Surgical treatment and outcomes in 15 patients with anterior interosseous nerve entrapments and injuries

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Object. The authors present data obtained in 15 surgically treated patients with anterior interosseous nerve (AIN) entrapments and injuries.

Methods. Fifteen patients with AIN entrapments and injuries underwent surgery between 1967 and 1997 at Louisiana State University Health Sciences Center (LSUHSC) or Stanford University Medical Center. Patient charts were reviewed retrospectively. The LSUHSC grading system was used to evaluate the function of muscles supplied by the AIN.

Nontraumatic injuries included seven AIN compressions by bone or soft tissue. Traumatic injury mechanisms consisted of stretch or contusion (six patients), injection (one patient), and burn scar (one patient). Presentations included weakness in the flexor digitorum profundus (FDP) muscle to the index finger, FDP muscle to the middle finger, pronator quadratus muscle, and flexion of the distal phalanx of the thumb. Preoperative evaluations included electromyography and nerve conduction studies as well as elbow and forearm plain radiographs.

On surgery, lesions in continuity involved seven compressions, four stretch or contusion injuries, and one injection injury, all of which demonstrated nerve action potentials (NAPs) and were treated with neurolysis. Among the seven compression and four stretch or contusion injury cases, six and three patients, respectively, had LSUHSC Grade 3 or better functional recoveries postoperatively. Two stretch or contusion injuries involved lesions in continuity but demonstrated negative NAPs at surgery. Thus, each was treated using a graft repair after resection of a neuroma. There was one burn scar injury, which was treated via an end-to-end suture anastomosis, leading to a functional recovery better than Grade 3.

Conclusions. Fifteen AIN entrapments or injuries responded favorably to nerve release and/or repair.

Key Words • neurolysis • suture repair • graft repair • anterior interosseous nerve

The AIN is the largest motor branch of the median nerve. It arises in the upper forearm and there supplies the FDP muscles I and II to the index and middle fingers, respectively, and the FPL muscle. In the distal forearm the AIN supplies the PQ muscle. Compression of this nerve results in anterior interosseous, or Kiloh–Nevin, syndrome, originally described by Kiloh and Nevin in 1952. Patients with this syndrome present with a loss of flexion in the distal phalanx of the thumb due to a weak FPL muscle, loss of flexion in the index and middle fingers from paresis of the FDP muscles I and II, respectively, and weakness in the PQ muscle. Although spontaneous pain in the proximal forearm in the region of the pronator teres muscle and the volar wrist is a common feature of this syndrome, there is no sensory loss—a factor differentiating this complex from other syndromes caused by median nerve lesions.

An incomplete AIN syndrome can also occur,14,30 featuring various components of the fully developed syndrome. Duteille, et al.,9 reported on two such cases of incomplete AIN compression, each caused by forearm edema following an intravenous infusion and characterized by an isolated FPL muscle palsy. Palsies involving the AIN due to entrapment or injury compose less than 1% of all upper-extremity nerve palsies.26 Series reported on in the recent literature include 20 patients with 21 cases of AIN syndrome; Schantz and Riegels-Nielsen28 reported on 15 of these patients. Nagano23 reported on 31 cases of nontraumatic AIN palsy, 10 of which the author explored in depth. Arishita and Tsai2 published data on a series of six patients with this syndrome.

In the present paper we report findings in 15 patients with AIN syndrome who were treated surgically during a 30-year period at LSUHSC and SUMC. The types of surgery
performed and outcomes realized in these cases are described.

Clinical Material and Methods

Background: AIN Injury

Anatomical Review. The median nerve provides branches to the pronator teres muscle (Fig. 1) and then passes between that muscle's superficial head, which arises from the medial epicondyle of the humerus, and deep head (not featured in Fig. 1), which arises from the medial ulna. After the median nerve exits from the distal border of the pronator teres muscle, the AIN arises from its radial aspect 5 to 8 cm distal to the medial epicondyle and is the largest motor branch of the median nerve. The AIN then passes beneath the fascial arcade created by the proximal margin of the two heads of the FDS muscle (Fig. 2). At this point, the nerve is accompanied by the anterior interosseous branch of the ulnar artery as it descends on the anterior interosseous membrane surface between the superficial FDS and the deep FDP muscles. The AIN travels between the FDP and FPL muscles and supplies the radial half of the FDP muscle in the proximal forearm; its supply to the FPL muscle is classically described as being close to the junction of the middle and distal thirds of the forearm. The nerve becomes more superficial near the wrist and ends in and supplies the PQ muscle proximal to the wrist in the distal third of the forearm.

Thus, the AIN supplies all the deep muscles on the front of the forearm (except the ulnar half of the FDP muscle), that is, the FDP muscle of the forefinger and long finger, the FPL muscle, and the PQ muscle. This nerve also contains proprioceptive fibers for the intercarpal, radiocarpal, and distal radioulnar joints, which are the only sensory fibers found in the AIN.

Origins of AIN Syndrome. The AIN syndrome can occur spontaneously from entrapment, penetrating injuries, or, less frequently, contusive injuries involving the AIN in the forearm. Patients with entrapment syndrome usually present with spontaneous pain in the proximal forearm lasting from several hours to days and accompanied by a loss of dexterity in the use of the thumb and forefinger. Occasionally, ulnar nerve fibers travel in the AIN branch, and if so, then the functional loss involves not only the thumb and forefinger pinch mechanisms but also the more distal ulnar intrinsic muscles. The latter phenomenon is not part of the classic presentation, however. The AIN syndrome can also occur because of local pressure caused by sleeping on the affected arm, a poorly applied cast, excessive exercise, weight lifting, and viral neuritis. Sometimes compression of the median nerve at the pronator teres muscle level may selectively damage fascicles destined for the AIN and mimic a more distal AIN lesion.
Clinical Features of AIN Syndrome. Up to 85% of patients with AIN syndrome present with pain that tends to be located in the proximal forearm and increases with exercise and decreases with rest.26

The clinical syndrome resulting from dysfunction of the AIN is documented by distinctive results in performing the pinch sign (Fig. 3). In a patient with a complete AIN lesion who attempts to pinch the tips of the terminal phalanges of the index finger and thumb, extension of the distal phalanges occurs. Thus, the pulps rather than the tips of these two digits approximate. As a result, the patient is unable to make an O, that is, unable to flex the distal phalanges of the index finger and thumb.23,30 Furthermore, the patient usually has difficulty gripping objects with the affected hand.

Patients may also suffer slight forearm weakness in pronation due to a lack of strength in the AIN-innervated PQ muscle,26 although this weakness may be masked by the concurrent action of the pronator teres muscle. There is no sensory loss in this syndrome, however.

Note that the FDP muscle of the forefinger is always innervated by the median nerve and thus is a marker muscle for this nerve. Even on complete injury of the median nerve, the tip of the long finger will flex, because the long finger FDP muscle shares a slip of tendon with that of the ring finger so that when the latter tendon (innervated by the ulnar nerve) contracts so too does that to the long finger.

Although uncommon, a partial AIN syndrome is frequently misdiagnosed as a tendon rupture.31

Electrophysiological Findings. All pre- and postoperative electromyograms obtained in the LSUHSC and SUMC patients with AIN palsy showed spontaneous fibrillation potentials by 3 to 6 weeks after injury. The latency of the PQ muscle was also abnormal in most patients in the present study. The electromyography studies demonstrated evidence of reinnervation in the AIN-innervated muscles before there was any clinical evidence of functional recovery, but the extent of functional recovery was not well predicted.

Needle examination of the FPL or FDP muscle is rarely performed but can be valuable given that it may demonstrate abnormalities when the PQ muscle shows no spontaneous fibrillation or sharp waves.30

Fig. 2. Photograph depicting the right AIN passing beneath the fascial arcade of the two heads of the FDS muscles. Reprinted from Atlas of Peripheral Nerve Surgery, Kline DG, Hudson AR, Kim DH, p 261, copyright 2001, with permission from Elsevier.

Fig. 3. Photograph showing the distinctive findings of a right AIN syndrome on performing the pinch test. There is loss of function in the AIN-innervated FPL and FDP muscles, leading to weakness in the distal phalanges of the thumb and forefinger. The left hand (left side of photograph) shows the normal function when attempting this hand posture.
External neurolysis was performed on the AIN in a circumferential manner both proximally and distally to the site of injury on the nerve. Epineurial scarring was resected using fine dissecting scissors, microscissors, or a scalpel blade. Bleeding at the sub- and/or epineurial levels was coagulated using irrigating bipolar forceps.

**Surgical Techniques**

Each of the following surgical techniques was used when appropriate for the AIN repairs at LSUHSC and SUMC.

**Neurolysis.** External neurolysis was performed on the AIN in a circumferential manner both proximally and distally to the site of injury on the nerve. Epineurial scarring was resected using fine dissecting scissors, microscissors, or a scalpel blade. Bleeding at the sub- and/or epineurial levels was coagulated using irrigating bipolar forceps.

**Surgical Exposure.** Exploration of the AIN required identification of the median nerve proximal to the elbow joint. Thus, the incision was begun approximately 5 cm proximal to the antecubital crease in the medial upper arm between the biceps and triceps muscles. It was continued over the elbow’s volar surface, medial to the biceps tendon and the lacertus fibrosus, and was extended to the midforearm via a curved longitudinal incision (Fig. 4).

The incision usually exposed a number of subcutaneous veins and small arteries and branches of the cutaneous nerves, also called the “ABC forearm nerves” (Fig. 5 upper). Some of the vessels needed to be isolated and ligated, whereas others could be swept to one side or another, as could the ABC branches. At and above the elbow, the median nerve was occasionally covered by brachial veins as well as fat.

At the elbow level, the median nerve was initially located inferolateral to the lateral border of the pronator teres muscle. At the proximal end of the incision, the median nerve was first isolated from the brachial artery. The lacertus fibrosus was incised (Fig. 5 center). The nerve was then followed as it passed between the superficial and the deep heads of the pronator teres muscle. Branches to the pronator teres muscle and to the flexors of the wrist and FDS muscle could be seen to emanate from the median nerve’s ulnar aspect.

A pair of Metzenbaum scissors was then placed superficial to both the median nerve and the lateral edge of the superficial head of the pronator teres muscle to protect these two structures distally, while a scalpel was used to divide the overlying pronator teres muscle fascia. The superficial head of the pronator teres muscle was mobilized to visualize the AIN. This head was occasionally divided near its tendinous insertion into the radius and marked with a suture to facilitate later reattachment (Fig. 5 lower). Because the AIN accompanies the median nerve beneath the FDS fascial arch, this arch had to be divided. The FDS muscle was then split in the direction of its fibers to further expose the AIN and median nerve (Fig. 6).

**Internal Neurolysis.** Internal neurolysis was performed for two coexisting indications: 1) when the patient demonstrated an NAP across a lesion in continuity, but the lesion involved one portion of the nerve more than another; and 2) when the patient had a concomitant pharmacologically resistant neuralgia.

**Split (Partial) Graft Repair.** Occasionally, a small area of contusion was found within an in continuity lesion together with preservation of the remainder of the AIN. In this instance, however, the involved individual fascicles or bundles of individual fascicles within the contused region demonstrated no NAP. A split repair was then necessary. Multiple sural nerve cables were grafted to the proximal and then the distal end of the involved region. Fascicular alignment and end-to-end suture anastomosis were performed.
Fig. 5. Drawings depicting the anatomy surrounding the AIN.  

Upper: The skin incision exposes the cutaneous nerve branches, also called the “ABC nerve branches,” and a number of subcutaneous veins and small arteries. Some of the vessels, such as the cephalic vein, should be isolated and ligated. Others can be swept to one side or another, as can the ABC branches. The medial cutaneous nerve of the forearm, also called the “medial ABC nerve of the forearm,” should be preserved. The median nerve is located below the lacertus fibrosis and inferolateral to the lateral border of the pronator teres muscle. The close association between the median and brachial arteries, from which the median nerve must be isolated, is also depicted. The median nerve can be followed as it passes between the superficial head (shown) and the deep head of the pronator teres muscle (not shown).  

Center: After incising the lacertus fibrosis, the medial edge of the pronator teres muscle is incised. A pair of Metzenbaum scissors is placed on top of the nerve and gradually edged distally as the scalpel divides the overlying pronator teres muscle fascia.  

Lower: After dividing the lacertus fibrosus and, if necessary, the superior head of the pronator teres muscle, the FDS muscle is split in the direction of its fibers to expose the AIN and the main median nerve.
End-to-End Suture Anastomosis. If the nerve segment as a whole did not transmit an NAP, then resection was performed. An epineurial end-to-end suture anastomosis could sometimes be performed. Sharp dissection permitted mobilization of the proximal and distal stumps. Adequate resection to healthy epineurium and fascicular structure was necessary before end-to-end suture anastomosis or graft repair. After hemostasis and avoidance of excessive tension at the suture site, the repair was performed with 6-0 or 8-0 monofilament interrupted nylon suture. It is important to align the fascicles during repair.

Graft Repair. If an end-to-end suture anastomosis could be performed without excessive tension after mobilization, then a graft repair was necessary. Usually multiple strands of sural nerve were sewn in place by applying an interfascicular technique.

Patient Population

The charts of 19 patients who had been treated at LSUHSC and SUMC for AIN syndrome between 1967 and 1997 were reviewed retrospectively. The LSUHSC grading system was used to evaluate AIN functional loss and recovery (Table 1). All patients had loss of function in the FDP I and FPL muscles and less severe weakness involving the FDP II and PQ muscles. Four (21%) of 19 patients experienced a spontaneous recovery and did not require surgical intervention; 15 (79%) underwent surgery after showing no or few signs of clinical and electrophysiological functional recovery. Of these 15 patients, seven had injuries involving the AIN due to compression by bone or soft tissue, six had a stretch or contusion injury, and one each had an injection or burn scar injury (Table 2).

Surgical intervention was performed in all seven patients with nerve compression injuries. These patients demonstrated NAPs intraoperatively. External neurolysis was performed, and following this procedure, six of seven patients had a Grade 3 or better functional recovery. Stretch or contusion injuries resulting in AIN palsies necessitated surgery in six patients. Four of these six patients were found at surgery to have an in continuity AIN injury, and intraoperative NAPs were present. These four injuries required neurolysis; three of four of these procedures resulted in Grade 3 or better functional outcomes. Two stretch injury lesions were also in continuity but demonstrated no NAPs intraoperatively; graft repair after resection of a neuroma was undertaken in each case (Table 3). One injection injury necessitated neurolysis and one burn scar injury, end-to-end suture anastomosis. The patients in both of these cases realized Grade 3 or better functional outcomes.

Results

Nineteen patients were evaluated at LSUHSC and SUMC for AIN lesions. There was severe or complete loss of function in the FDP I and FPL muscles and less severe weakness involving the FDP II and PQ muscles. Four (21%) of 19 patients experienced a spontaneous recovery and did not require surgical intervention; 15 (79%) underwent surgery after showing no or few signs of clinical and electrophysiological functional recovery. Of these 15 patients, seven had injuries involving the AIN due to compression by bone or soft tissue, six had a stretch or contusion injury, and one each had an injection or burn scar injury (Table 2).

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Discussion

Origins of AIN Entrapment or Injury Syndrome

In a review of the recent literature, we found sporadic cases of AIN injury resulting from venous access mishaps. Kagel and Rayan17 reported on one case of AIN palsy, which occurred among 67 patients who suffered intravenous catheter-related complications in the hand and forearm. One case of AIN syndrome, described by Puhaindran and Wong,26 developed after a peripherally inserted central catheter line was inserted into the brachial vein. In an abstract Dolderer and colleagues’ reported on four patients who each experienced isolated FPL muscle palsies following attempted venipuncture of the median cubital vein.

Fractures are another source of AIN palsy. Stahl, et al.,35 described such a case, which occurred after a closed Galeazzi fracture, that is, a fracture of the radius at the junction of its middle and distal thirds together with an associated subluxation of the distal ulna. Arenas, et al.,1 reported on one case of AIN palsy subsequent to a Monteggia ulna frac-
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Fascicular torsion of the AIN in three patients with non-traumatic AIN palsy was described by Yasunaga, et al. Each of the three patients had an AIN that intraoperatively showed hourglass-like constrictions and 30° of fascicular torsion. These abnormalities were believed to have been formed on branching of the AIN from the median nerve. An environmental change after birth was postulated to have induced edema and resulted in the AIN palsy.

Kato, et al., reported a patient with an acute AIN palsy caused by septic arthritis of the elbow. Another case of AIN palsy occurred when the patient was in a resuscitator after cardiopulmonary resuscitation had been performed. An anomalous palmaris longus muscle was found in this individual at the time of surgery.

Mononeuritis or neuralgic amyotrophy is actually the most frequent cause of AIN palsy, although this precipitating scenario never requires surgery. This type of AIN involvement leading to a bilateral anterior interosseous neuropathy after surgery has been detailed in a case report by Szwedata, et al. Suzuki, et al. described a similar bilateral presentation in the context of Parsonage–Turner syndrome.

Traumatic lesions occur less frequently and usually resolve spontaneously. If surgery is required, it is rarely performed earlier than 12 to 18 months posttrauma if there is no recovery.

**Anatomical Variations**

There can be variability in the origin of the FPL muscle and its tendon. These variations and their relation to the median nerve and AIN must be considered if they are found while decompressing these nerves in the proximal forearm or while performing nerve and tendon repairs in this region.

Dolderer, et al., undertook cadaveric dissections of the AIN to determine any anatomical factors predisposing to AIN injuries that were associated with attempted venipuncture of the median cubital vein. These authors found that the AIN arises more proximally than previously described in the literature. On tracing the FPL muscle nerve branch retrograde from its insertion into the muscle, they noted that this branch joined the AIN at the level of the proximal third of the forearm and continued proximally as an easily separable fascicular group without intraneural cross-connections well up to the median nerve trunk at the cubital fossa. There was a direct relationship between the superficial cubital vein and the solitary nerve branch to the FPL muscle. Injury to this nerve branch is potentially significant because of the absence of any interfascicular cross-over from other territories within the AIN. The relative separation of the FPL fascicular bundle within the median nerve at the level of the cubital fossa as well as its radial position might render it more prone to injury than if it were clustered more securely within the dominant bulk of the median nerve trunk.

Dellon and Mackinnon dissected 31 cadaveric arms to study the variations in the anatomy of the muscles and fibrous arches, which might cause median nerve compression in the forearm. They found the Gantzer muscle, an accessory head of the FPL muscle, to be present in 45% of cadavers.

There are reports of an incomplete AIN syndrome, in which there is isolated paralysis of the FPL muscle, as occurred in the cases discussed in the introduction of the present paper. These atypical presentations may be caused by yet other anatomical variations; that is, the ulnar nerve may innervate the long finger FDP muscles, as occurred in the cases discussed in the present paper. Nicholls, et al., described an incomplete AIN syndrome due to mechanical compression by the Gantzer muscle.

Martin–Gruber anastomosis, which is present in 15% of limbs, is the term used to describe the presence of various interconnections between the median nerve or AIN and the ulnar nerve. One variant involves a connection from the AIN to the ulnar nerve, and this situation could result in an AIN syndrome together with additional intrinsic muscle weakness. The AIN may also innervate the FDP muscle in its entirety; thus, weakness in all fingers will result if the AIN is injured or entrapped. In some people, the AIN supplies only part of the FDS muscle, which may portend even additional anomalies in an AIN clinical presentation.

Neal and Rayan have described a patient with an AIN syndrome due to an anomalous fibrous band of the FDS muscle extending to the brachialis muscle fascia and separate from the normal arcade passing over the median nerve. There was a venous ring encircling the nerve at the level of the anomalous arcade.

**Imaging Studies**

Ultrasoundography is becoming an important diagnostic tool for establishing the diagnosis of AIN syndrome. Hide, et al., have reported previously undescribed ultrasonography findings in AIN syndrome. These data include loss of AIN continuity.

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**Table 2: Mechanisms of injury in patients with AIN lesions**

<table>
<thead>
<tr>
<th>Mechanism of Injury</th>
<th>No. of Cases</th>
<th>Surgically Treated</th>
<th>Grade ≥ 3</th>
<th>Outcome</th>
</tr>
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<tbody>
<tr>
<td>Compression by bone or soft tissue</td>
<td>7</td>
<td>7</td>
<td>4</td>
<td></td>
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<tr>
<td>Injection</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Stretch</td>
<td>6</td>
<td>6</td>
<td>3</td>
<td></td>
</tr>
<tr>
<td>Burn scar</td>
<td>3</td>
<td>1</td>
<td>1</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>19</td>
<td>15</td>
<td>9</td>
<td></td>
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</table>

**Table 3: Anterior interosseous nerve lesion category and types of surgery performed**

<table>
<thead>
<tr>
<th>Lesion Category</th>
<th>Type of Surgery</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>AIN palsy</td>
<td>Neurolysis</td>
<td>12</td>
</tr>
<tr>
<td>AIN absent</td>
<td>Suture repair</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>Graft repair</td>
<td>2</td>
</tr>
</tbody>
</table>

* No lesion in the present series was not in continuity, which would have required 1° or 2° suture repair or 2° graft repair.
of muscle bulk, increased reflectivity, reduced perfusion on Doppler ultrasonography studies, and lack of active contraction of the affected muscle, all of which were observed in their group of patients with AIN syndrome. The authors stated that these findings can aid in the localization of the pathological process and the exclusion of tendon rupture. Dynamic observation of muscle function and Doppler ultrasonography changes after exercise were also found to be helpful in identifying the muscles involved in AIN syndrome. In a paper on entrapment neuropathies including the AIN syndrome, Martinoli, et al., stated that high-resolution ultrasonography can reveal changes in the nerve’s shape and echo texture, and thus can depict many extrinsic causes of nerve entrapment, such as those that occur in AIN syndrome.

**Treatment Options**

Conservative treatment includes arm rest, for example, the avoidance of any strenuous forearm activity, especially if the activity was the precipitating cause. Other nonsurgical means of treatment consist of actual immobilization, steroid injections, and antiinflammatory medications. Schmitt, et al., described the use of neuromuscular electrical stimulation in a case of AIN injury due to a forearm fracture, which resulted in a good outcome; without a controlled study, however, it is hard to prove that this treatment is beneficial.

Some cases in the literature have shown spontaneous recovery 1 year after onset without surgical intervention. Other authors advocating tendon transfers to restore function in AIN syndrome believe that these transfers should be delayed for 1 year as well.

**Conclusions**

Anterior interosseous nerve lesions are uncommon nerve entrapments or injuries. This LSUHSC and SUMC series features the treatment and outcomes in 15 surgical lesions presenting as AIN palsies. Four additional patients with AIN palsies experienced spontaneous recoveries. We describe the various types of surgical repairs, whose selection depended on whether the lesion in continuity, which was the universal type of lesion in the patients who had undergone surgery, had an NAP. Most patients fared very well postoperatively. Thus, the AIN palsies respond to surgical treatment.

**References**


D. H. Kim, et al.
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Manuscript received May 25, 2005.
Accepted in final form January 11, 2006.

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