

Median nerve compression: Pronator and anterior interosseous syndromes

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Abstract

Proximal compression or neuropathy of the median nerve is rare compared with carpal tunnel syndrome but recognition and management of these conditions is important. Pronator syndrome refers to compression of the median nerve around the elbow, which may present with pain in the forearm and paraesthesia in the hand. Anterior interosseous syndrome (palsy) can result from mechanical compression or inflammatory neuropathy causing significant impairment of dexterity in the hand. Clinical features and the place of neurophysiological testing are discussed. Surgical decompression is indicated when mechanical compression is suspected.

Pronator syndrome

Introduction

Pronator syndrome is the most proximal compression neuropathy of the median nerve. Originally described by Seyffarth in 1951 [1], pronator syndrome (PS) is a somewhat misleading name as the syndrome encompasses compression of the median nerve around the elbow at more anatomical sites than just the pronator teres. From proximal to distal these include the ligament of Struthers, the bicipital bursa, anomalous arteries, the bicipital aponeurosis within the deep fascia (also called the lacertus fibrosus), between the two heads of pronator teres (PT), and the fibrous arch of the flexor digitorum superficialis (FDS) [2] (see figure 1).



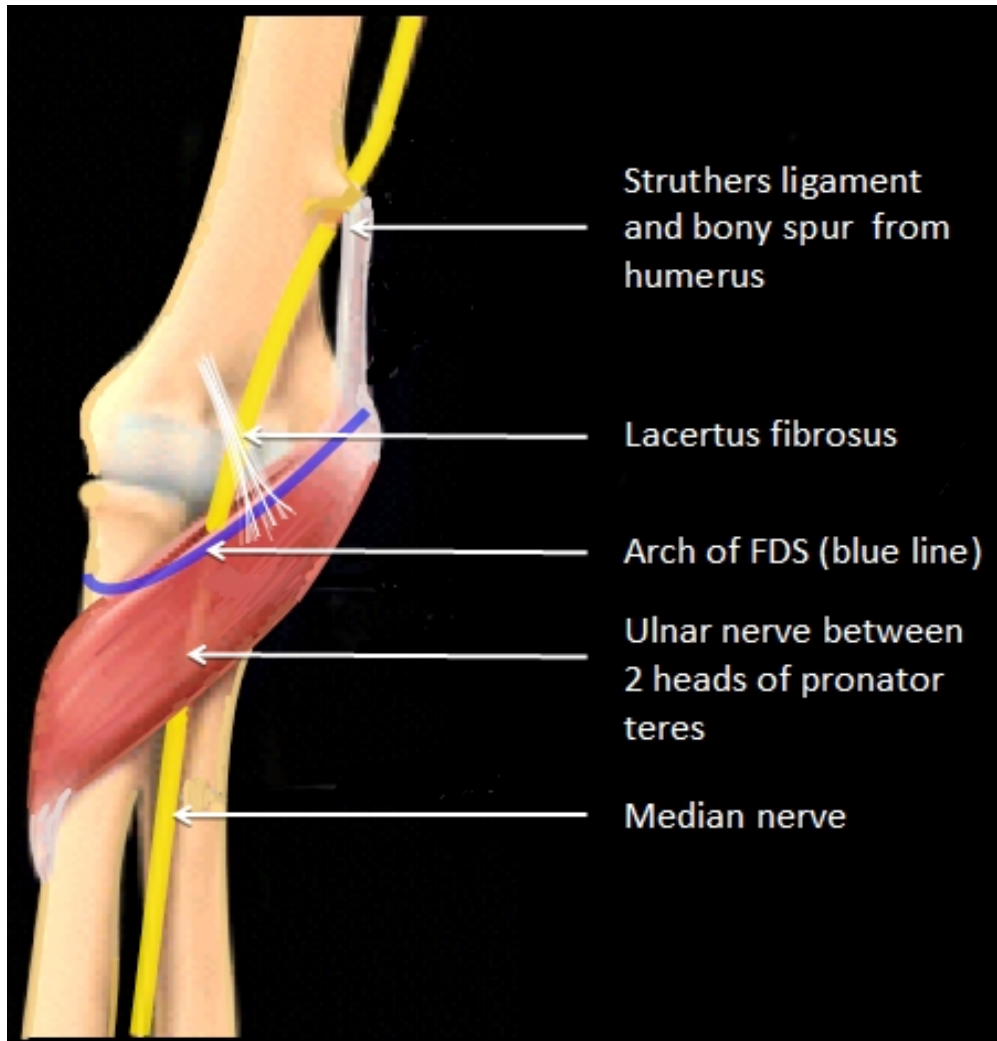


Figure 1: Site of compression of proximal median nerve

Anatomy

Dellon and Mackinnon further elucidate on the musculoaponeurotic arches and tunnels formed by PT and FDS in a dissection study on 31 cadaver arms [3]. One, two or no arches may be present depending on the anatomical relationship of PT to FDS and the deep fascia. For instance a high/proximal origin of the superficial head of PT results in its fusing with the deep fascia (lacertus fibrosus) creating a proximal arch. A single arch is formed when the FDS originates from the undersurface of the two heads of PT, whereas if the FDS has a second origin from the interosseous membrane (in addition to its humeral origin) then two arches may be present (one formed by PT and one by FDS). Dellon and Mackinnon found that all but one cadaver had a fibrous arch (97%). Other authors describe fibrous bridges of either the PT or FDS, which makes comparison difficult, however the descriptions and/or illustrations in these papers depict both the PT and FDS. In these papers frequency of fibrous bands ranged between 40 to 75% [4], [5], [6].

In 1822 Tiedemann identified a vestigial fibrous band arising from a supracondylar process of the medial humerus and inserting onto the medial epicondyle [7] that was suggested to represent an anomalous high origin of the PT. In 1848 Struthers was the first to describe median nerve entrapment by this structure, known today as the ligament of Struthers [8]. In 1889, Testut found only 8 supracondylar processes in 929 dissections, whereas Struthers reported an incidence of “about one in fifty cadaver dissections”. Terry examined 1000 living adults and could palpate a supracondylar process in seven (0.7%), which he then confirmed radiographically, and in only one individual was it bilateral [9]. Dellon found only one incomplete supracondylar process in 104 extremities [3]. Overall approximately 0.6 to 2.7% of individuals have a residual supracondylar process and ligament of Struthers [4], [7].

Clinical aspects

Pronator syndrome (PS) most commonly presents with pain radiating from the volar proximal forearm to the wrist. However, pronator syndrome may be misdiagnosed as carpal tunnel syndrome (CTS) as both can result in altered sensibility in the radial three and a half digits and are associated with clumsiness of the hand [4]. However, differences in symptoms and provocation test findings can help differentiate PS from CTS. The palmar branch of the median nerve arises 4 to 5 cm proximal to the transverse carpal ligament [10] and is not involved in CTS, whereas patients with PS may report reduced sensation over the thenar eminence supplied by this nerve. In addition, PS patients do not characteristically complain of nocturnal symptom and they should not elicit a positive Tinel or Phalen's sign at the wrist. Grip strength and thenar muscle strength is not normally reduced in PS patients [4].

Provocation tests

Patients with PS typically present with proximal volar forearm pain, of insidious onset of months to years, made worse by repetitive pronation and supination of the forearm [4], [11]. A number of clinical examination maneuvers which reproduce this forearm pain have been described for diagnosing PS, and may even discriminate at what level the median nerve is being compressed.

The patient is asked to hold his elbows by his side bent to 90 degrees, with the forearm in neutral. The examiner holds the patient's hand and tries to passively supinate the patient's forearm against resistance, such that the patient actively contracts pronator teres in order to maintain the forearm position at neutral. The elbow is then extended, and if pain or paraesthesia are reproduced the median nerve is suspected of being compressed between the two heads of pronator teres [12]. The senior author (MAP) performs this provocation test by asking the patient to pronate the forearm from a position of maximum supination with the elbow extended; this places maximum compressive force upon the nerve by fully tensioning the PT passively and adding the effort of voluntary contraction.

If symptoms are reproduced from resisted flexion of the fully supinated forearm, then compression by the lacertus fibrosus is implied [13], whereas symptoms from resisted flexion of the FDS to middle finger implicates compression at the level of the fibrous arch of FDS [12]. An anatomical study by Tubbs et al. of the sublime bridge in 33 cadavers found that compression of the median nerve by this structure only occurred with the elbow in extension [6], which suggests that elbow extension should be incorporated as part of the middle finger FDS resisted flexion test. A fourth test is the pronator compression test, performed by exerting pressure over the pronator teres in both forearms simultaneously. Reproduction of paraesthesia in the radial three and a half digits, or forearm pain, within 30 seconds indicates a positive test [14]. In a study of 71 patients Johnson et al. [4] found that causes of compression of the proximal forearm median nerve was in the following order of frequency: the pronator teres, the flexor superficialis arch and lastly by the lacertus fibrosus.

Diagnostics

MRI and electrophysiology testing

MRI may be of use in identifying fascial structures or even a point of nerve compression, as well as the presence of a space occupying lesion (SOL) [15]. Nerve conduction studies (NCS), however, have not proven reliable in confirming a clinical diagnosis of PS. Three separate studies found abnormal preoperative NCSs in less than a third of patients treated for PS who improved after surgical decompression [11], [16], [17]. Furthermore, Hartz et al. [11] found intraoperative testing demonstrated slowed conduction across the pronator tunnel in only 1 of 10 patients, and intraoperative NCT improvement in only 3 of 10 patients following median nerve decompression. In the series reported by Buchthal and Eversman, patients with an abnormal preoperative NCT had a 66% incidence of complete or partial relief of symptoms postoperative compared with 80% recovery in patients with a normal preoperative NCT who were also decompressed [17], [18]. This suggests that abnormal NCTs may only be present in patients with more extensive or severe compression of the proximal median nerve. It is possible that in patients with negative NCTs, nerve compression is only intermittent and dependent on the position of the elbow and forearm and that NCT should be performed in a position provoking symptoms.

The lack of specific diagnostic investigations highlights the importance of clinical assessment. Several papers report on patients presenting with PS after their symptoms failing to resolve following carpal tunnel decompression [11], [14], [16]. The overlap in the clinical picture of these two syndromes is highlighted by Olehnik et al. [16] who found a positive Tinel and Phalens tests at the wrist in around half

the 20 patients who had not undergone previous carpal tunnel decompression. Unfortunately they do not report if these provocative tests were subsequently negative after PS decompression. They also note that patients with carpal tunnel syndrome can have a positive pronator compression test, and attribute these findings to the double crush phenomena. In such patients they perform a CTD as the first procedure if abnormal NCTs are localised to the wrist. However if NCTs are negative then surgical release in the proximal forearm is the initial procedure [16].

Therapy

Conservative management is normally the first-line treatment of PS and has been shown to be effective in 50–70% of patients [4], [19]. Conservative management comprises avoidance of aggravating activities, resting rather than splinting the limb, and pain relief with non-steroidal anti-inflammatories, although corticosteroids have also shown to be beneficial [20]. No consensus exists on how long to pursue nonsurgical treatments. Most surgeons consider that after several months of failed conservative treatment, or if a SOL is identified, surgery may be indicated. Surgeons who are skeptical of the over diagnosis, or even the existence, of pronator syndrome, will not offer surgery unless there is positive MRI or electrophysiological testing to demonstrate proximal median nerve compression. Most surgeons recommend surgery on patients whom demonstrate positive provocative signs on clinical examination. Presciutti and Rodner instead prefer to describe their patients as having “arm ache” rather than give them the presumptive diagnosis of pronator syndrome in the absence of any positive findings [21].

Surgery for Pronator Syndrome

Published outcomes following decompressive surgery for PS report success rates between 77% to 92% of partial or complete resolution of symptoms [4], [11], [16], [19]. Olehnik et al.'s study of 39 proximal, median nerve decompressions found no correlation of a successful outcome following surgery when comparing patients with abnormal or normal preoperative nerve conduction studies. As with other less frequently seen upper limb compressive syndromes there are no randomized clinical trials comparing surgery with conservative treatments in managing pronator syndrome.

Anterior Interosseous Syndrome (AIS)

Anatomy

The anterior interosseous nerve (AIN) arises from the median nerve 5 to 8 cm distal to the medial epicondyle [22] at the level of the deep head of the pronator teres muscle. It runs distally with the anterior interosseous artery on the volar surface of the interosseous membrane, between the FPL and FDP muscles, beneath the pronator quadratus (PQ) muscle, and has terminal fibres supplying the wrist joint. It innervates FPL and PQ and the radial portion of the FDP muscle belly to the index finger (FDP1) and usually to the middle finger (FDP2). It is frequently described as a motor only nerve as the AIN has no cutaneous sensory distribution, however, it does contain sensory afferents fibres from the wrist [23].

In a cadaver dissection study of 31 arms, Dellon and Mackinnon found the AIN to originate from the deep (posterior) aspect of the median nerve in 12 (39%) cases. In the remaining 19 (61%) the nerve arose in the classically described fashion from the radial border of the median nerve, of which 16 (52%) were found to be in relationship to a fibrous arch, and therefore susceptible to compression [3].

In 1960 Mangini described Ganzter's muscle (an accessory slip of the flexor pollicis longus muscle frequently cited as a cause of AIN compression) having a 74% incidence in 76 dissected arms, with 62% being bilateral. The muscle arose from the medial epicondyle (61%), coronoid of the ulna (22%), the intermuscular septum (9%) or had a combination of origins. All were reported as lying between the median nerve and AIN, such that the AIN could be compressed posterior to this muscle [24]. However, Dellon and Mackinnon only found a 45% incidence of Ganzter's muscle, all of which arose from the medial epicondyle and did not cross superficial to the anterior interosseous nerve. They noted that the body and tendinous portions of the muscle varied considerably, and that a hypertrophied muscle belly could compress the AIN against an overlying aponeurotic structure, or that a tendinous portion could distally compress the AIN such that not all AIN innervated muscles would be affected [3].

Clinical aspects

The first description of an AIN neuropathy, resulting in an isolated FPL palsy, can be attributed to Duchenne de Boulogne in 1872 [25]. AIN syndrome is classically associated with motor weakness to the FPL, FDP to the index and middle fingers, and PQ. However, as has been shown by Sunderland (1945) [22], there is considerable individual variation regarding the proportions of the FDP muscle supplied by the median and ulnar nerves. In patients where the FDP2 is entirely supplied by the ulnar nerve, rather than AIN, there will be no motor deficit to the middle finger. Also in anatomical studies Spinner, in 1972, found that the branch to FPL arises around 4cm distal to the origin of the AIN, and is vulnerable to isolated compression by the tendinous origin of FDS, resulting in isolated FPL palsy [5]. Isolated palsy of either the FPL or FDS is termed variably as incomplete or isolated AIS depending on the author. Paralysis of pronator teres has also been reported [26]. Clinical presentation may also vary in patients with the Martin-Gruber anastomosis.

Patients most commonly recognise weakness of FPL function, and a characteristic examination finding is the patient's inability to make an "OK" sign (see figure 2).

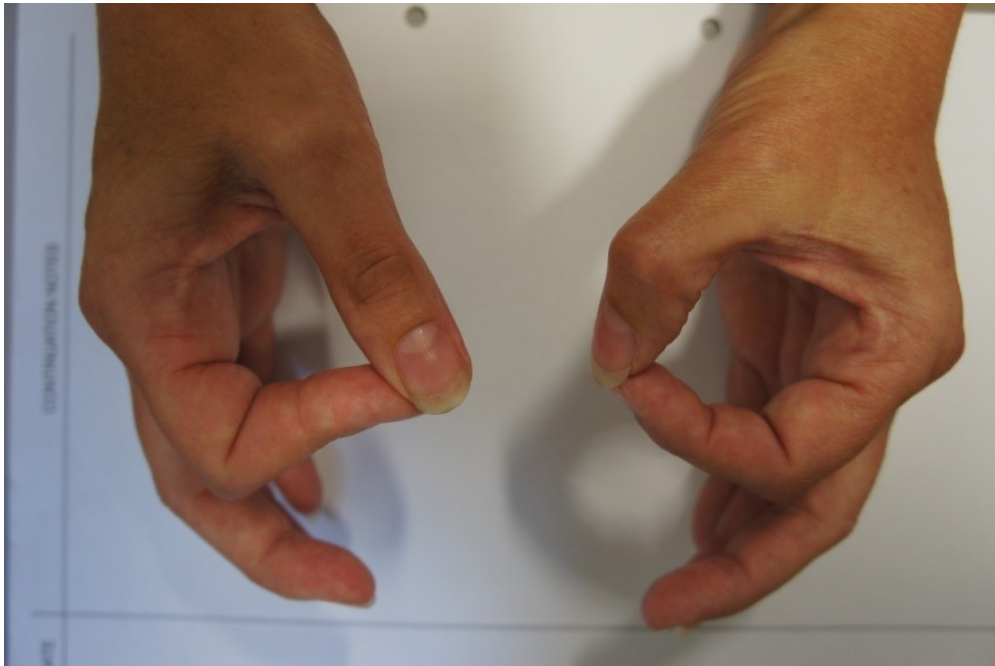


Figure 2: Patient with anterior interosseous nerve syndrome of right arm, with the patient unable to make the "OK" sign with her right index finger and thumb

An alternative test is to ask the patient to pinch a piece of paper between the tips of their thumb and index finger. AIS patients will assume a flatter key-type grip, due to the inability to flex at the thumb IPJ or index DIPJ (see Figure 2). The tenodesis effect of the intact FPL and FDP tendons establish their continuity, and is important in order to differentiate AIS from an isolated FPL and/or FDP rupture, a frequent misdiagnosis, particularly in cases of isolated AIS of the FPL [27].

Despite the AIN lacking a cutaneous distribution, most case series report that 75–100% of their patients experienced forearm pain, or less frequently elbow, upper arm or shoulder pain. The pain frequently preceded any AIN dysfunction by several weeks, and would often improve or resolve with onset of paralysis [26], [28], [29], [30]. This is likely to represent referred pain from compression of wrist sensory afferent fibres. Seror reviewed 117 published cases of AIS, and found that 85% reported symptoms of pain, but that pain was not predictive of an inflammatory or mechanical origin [31].

Pathophysiology

Parsonage-Turner Syndrome is neuritis of the brachial plexus, typically preceded by flu-like symptoms. It presents with shoulder pain and weakness but may also include features of AIS in the forearm [32]. AIS has been described following ante-cubital/forearm cannulation, vigorous weight lifting [33] and other activities involving forceful hyperpronation [34]. Forearm bone fracture [31] and a fall on an outstretched hand [35] have also been linked with AIS. However the majority of AIN palsies are idiopathic and a site of anatomical compression is usually implicated. In reported series of surgical decompressions, no

discernable point of compression was observed on inspecting the nerve. Dissection studies frequently report sites of “potential constriction of the AIN”, for example, Spinner found a potential site of compression in all of his 25 cadavers. However, this does not correlate with the rarity of the condition in the population. Those in favour of an underlying inflammatory aetiology point to Sunderland's [36] detailed anatomic studies of the median nerve isolating fibres destined to become the AIN as far proximal as the brachial plexus. Despite this the actual site of neuritis has never been conclusively identified [30], [31]. The cause of AIS remains uncertain with multiple possible aetiologies, both inflammatory and mechanical.

Diagnosics

Electromyography (EMG) is important in the work-up of AIN to confirm the diagnosis objectively, identify the muscles involved, assess the severity of the neuropathy and to follow recovery. Electrophysiology also helps rule out more proximal lesions, and identify patients with tendon ruptures in whom tenodesis tests cannot be performed (eg. patients with painful wrists). Seror reports that the most sensitive electrodiagnostic test is based on examination of the pronator quadratus, dysfunction of which may not be obvious clinically due to a normally functioning pronator teres [31]. Similarly Dunn et al. reviewed the pre-op magnetic resonance imaging (MRI) of cases of surgically proven AIN compression. Oedema was evident within all AIN innervated muscles, but specifically oedema within PQ was the most reliable sign of AIS [37]. Signal intensity changes within muscles are seen as early as 4 days post-injury, such that MRI may in fact have the advantage over electromyography of earlier detection of abnormalities. EMG studies require 2 to 3 weeks for changes to become evident [38].

Therapy

The paucity of experience and lack of randomised studies with AIS means that treatment is predominantly surgical or conservative depending on the speciality to which the patient is initially referred. Spontaneous recovery can occur even after 12 months of conservative treatment, especially in lesions due to Parsonage-Turner or paralytic neuritis syndrome [27], [31], [32]. In publications that differentiate complete from incomplete AIS (palsy isolated to FPL or FDP1), there was good to complete recovery in all incomplete AIS irrespective of type of treatment. These authors do not recommend surgery until at least a 6 to 12 month period of observation has elapsed, and advise against treatment in cases where neuritis is identified as the aetiology [28], [31]. Seki et al. [29] treated 21 patients with AIS (complete and incomplete) conservatively with vitamin B12 and electrical stimulation therapy. Recovery occurred within 12 months in patients under the age of 40 years, and achieved a final British Medical Research Council grade of 4 or better. They recommend non-operative management of patients under 40 years of age.

The role of surgery in AIS

Spinner recommended surgical exploration of AIS after 12 weeks without clinical improvement or with continued EMG abnormalities. A trial of conservative management of at least 12 weeks is recommended by most surgeons. Schantz and Reigels-Nielsen [39] operated on 15 out of 20 patients with AIS at an average of 17 weeks after onset of symptoms. 11 patients (73%) had satisfactory return of function, whereas as only 2 of the 5 (40%) treated non-surgically recovered, with the remaining 3 having a persistent palsy at 4 years follow-up [39]. They concluded that exploration of the AIN is the treatment of choice in AIS. Their results may be explained by the high proportion of their patient cohort (65%) identified as having a mechanical compression of the AIN intraoperatively. They do not state the underlying aetiology of the 5 conservatively treated patients. However, an untreated mechanical aetiology is less likely to recover than an AIN neuritis in which spontaneous resolution appears to be the expected outcome.

Surgical approach for Pronator and Anterior Interosseous Syndromes

A lazy-S and zig-zag incision across the ante-cubital is the recommended surgical approach in PS and AIS (Figure 3), and our preferred approach.



Figure 3: Incisions used to access proximal median nerve with lazy-S across elbow, and zig-zag incision distally

The median nerve is identified and explored from proximal to distal, dissecting it from the lacertus fibrosus (Figure 4), the humeral head of pronator teres, and the fascial edge of the FDS arch (Figure 5), dividing these structures if necessary.



Figure 4: Scissors placed under lacertus fibrosus

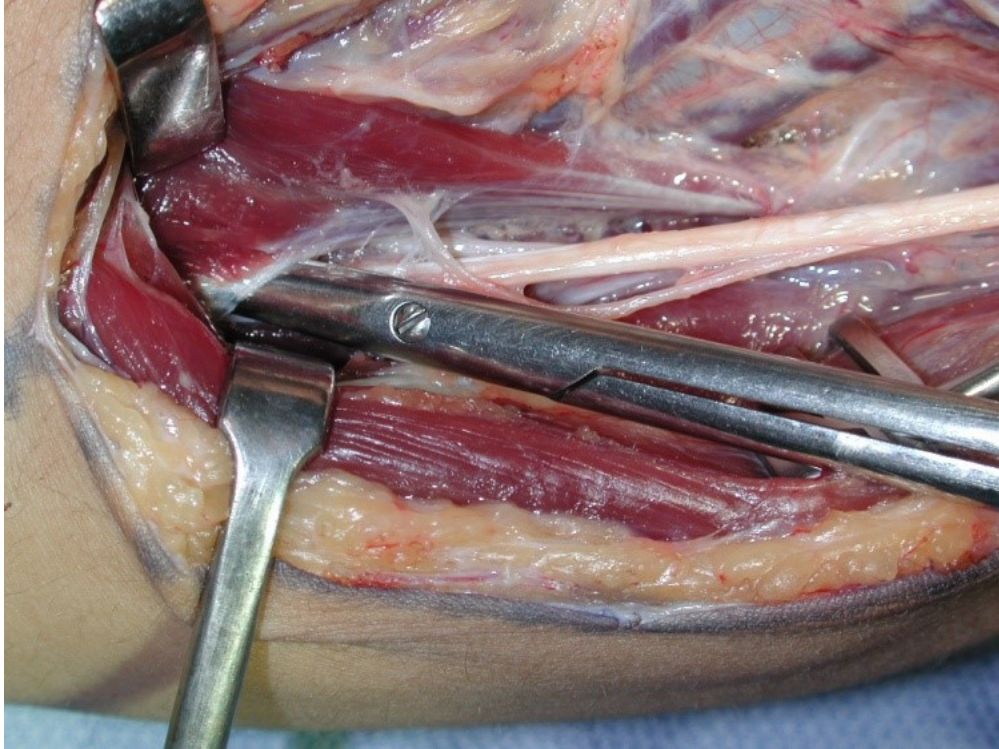


Figure 5: Scissors placed under conjoined arch of superficial head of pronator teres and flexor digitorum superficialis

The surgeon's finger should be able to pass smoothly under these structures next to the nerve in supination and pronation and with the elbow in both flexion and extension. It should be noted that at this level nearly all the median nerve's branches are from its ulnar side, with the exception of the AIN. The extent of the incision is adapted to the areas of specific interest. If examination of the ligament of Struthers is required the incision can be extended proximally along the medial aspect of the arm, or distally along the forearm if Gantzer's muscle is implicated, and these structures divided (Figure 6).

The elbow may be splinted for comfort post-operatively, but otherwise early active motion is encouraged.

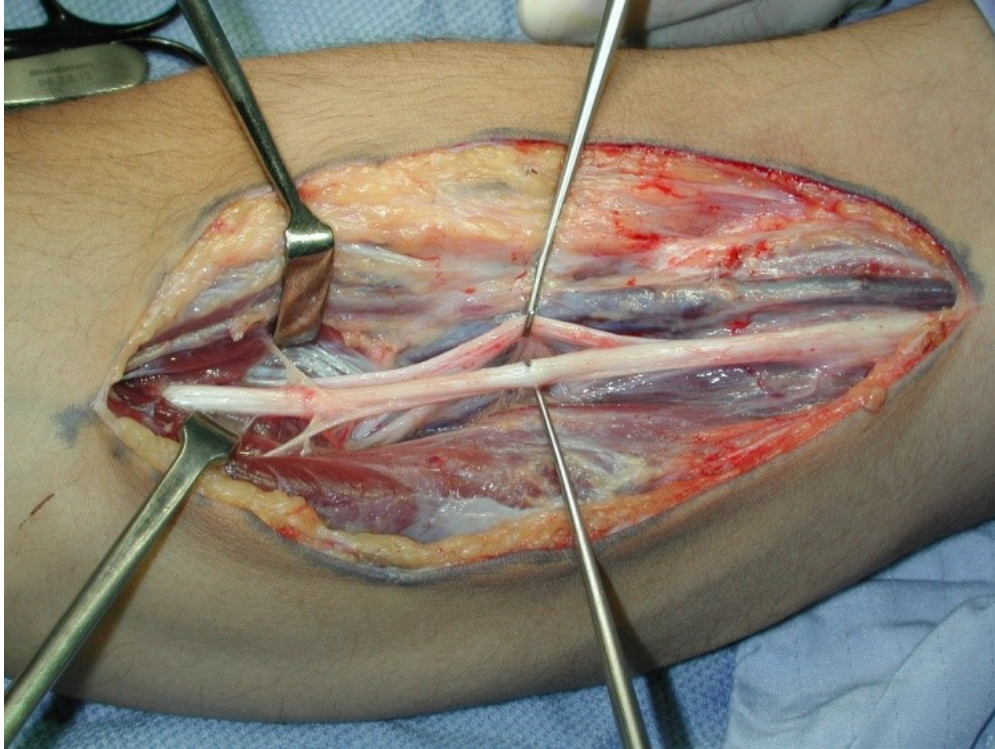


Figure 6: Anterior interosseous and median nerves following release of fibrous arches distally and proximal exploration above elbow

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