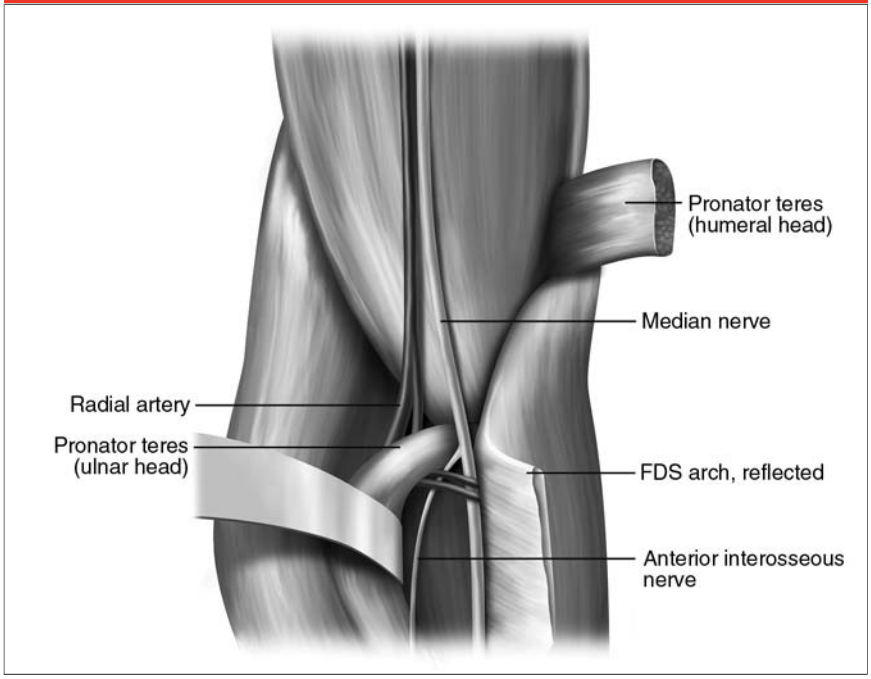


Illustration demonstrating the course of the anterior interosseous nerve as it branches off of the main trunk of the median nerve radially and deep to the flexor digitorum superficialis (FDS).

Diagnosis

The differential diagnosis for PS should include more proximal neuropathies (compression at the cervical spine or brachial plexus), AIN syndrome (if motor function is affected), and CTS. PS can be distinguished from CTS on physical examination. Although the precise anatomic area of compression is difficult to determine on examination, compression at the carpal tunnel can be distinguished from a more proximal etiology. If the site of nerve dysfunction is proximal to the carpal tunnel, provocative maneuvers that result in increased pressure within the carpal tunnel should not exacerbate symptoms. Thus, patients with isolated PS should not have a Tinel sign (ie, tingling elicited by light tapping over the nerve) at the wrist, and symptoms should not be provoked with prolonged wrist flexion. Patients with PS typically do not report waking at night with pain or paresthesias, symptoms frequently associated with CTS.[6,11,13,14]

Three main maneuvers are performed during the physical examination to evaluate for PS. The first is the pronator compression test; it is performed by applying pressure proximal and lateral to the proximal edge of the PT muscle belly on the volar forearm. A positive test is the most common sign of PS,[11,13,14] reproducing pain or paresthesias within 30 seconds of compression.[15] Resisted pronation and supination are tested to determine whether these can reproduce symptoms caused by median nerve compression by the PT or the lacertus fibrosus. The third test, resisted flexion of the proximal interphalangeal joint of the middle finger, may cause pain and paresthesias in patients with PS because the median nerve is compressed by the heads of the FDS; however, this finding may also be seen in those with CTS.[16] In addition to these maneuvers, a positive Tinel sign over the proximal volar forearm has also been reported to be indicative of PS.[13-15]

Physical examination findings can be subtle in patients with PS. It remains unclear whether electromyography (EMG) and nerve conduction velocity (NCV) studies can aid in diagnosis. Several studies have reported that results of nerve testing in patients with PS are predominantly normal, with abnormalities reported in 7% to 31% of patients treated surgically.[13,14,17] However, in a study of 13 patients with a clinical diagnosis of PS, Lee et al[11] found that all patients had abnormal electrodiagnostic studies, an unusual finding in the PS literature. Typically, we do not recommend EMG/NCV for the diagnosis of PS; however, electrodi-

agnostic testing may be helpful to rule out other sites of compression in patients with distal sensory symptoms.

A radiographic series of the elbow should be obtained initially to evaluate for bony pathology; the presence of a supracondylar process can indicate the presence of a ligament of Struthers.[2] To our knowledge, no studies have evaluated the efficacy of MRI or ultrasound for the diagnosis of PS.

It should be emphasized that PS is a controversial diagnosis among many orthopaedic surgeons. Patients who present with aching proximal forearm pain, occasional paresthesias, and symptoms that are not reliably reproduced by clinical examination represent a puzzling clinical presentation that may not necessarily merit a more specific label or diagnosis. Therefore, a diagnosis of nonspecific arm pain is preferable to a diagnosis of tendinitis or PS if consistent evidence is lacking.[18]

Nonsurgical Management

Evidence regarding treatment outcomes in patients with PS is limited. The few small studies that follow nonsurgical cases have reported that from 29% to 100% of patients improve with nonsurgical treatment; however, no randomized controlled studies have been performed.[5,13,19] In 1951, Seyffarth[5] first described PS in a series of 17 patients, who all improved after injection of local anesthetic. In a retrospective review of seven patients with PS, Morris and Peters[19] found that corticosteroid injection into the PT muscle combined with activity modification resulted in subjective improvement of symptoms in five of the seven patients. In a study of 39 patients with PS, 7 were treated nonsurgically.[13] Follow-up ranged from 5 to 72 months. The authors reported improvement in two patients, no change in four patients, and worsened symptoms in one patient.

No studies have compared the duration or effectiveness of nonsurgical modalities for management of PS. Because high-level clinical outcome data are lacking, we recommend an initial nonsurgical approach to management initially. Although some authors suggest a period of immobilization, we recommend a regimen that includes rest, forearm flexor muscle stretching exercises, activity modification, and a course of anti-inflammatory medication for a minimum of 6 months.[20]

Surgical Management

Like its diagnosis, surgical management of PS is controversial. Consensus is lacking with regard to the duration of nonsurgical management required before a surgical release is attempted. No controlled trials and few outcome studies have evaluated the effectiveness of surgical treatment. In fact, small case series and technique papers comprise most of the literature.

Various surgical approaches have been described, including oblique,[14] transverse,[21] and lazy S-shaped incisions.[20,22] Although consensus on the optimal surgical technique for surgical management of PS is lacking, most authors have traditionally recommended a complete decompression of the median nerve throughout its course in the proximal forearm. Recently described techniques include an endoscopic-assisted release,[11] an open technique in which only the lacertus fibrosus is released,[7] and a minimally invasive approach[17] in which only the deep fascia of the superficial head of the PT is released. After surgery, we encourage active range of motion as early as postoperative day 2, with full return to activity by 6 to 8 weeks.

Several retrospective studies have reported on the outcomes of surgical management of PS. In a study of 36 forearms (32 patients) with PS treated with surgical decompression, Hartz et al[13] reported complete resolution of symptoms in 8 forearms, with reduced symptoms in 20, persistent symptoms in 5, and unchanged symptoms in 3. Follow-up ranged from 3 to 88 months. Patients with reduced symptoms were able to perform activities that had previously been limited. Patients with persistent symptoms could return to part-time work only. Interestingly, seven of the patients underwent carpal tunnel release secondary to misdiagnosis. Olehnik et al[14] reported on a series of 39 forearms (36 patients) treated with surgical decompression of the median nerve; 30 forearms had complete or partial relief of symptoms, and 9 had no improvement. Other retrospective studies have reported similar results.[6,15] The weaknesses of these studies include their retrospective nature, lack of a control group, no clear inclusion and/or exclusion criteria, and limited use of validated outcome measures.

In a recent retrospective study by Lee et al,[11] 13 patients with PS underwent surgical treatment with an endoscopically assisted technique after failing a 3- to 8-month trial of nonsurgical treatment. All patients had improvement in subjective symptoms and Disabilities of the Arm, Shoulder, and Hand scores at 11 to 37 months after decompression. Eight patients had complete or near complete resolution of symptoms, two had occasional mild symptoms, and three had only partial relief of symptoms and residual forearm pain. Although most patients reportedly did well, the preoperative and postoperative Disabilities of the Arm, Shoulder, and Hand scores were obtained retrospectively and, consequently, are susceptible to recall bias.

Anterior Interosseous Nerve Syndrome

As described previously, the AIN innervates the deep muscles of the forearm, including the FPL, the FDP to the index (FDP1) and middle (FDP2) fingers, and the PQ muscle. AIN syndrome is typically characterized by forearm pain and partial or complete dysfunction of the AIN-innervated muscles; the exact etiology and pathophysiology of the disorder remain unclear. Although nerve compression at sites similar to those associated with PS may contribute to AIN syndrome, it is most commonly the result of neuritis.

In 1948, Parsonage and Turner[23] reported on several cases of isolated AIN palsy caused by neuralgic amyotrophy (ie, Parsonage-Turner syndrome [PTS] or brachial plexus neuritis). Later reports included a description of a self-limited loss of function that involved only the AIN distribution.[24] More recent studies of AIN syndrome suggest that its etiology is typically a transient neuritis[25,26] and is likely related to PTS. PTS presentation varies; symptoms may include pain and motor and/or sensory dysfunction in one or in multiple peripheral nerves of the upper extremity, including isolated AIN palsy.[27,28] The etiology of PTS remains unknown but appears to be immune mediated.[27] Possible triggers may include viral illness (25% to 55%), immunizations (15%), perioperative and peripartum periods (>14%), and strenuous exercise (8%).[27,28]

Case reports describing AIN syndrome as the result of compression that was discovered intraoperatively should be interpreted carefully. For example, in one case report of an acute AIN palsy following laparotomy, the authors found that the Gantzer muscle, which was found during surgical exploration, was the cause of symptoms.[29] However, the patient's presentation could be consistent with the type of precipitating event associated with PTS. Because the Gantzer muscle is a common finding (in 50% of cadaver samples[8]) and AIN syndrome is relatively rare, this case report may not represent a causal association.

Clinical Manifestations

Typically, patients with AIN syndrome present with vague forearm pain and spontaneous weakness or complete absence of FPL function. FDP1 and FDP2 function is also generally affected, although FDP2 function may be preserved because of cross innervation by the ulnar nerve. Nerve anomalies, such as Martin-Gruber anastomosis (ie, a nerve branch connecting the median and ulnar nerves in the forearm), are present in 10% to 15% of the population.[9] Because the AIN does not provide sensory innervation to the skin, sensory deficits do not occur with an isolated AIN palsy; however, altered sensation may be seen if other nerves are involved in the setting of PTS.

Diagnosis

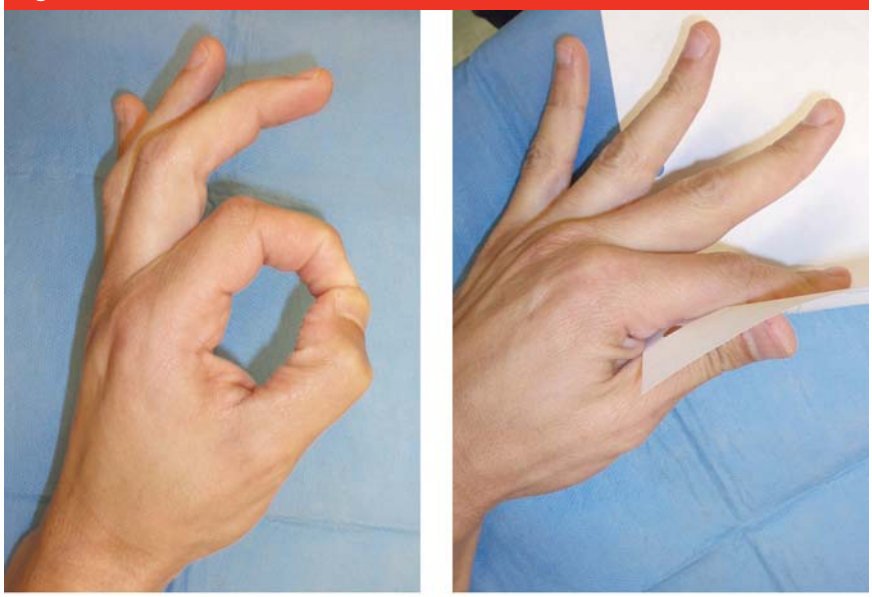
Patients' initial complaints may include forearm pain and loss of a pinch grip. The Kiloh-Nevin sign is a characteristic physical examination finding of AIN syndrome; the patient is unable to make an "OK" sign with the thumb and index finger. The inability to flex the thumb interphalangeal (IP) joint and the distal interphalangeal (DIP) joint of the index finger indicates a weakness or complete loss of function of the FPL and FDP1. The weakness may be subtle and can be further evaluated by having the patient pinch a sheet of paper between the thumb and index finger using only the fingertips. The examiner then slowly pulls the paper away; a patient with AIN syndrome may compensate for FPL and FDP1 weakness by using the intrinsic muscles innervated by the ulnar nerve rather than using a pinch grip (Figure 5).

The differential diagnosis of AIN syndrome includes trauma, tendon rupture, proximal sites of nerve compression (eg, cervical nerves, brachial plexus), thoracic outlet syndrome, PS, and CTS. Mechanical etiology must be ruled out prior to diagnosis of AIN syndrome. For example, an FPL tendon rupture can mimic a complete FPL palsy in a patient with AIN syndrome. To exclude tendon rupture, the examiner can evaluate the tenodesis effect. If the tendons are intact and the wrist is passively extended, the thumb IP joint and DIP joint of the index finger assume a flexed position; these joints extend when the wrist is passively flexed.

Electrodiagnostic studies serve an important role in the workup for AIN syndrome; these studies can support the diagnosis. The affected muscles may exhibit fibrillations, sharp waves, abnormal latency, and abnormal compound motor action potentials on EMG/NCV testing.[25] Nerve testing may also help rule out other lesions as a cause of symptoms. For example, in a patient with rheumatoid arthritis, it may be difficult to assess an intact tendodesis effect secondary to limited wrist motion. In this case, EMG can help the orthopaedic surgeon distinguish AIN syndrome from an attritional flexor tendon rupture.

Typically, imaging is not helpful for diagnosis and subsequent management of AIN syndrome. Radiographic findings are usually normal. MRI may show edema in the AIN-innervated muscles with either a surgically observed site of compression or a brachial neuritis.[30] However, unless a mass or tendon rupture is

Figure 5



A B

Clinical photographs of the hand demonstrating normal (**A**) flexion of the interphalangeal (IP) joint of the thumb and the distal interphalangeal (DIP) joint of the index finger, and an example of an adaptive grip (**B**) seen in patients with anterior interosseous nerve (AIN) syndrome. In the patient with AIN syndrome, the thumb IP joint and the DIP joints of the index finger remain extended while the patient tries to pinch a sheet of paper. (Reproduced with permission from Dang AC, Rodner CM: Unusual compression neuropathies of the forearm: Part II. Median nerve. *J Hand Surg Am* 2009;34[10]:1917-1918.)

found on imaging, initial management of suspected AIN syndrome is typically not altered by MRI findings.

Nonsurgical Management

Because the natural history of AIN syndrome has not yet been fully determined, controversy exists regarding its management. No randomized, controlled trials have compared nonsurgical and surgical management methods, and only limited data exist regarding the duration of nonsurgical management. Because of these limitations, it is difficult to make evidence-based recommendations on the timing of surgical intervention.

Several reports have described spontaneous recovery in patients with AIN syndrome more than 1 year after the onset of symptoms.[25,31-34] Miller-Breslow et al[31] reported on a series of 10 extremities (9 patients) with AIN paralysis, 8 of which were treated conservatively. All eight nonsurgically treated extremities began to show signs of recovery beginning 3 to 12 months after onset, with full resolution of symptoms at 5 to 18 months. Seki et al[34] evaluated AIN function with EMG/NCV testing every 3 to 5 weeks in a series of 21 patients with AIN syndrome (age range, 17 to 65 years). The authors found that the time from onset of AIN palsy to the first FDP1 muscle contraction ranged from 2 to 18 months and, for FPL contraction, from 1 to 24 months. All patients recovered; however, patients aged <40 years had initial return of muscle contractions within 12 months of onset.

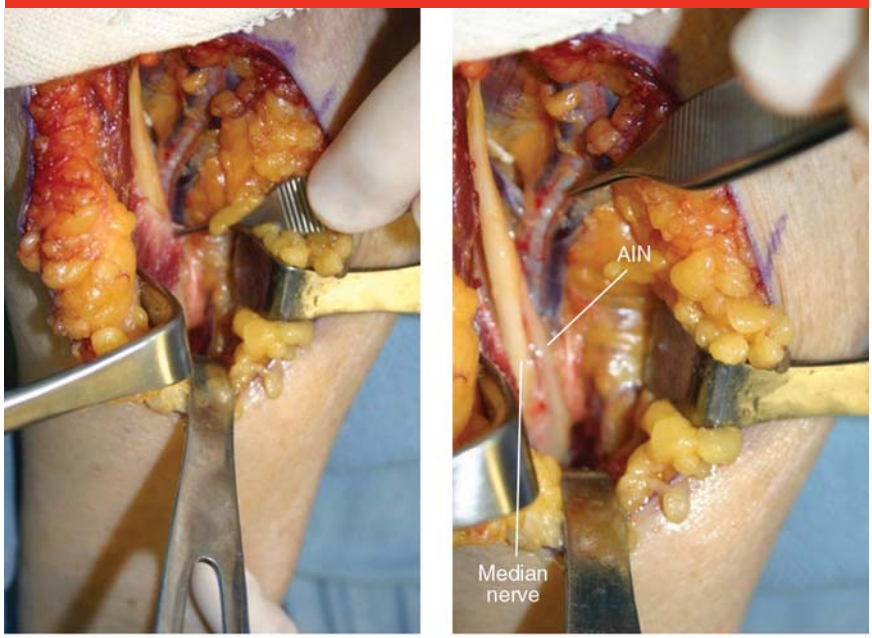
In a retrospective review of 14 patients with AIN syndrome, Ulrich et al[33] found that 8 patients who were treated nonsurgically had spontaneous recovery beginning 3 to 12 months after symptom onset. The remaining six patients underwent surgical decompression after a failed 3-month trial of nonsurgical treatment. One patient in the surgical group had persistent paralysis and eventually underwent tendon transfer; in the remaining 13 patients, return of strength was within 20% of the unaffected side. At final follow-up, there was no significant difference in Disabilities of the Arm, Shoulder, and Hand scores between the surgical and nonsurgical groups.

Because of the high probability of spontaneous resolution after even 1 year of symptoms, a prolonged period of observation should be the cornerstone of management of AIN palsy. Unless there is evidence of a space-occupying lesion, it is difficult to determine whether the etiology of an individual case of AIN palsy is the result of compression or neuritis. In the setting of a confirmed mass, early surgical excision may be warranted. However, in most patients with AIN syndrome, we presume the presence of a neuritic etiology and recommend waiting approximately 12 months before attempting surgical decompression.

Surgical Management

Surgical management should be considered in patients with a known compressing lesion or in those who have failed approximately 12 months of nonsurgical treatment. Typically, the surgical approach for patients with AIN or PS syndrome begins by making a lazy S-shaped incision over the volar forearm, with the proximal extent based on the surgeon's area of preoperative concern. Exploration proximal to the antecubital flexion crease may be

Figure 6



Intraoperative photographs of the volar left elbow/forearm in a patient undergoing decompression for anterior interosseous nerve (AIN) syndrome. **A,** The fibrous arch of the flexor digitorum superficialis is held with forceps overlying the median nerve. **B,** After decompression, the AIN branch is well visualized, coursing radially off of the median nerve.

done first, allowing for identification of the median nerve as well as the ligament of Struthers. After the nerve is released proximally, all points of compression from proximal to distal are then identified and incised, including the overlying lacertus fibrosus, the humeral head of the PT muscle, the proximal fascial margin of the FDS arch, and the Gantzer muscle.[35] The AIN should be decompressed from any overlying tissue and completely visualized as it travels into the distal forearm (Figure 6). Several case reports have described an hourglass-like constriction of the nerve observed intraoperatively. Theories regarding the cause of this constriction have been described, including fascicular torsion, whereby nerve fascicles twist around one another, and postinflammatory adhesions resulting from infection or an autoimmune response.[36,37] The underlying cause and clinical significance of these lesions remain unknown. Surgical release of these constrictions has been reported, but it is unclear whether it hastened recovery.[37] Active range of motion as early as postoperative day 2 is encouraged, with exercises focused on regaining full elbow extension with forearm supination. Full activity and return to work without restriction by 6 to 8 weeks is also encouraged.

The causal relationship between compressive structures discovered intraoperatively and AIN syndrome is unclear. In many studies, patients treated surgically were treated before the mean recovery time of those patients who were successfully treated nonsurgically. Proponents of surgical decompression typically cite the study by Schantz and Riegels-Nielsen,[38] in which 15 of 20 patients with AIN syndrome were treated with surgical decompression. The authors reported satisfactory results in 11 of 15 (73%) patients treated surgically versus 2 of 5 of those treated with observation. However, this interpretation may be misleading. Nine surgical patients began to show improvement 2 to 18 weeks after nerve decompression. The remaining two surgical patients with satisfactory results did not show any improvement at 2 and 7 months after decompression; however, they had full function at a final follow-up of 1 and 5 years. Surgery failed in three surgical patients, and they subsequently had tendon transfers. In this study, the time required for postoperative improvement in many of the patients with satisfactory outcomes actually may be on par with the natural history of nonsurgical treatment. Furthermore, the surgical and nonsurgical patients were not randomized, leading to a selection bias. Three of the five nonsurgical patients had a persistent palsy for >2 years, whereas the surgically treated patients had been symptomatic for an average of 17 weeks.

Summary

The natural history of PS remains unknown. No high-quality evidence exists to determine the appropriate duration of nonsurgical management. Appropriate indications for surgical management remain controversial. In our opinion, surgical decompression for PS is rarely indicated. Surgery may be considered in the patient who remains persistently debilitated for >6 months despite thorough nonsurgical treatment.

AIN syndrome is a rare diagnosis, and consensus on the timing of surgical intervention is lacking. The literature describes spontaneous recovery even 1 year after the onset of symptoms. Increasingly, AIN syndrome is

thought to be the result of neuritis rather than being a mechanical compression. In the absence of a space-occupying lesion, which is rarely the cause of AIN syndrome, we recommend a prolonged nonsurgical approach of approximately 12 months.

References

Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 30, 32, 33, and 38 are level III studies. References 5-7, 11, 13-15, 17, 19, 21, 24, 25, 28, 31, and 34 are level IV studies. The remaining references are level V expert opinion.

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