

Median and anterior interosseous nerve entrapment syndromes versus carpal tunnel syndrome: a study of two cases

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Two patients presented with forearm and hand pain, and were initially examined by their medical doctors. The first case was diagnosed as a possible neuralgia due to congenital cervical spinal fusion. The second case had a radiographic study taken of the elbow and hand, which was negative and therefore no conclusive diagnosis or treatment was given. This article will discuss the anatomical path of the median and anterior interosseous nerves in the forearm, the possible areas of neural entrapment or irritation and the resulting symptoms and signs as compared to carpal tunnel syndrome. The patient's presenting symptoms were found to be the most significant detail when differentiating the type of neurological entrapment. In both cases, the patients presented with a gradual progression of anterior forearm pain, numbness or discomfort that radiated to the hand and fingers. To find the cause of this repetitive type of irritation, the functional movement patterns of the upper extremity kinetic chain was assessed including the cervicothoracic and scapulothoracic regions. Provocative tests were used to confirm the site of irritation. The first case showed pronator quadratus weakness. The second patient's symptoms were reproduced with resisted elbow flexion and pronation while digital pressure was applied to the median nerve. Acute care was directed at the specific area of irritation/inflammation with electrotherapeusis. The treatment also consisted of spinal and joint manipulation, proprioceptive neuromuscular facilitation techniques, strengthening and endurance rehabilitation exercises

Voici le cas de deux patients qui ont consulté un chiropraticien pour douleurs à l'avant-bras et à la main, et les deux avaient déjà été examinés par un médecin. Chez le premier, on a diagnostiqué une névralgie possible attribuable à une fusion congénitale de la colonne cervicale; quant au deuxième, on a pris une radiographie du coude et de la main mais, comme l'examen s'est révélé négatif, aucun diagnostic clair n'a été posé et aucun traitement n'a été entrepris. Il sera question, dans le présent article, du trajet anatomique des nerfs interosseux médian et antérieur de l'avant-bras, des régions possibles de compression ou d'irritation de ces nerfs et des signes et symptômes qui en résultent comparativement à ceux du syndrome du canal carpien. Les symptômes présentés se sont avérés les éléments les plus significatifs quand est venu le temps de différencier le type de compression neurologique. Dans les deux cas, il y a eu apparition graduelle de la douleur, de l'engourdissement ou du malaise ressentis à l'avant-bras antérieur, accompagnés d'irradiation dans la main et les doigts. Pour trouver la cause de cette irritation récurrente, on a procédé à une évaluation des mouvements fonctionnels de la chaîne cinétique du membre supérieur, y compris des régions cervico-thoracique et scapulo-thoracique. Des tests de provocation ont également été effectués pour confirmer les points d'irritation. Dans le premier cas, on a conclu à une faiblesse du carré pronateur; dans le deuxième, les symptômes ont été reproduits par une flexion et une pronation contrariées du coude avec application d'une

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aimed at restoring the proper kinematics of the upper extremity
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Introduction

Median and Anterior Interosseous Nerve (AIN) entrapment syndromes are thought to be less common than Carpal Tunnel Syndromes.^{1,2} Crawford and Noble in their paper on AIN paralysis suggested that perhaps AIN entrapment may not be as rare as perceived, and that there may only be a high incidence of nonrecognition.³ When looking at the anatomy of the forearm and correlating that with the physical signs and symptoms of the presenting complaint, the clinician may establish the site of entrapment without invasive procedures. Once the area of aggravation has been found, a rational and comprehensive treatment program can be initiated. The entire kinetic chain has to be taken into account, not only for diagnostic purposes, as related to the cause of the problem, but for therapeutic and rehabilitative management.^{4,5} As Lieben-son⁴ states, finding the chain reaction is crucial to removing the key perpetuating factors for motor system disorders.

Case Report A

A 27-year-old male involved in computer sales, presented with what he described as numbness in the right hand and lateral aspect of the forearm. He reported that this 10-year condition was aggravated by repetitive activities, riding motocross, keying at the computer or any activity requiring use of the hand. Shaking the arm in extension would relieve his symptoms. He did see his family medical physi-

pression digitale sur le nerf médian. Les zones d'irritation ou d'inflammation ont alors été soumises à des soins actifs d'électrothérapie. Des manipulations de la colonne et des articulations, des techniques de facilitation neuromusculaire proprioceptive et des exercices de réadaptation de renforcement et d'endurance visant à rétablir la cinétique normale du membre supérieur composaient également le traitement.
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MOTS CLÉS : nerf médian, nerf interosseux antérieur, rond pronateur, compression, réadaptation.

cian, 2 years prior. X-rays were taken and the patient was told that there was fusion of the cervical spine and that his condition would worsen as he gets older.

The examination of his upper extremity began with the cervical spine and then proceeded to the elbow and hand. Cervical spine rotation to the right and right lateral flexion was limited. There was anterior head carriage with significant bilateral upper trapezius, bilateral scaleni and sternocleidomastoid hypertonicity. The thoracic spine was hyperkyphotic and the shoulders were elevated and internally rotated. This qualitative presentation and unbalanced posture, contributed to the anterior head carriage, shoulder elevation and exaggerated thoracic kyphosis as described by Janda⁴ in upper crossed syndrome, leading to abnormal upper extremity mechanics.

Janda's⁴ shoulder abduction test was positive. Janda⁴ states that this test provides information about the coordination of the muscles of the shoulder girdle. It is tested while the patient is seated, with the elbow flexed. The patient then abducts their arm. Abduction is a result of three components: abduction in the glenohumeral joint, rotation of the scapula and elevation of the shoulder girdle. The identifying movement is the elevation that normally starts at 60 degrees of abduction, as the first 60 degrees should be entirely glenohumeral movement and not trapezius elevation.⁴

Cranial nerve testing and upper limb flexion/extension, abduction/adduction were normal, as was elbow and wrist

flexion/extension. Sensation to fine touch and pinwheel was normal. Upper limb reflexes were 2+. Testing for vertebrobasilar insufficiency, using George's tests was also unremarkable. Orthopaedically, cervical Kemp's (Jackson's compression with rotation and extension) test was positive on the right. Adson's test, modified Adson's test, cervical compression, cervical distraction and doorbell were negative. Trigger points were found bilaterally in the scalenii.

Facet joint dysfunction was noted at C1–C2, C2–C3, C4–C5 and T3–T4. Elbow and wrist range of motion was normal. Tinel's tap test at the elbow and wrist was negative. However, Tinel's tapping was provocative along the anterior interosseous nerve. Phalen's test, reverse Phalen's and compression of the wrist were also negative. There was tenderness within the pronator teres muscle. Direct pressure along the proximal, medial aspect of flexor pollicis longus aggravated the patient's forearm and hand symptomology. Resisted pronation with the elbow flexed showed significant weakness of pronator quadratus.

A cervical spine radiographic examination revealed congenital fusion of the vertebral bodies and posterior elements at C4–C5. The remaining disc spaces, facet joints, intervertebral foramina were within normal limits as was cervical alignment. No other soft tissue or cortical abnormalities were observed. It was apparent that this fusion had no effect on the pronator quadratus weakness, which is innervated by C8 and T1 nerve roots.²

A diagnosis of anterior interosseous nerve entrapment syndrome was made.^{6,7,8,9} This diagnosis was based on the presenting symptoms, the inability of the patient to pronate the forearm while the elbow was flexed,^{6,7,8,10} and the fact that the syndrome may present with both episodic pain and paresthesia.³ With this case, the patient presented with what he described as numbness in the affected forearm; however, fine touch and pinwheel testing was negative. It is possible that the median nerve was also compromised, due to the static compression while he was on his motorcycle or working on the computer thus affecting the sensory component.⁶ Spinner⁸ indicated that the clinical manifestation may vary depending on whether the neural lesion is complete or partial and whether there are anatomical variations present. Thus, the clinical presentation may differ even with complete lesions.⁸

Treatments initially consisted of interferential current therapy along the course of the anterior interosseous nerve,

spinal and extremity mobilization techniques such as proprioceptive neuromuscular facilitation and post isometric relaxation. Spinal and extremity adjusting techniques and postural exercises such as the isometric chin tuck were also used. The patient was shown dynamic range of motion stretches for the upper extremity, the scapulothoracic and shoulder region as outlined by Murphy.¹¹ Thera-tube and Neck Ball exercises were then introduced to help regain proper closed-chain dynamics of the entire shoulder girdle and extremity. These exercises helped relax the tight trapezius, latissimus dorsi, scalenius, sternocleidomastoids as well as the suboccipitals while strengthening the neurologically inhibited lower and middle trapezius, rhomboids, longus colli and shoulder external rotators as described by Janda⁴ in upper crossed syndrome. This allowed for proper shoulder hike and allowed for proper scapular and humeral rotation and rhythm.

Strengthening exercises began with shoulder flexion and extension and progressed to external rotation and scapular retraction-protraction. From there, thera-tube exercises recruited the forearm musculature, and simple elbow flexion/extension exercises advanced to pronation/supination and diagonal movements. The goal of the treatment was to reduce the entrapment, restore function and build endurance.

Following the third treatment the patient was able to go cycling with minimal symptoms and he was discharged after the ninth treatment, being symptom free and most importantly having a program of exercises that he could use to help avoid the problem in the future. Unfortunately, the patient moved to northern Ontario and no further follow-up was made.

Case Report B

A 42-year-old female, warehouse worker presented with a diffuse, non-specific "steady ache, burning and tingling" in the right forearm and fingers. The patient indicated that her symptoms were over the anterior aspect of her forearm and lateral fingers. She also complained of a sore right elbow. The patient mentioned that she was taking a welding course in the evenings. As a result of her deteriorating condition she saw her family medical doctor a month prior to presentation in this office. Her family physician requested a radiographic study of her elbow and hand. This study was normal and no further treatment plan or diagnosis was given.

The patient indicated that any kind of activity would aggravate her symptoms, including her warehouse work, which involved lifting boxes and her welding classes at night school. The patient found that maintaining her right arm in a flexed and semipronated position, with the wrist deviated to ulnar side aggravated her condition. This was the position, as described by the patient, when holding a welding torch. Her condition would also worsen while sleeping. She would sleep with her elbow flexed and forearm pronated. To relieve her symptoms, she would stretch her right arm. The stretch, as demonstrated by the patient, consisted of abduction of the arm to 90 degrees, external rotation of the arm, complete supination of the forearm and extension of the elbow and wrist.

On examination, cervical spine rotation was limited to the right. Upper limb reflexes were 2+, bilaterally. Fine touch sensation in the upper limb was normal, as was cranial nerve testing. George's tests were also non-provocational. Facet joint dysfunction was noted at C2–C3, C4–C5, T2–3 and T4–T5. The right scaphoid was also restricted in a posterior to anterior glide. Cervical spine orthopaedic tests were negative. The right forearm was significantly more developed than the left. Firmness and definite tenderness was palpated over the right pronator teres muscle. The medial epicondyle was also tender to digital palpation. Resisted elbow flexion and pronation reproduced her paresthesia, especially when also applying digital pressure over the median nerve as it travels deep between the two heads of pronator teres. No weakness was evident in the pronator teres muscle or with pronation. Phalen's test, reverse Phalen's, and Finkelstein's tests were all negative, as was wrist compression. Resisted extension and supination testing of the elbow and resisted testing of the wrist were also negative.

A diagnosis of pronator teres syndrome was made.^{6,7,8,9} This diagnosis was based on the fact that there was no weakness with pronation and her symptoms were reproduced with resisted elbow flexion, pronation and digital pressure applied to the median nerve as it lies under pronator teres. Another key component in diagnosing pronator teres syndrome was the stretch the patient was using to alleviate her symptoms. Hertling and Kessler¹² described a common flexor-pronator tendon stretch where the wrist was held in extension with forearm supination to relax the pronator teres muscle.

A series of treatments were given over the next 4 con-

secutive weeks. The treatments involved ultrasound, spinal as well extremity adjustments and rehabilitation. The rehabilitation involved extensive dynamic stretching exercises, the strengthening of weak muscles and the re-education of proper movement patterns. The patient progressed from simple flexion-extension movement to more complex, multiple joint and rotatory arm and shoulder patterns. The neck ball was also utilized in order to strengthen the cervicothoracic component of this dynamic chain. As with case "A", proper function of the scapulothoracic area was crucial, as it is the foundation for proper kinematics of the upper extremity. The patient indicated that she was able to comfortably sleep without any arm pain after her 7th treatment. She continued to work and go to school throughout the treatment program and was discharged after her 14th visit. Upon follow-up, one and one-half months after her last visit, the patient indicated that she was doing well and was symptom free. She indicated that she would contact the office if her symptoms returned.

Anatomy

The median nerve crosses the elbow joint and first passes beneath the lacertus fibrosus, which is the thick fascial tissue extending from the biceps tendon to the forearm fascia. It then passes between the two heads of the pronator teres. The humeral head of pronator teres attaches on the medial epicondyle and the ulnar head attaches on the coronoid process and inserts in the middle of the lateral aspect of the radius.² The median nerve then runs beneath the flexor digitorum superficialis arch.⁶ This muscle originates on the medial epicondyle, coronoid and ulnar ligaments of the elbow, and its second head attaches to the anterior proximal half of the radius. The insertion of superficialis is on the palmar surface of the middle phalanges of the four fingers.² The median nerve then runs between flexor digitorum superficialis and flexor digitorum profundus. Flexor digitorum profundus begins on the proximal aspect of the ulna and interosseous membrane and runs to the distal phalanges of the 4 fingers. Once the median nerve reaches the wrist, it becomes superficial and runs deep to palmaris longus between flexor digitorum superficialis and profundus.²

The median nerve has four branches. The articular branch innervates the elbow joint and the muscular branch directly supplies pronator teres, flexor carpi radialis, palmaris longus, flexor digitorum superficialis, the three

thenar muscles (abductor pollicis brevis, flexor pollicis brevis, opponens pollicis) and lumbricals to the 1st and 2nd fingers. The medial half of flexor digitorum profundus is supplied by the ulnar nerve.²

The anterior interosseous branch runs along the interosseous membrane after branching off of the median nerve at the inferior part of the cubital fossa. The anterior interosseous nerve lies between flexor digitorum profundus and flexor pollicis longus. Flexor pollicis longus originates on the distal aspect of the radius and the lateral half of the interosseous membrane. It inserts into the distal phalanx of the thumb. The anterior interosseous nerve supplies the lateral half of flexor digitorum profundus, flexor pollicis longus and pronator quadratus. The pronator quadratus arises on the anterior, distal ¼ of the ulna and inserts on the anterior distal ¼ of the radius. The anterior interosseous nerve ends by supplying the carpals.²

The 4th and last branch of the median nerve, the cutaneous branch, begins proximal to the flexor retinaculum and runs over top of the retinaculum to supply the lateral aspect of the hand.²

Clinical features

The clinical presentation of median nerve entrapment syndromes distal to the elbow will depend upon the cause and severity of the problem. In cases of trauma, there will be evidence of injury to the soft tissue and in some cases bony structures of the extremity. Spinner⁸ indicates that both anterior interosseous nerve paralysis and pronator teres syndrome are completely distinct and that the clinical picture will depend on the extent of the neural lesion being complete or partial and on the anatomical variations. The clinical presentation may even differ with complete lesions.⁸

When the median nerve is severely affected due to paralysis, pronation is very difficult due to paralysis of pronator teres and pronator quadratus.⁶ The brachioradialis, which is innervated by the radial nerve, may provide weak pronation with assistance of gravity.² Wrist flexion is weak and there is ulnar deviation due to continued supply to flexor carpi ulnaris by the ulnar nerve.⁶

Median nerve paralysis affects flexor pollicis longus, flexor digitorum superficialis and flexor digitorum profundus. This will result in lack of flexion of the interphalangeal joint of the thumb and the distal and proximal interphalangeal joint of the index, or first, finger.^{3,6,7,8,9,10}

However, due to flexor digitorum profundus receiving innervation from the ulnar nerve, the 4th and 5th digits will be spared and sometimes the 3rd, because it often receives dual innervation from both the median and ulnar nerves.⁶ With the flexor digitorum superficialis also being paralyzed, there will be weakness in grasping with the 4th and 5th digits. This can be demonstrated by holding all the fingers straight, thereby ignoring flexor digitorum profundus, and having the patient try to flex the proximal interphalangeal joint of the ring finger.⁶ Even with the loss of all the extrinsic flexors to the index finger with median nerve paralysis, flexion of the metacarpophalangeal joint is maintained because of the ulnar innervation of the interosseous muscles.

Pronator teres syndrome, clinical presentation

The most frequent symptom of pronator teres syndrome is mild to moderate aching pain in the proximal forearm that is sometimes described as a tiredness or heaviness.^{1,6,7,8,9} Repetition aggravates the condition.^{6,9} Stewart⁹ indicates that paresthesias are usually weak or absent because the compression is usually minimal and often intermittent, these symptoms may be ill defined and difficult to describe accurately. The physical findings are usually tenderness of the pronator teres muscle, paresthesias in the distribution of the median nerve when the forearm is pronated forcibly against resistance, a positive Tinel's sign over the pronator teres muscle and a firm pronator muscle.⁹ Use of the arm may intensify symptoms, which may begin insidiously or after repetitive elbow and forearm movement.^{1,6,8,9} Symptoms are often ill defined and misdiagnosed with carpal tunnel syndrome.⁷ Carpal tunnel syndromes are intensified when sleeping.¹³ However it is possible to have symptoms during sleep with pronator teres syndrome. The patient may maintain the forearm flexed and pronated, as in the fetal position, thus contracting the pronator teres and compromising the area of entrapment.

As pronator teres syndrome intensifies, it may radiate to the shoulder.⁶ Paresthesias in the radial 3½ digits may be presented.^{8,1} Paresthesias may also be present along the median nerve distribution, including the wrist and beyond,¹⁴ but not as severe or well localized as in carpal tunnel syndrome.⁷ In carpal tunnel syndrome, pain is reported in the lateral four digits of the hand but it may be perceived more proximally up to the forearm or shoulder.¹³ Ross and Kimura¹³ indicate that the pain and numb-

ness associated with carpal tunnel syndrome is often aggravated by hand use and the patient is characteristically awakened at night. Sensory loss usually affects the volar and dorsal aspects of the lateral three and one-half digits with carpal tunnel syndrome.¹³

Golding et al.¹⁴ found that when evaluating the tests for carpal tunnel syndrome: thenar wasting, Phalen's sign, Tinel's sign, sphygmomanometer test and comparison with median nerve conduction test, he found little diagnostic value in each. His study demonstrated the lack of sensitivity and specificity in these tests.¹⁴ Golding et al.¹⁴ believed that careful evaluation of the symptoms, rather than signs was important, and that confirmation should be made by median nerve conduction tests whenever possible.

Stewart⁹ mentions that signs of median nerve dysfunction, using electrophysiological studies, have usually been mild or absent. In one study that Stewart⁹ cites, six of seven patients showed slowing of median motor conduction in the forearm with a normal distal motor latency, indicating a nerve lesion in the forearm. In another study that Stewart⁹ cites, only 2 of 39 patients had definite evidence of median nerve damage. Ten of these patients had intraoperative electrophysiological studies, none of which showed evidence of median nerve damage within the pronator muscle.

Physical findings are often as diffuse as are the symptoms. The most important physical sign is direct pressure and tenderness with possible radiation.⁶ According to Dawson et al.⁶ superficial anatomical contour, markings and measurements may be different from the uninvolved side. Field¹⁵ clearly shows the osseous and muscular landmarks that the clinician should be aware of when evaluating the forearm.

According to Dawson et al.⁶ and Spinner,⁸ many tests help establish the level of compression. The test used most often, include:^{1,7}

- A** patient's forearm is held in pronation and the wrist flexed, the doctor tries to supinate and extend the wrist, implicating the pronator teres
- B** patient's forearm is flexed and supinated, and the doctor tries to pronate the forearm, this contracts the biceps and tightens the lacertus fibrosus
- C** patient tries to flex the 1st finger at the proximal interphalangeal joint against resistance, therefore using flexor digitorum superficialis and implicating the

superficialis arch

Anterior interosseous nerve syndrome, clinical presentation

Hozman and Skosey¹ believe that only 1% of upper extremity lesions is represented by anterior interosseous syndrome. Anterior interosseous nerve syndrome typically presents with acute pain in the proximal forearm or arm, lasting several hours or days.^{6,8} According to Crawford and Noble,³ pain associated with AIN syndrome may occur spontaneously or as the result of traumatic events. The pain may manifest as radiation from the shoulder, aching in the elbow and along the medial border of the forearm and even across the anterior aspect of the thumb.³ Pain subsides, to be followed by paresis or total paralysis of the flexor pollicis longus and the flexor profundus to the index finger and the middle finger.⁸ The presence of paresthesia should indicate median nerve compromise, however as Dawson et al.⁶ state, paresthesias are usually weak or absent due to minimal or intermittent compression. Crawford and Noble³ note that although the symptomology may include both episodic pain and paresthesia, they are not consistent features of anterior interosseous syndrome.

With isolated paralysis of the anterior interosseous nerve there is a characteristic disability or weakness when flexing the interphalangeal joint of the thumb, flexion of the distal interphalangeal joint of the index finger and pronation of the forearm when the elbow is flexed.¹⁰ The patient presents with the inability to perform a pinch grip, i.e. there is no flexion of the distal interphalangeal joints and therefore only the pads of the distal phalanges touch due to lack of flexor pollicis longus and flexor pollicis profundus activity.^{3,6,7,8,10} With anterior interosseous nerve paralysis, the index finger shows an increased flexion of the proximal interphalangeal joint and a hyperextension of the distal interphalangeal joint.¹⁰ Flexor digitorum superficialis, which is innervated by the median nerve, is not involved and can therefore continue to flex the proximal interphalangeal joint of all the fingers and even the metacarpophalangeal joints. This results in the contact point between the thumb and index finger shifting more proximally.¹⁰

The syndrome may be complete with both thumb and finger involvement, or incomplete with either the thumb or finger affected.⁷ Spinner⁸ indicates that it is possible for

the pronator quadratus alone to be paralyzed or even weakened, but the pathological process may be clinically unrecognized because of an uninvolved pronator teres. The pronator quadratus must be examined with the elbow flexed to eliminate the effect of the pronator teres.⁷ By flexing the elbow, the humeral head of pronator is eliminated and about 25 % of the muscle's pronatory strength from the ulnar head remains. Pecina et al.⁷ also notes that patients feel dull pain in the proximal third of the forearm that is aggravated by radial pressure at the tendinous arch of flexor digitorum superficialis.

Van Der Wurff et al.¹⁰ mentioned how Kiloh and Nevin first described two patients with isolated anterior interosseous nerve paralysis, thus only defining a motor deficiency because as mentioned earlier, this nerve's function is strictly motor.^{3,7,10}

EMG abnormalities and motor conduction studies help confirm and differentiate the level of involvement.⁹ Electrodiagnostic studies give an estimation of the severity of the median neuropathy and the contribution of axonal or demyelinating nerve damage.¹³ In the two case studies, electrophysiologic testing was not done and as Dawson et al.,⁶ Spinner⁸ and Buchberger et al.⁵ show, reproduction of symptoms with direct pressure and provocative tests suffice. Stewart⁹ and Radecki¹⁶ imply that perhaps electrodiagnostics can be misleading and unreliable when performed without any other supportive tests.

Nerve conduction testing has assumed an increasingly prominent role in the epidemiologic investigation of median nerve slowing and the associated carpal tunnel syndrome as it relates to the workplace because it is an objective test.¹⁶ Radecki¹⁶ examined 1472 patients with upper-extremity symptoms. His study was designed to discern whether determinants other than hand usage account for the variation in the median and ulnar latencies at the wrist.¹⁶ Radecki¹⁶ found that an increased wrist ratio, an increased body mass index, and aging were associated with prolongation of median latencies. Aging and increased height were associated with prolongation of ulnar latencies, whereas an increased body mass index was negatively correlated to ulnar latencies.¹⁶ The study showed how the patients with work related complaints were anthropometrically similar and the group without complaints was also physically identical. The patients with the diagnosis of "median slowing" were physically almost the same yet different from those without median slowing.

Radecki¹⁶ concluded that in order to interpret electrodiagnostic studies of median and ulnar nerves, age and anthropometric measures must be considered; otherwise, epidemiologic studies will be confounded and entrapment neuropathies such as carpal tunnel syndrome will be over diagnosed.

Discussion

The median nerve can be compressed in three areas, the least frequent of which is by the ligament of Struthers.¹² The ligament of Struthers, if present, may be found on the anteromedial surface of the humerus, running from an aberrant spur 5 cm above the medial epicondyle to the medial epicondyle. The median nerve and brachial artery and vein pass through beneath this ligament. It is estimated that .7 to 2.7 percent of the population have this spur and only a smaller fraction develops entrapment.⁶ The next region of compression is below the elbow where the pronator teres muscle may compress the median nerve. The last area of compression and most distal, is compression of the anterior interosseous nerve.⁶

Anatomical abnormalities

There are several areas of compression, once the median nerve passes the elbow. Spinner (8) observed six anatomical abnormalities associated with median nerve entrapment in this area:

- hypertrophied pronator teres
- fibrous band within the pronator teres
- median nerve passing posterior to both heads of the pronator teres
- thickened lacertus fibrosus
- thickened flexor superficialis arch
- an accessory tendinous origin of the flexor carpi radialis from the ulna.

These etiologies have the common characteristic, of compression secondary to static or dynamic stenosis.⁷ Hozman and Skosey¹ believe that in most cases the cause is vascular. They argue that the gradual increase in pressure within a limited space leads to venous congestion in the epineural and perineural vascular plexuses, which then slows the flow of blood to the nerve trunk itself. The resultant hypoxia of the nerve is followed by dilation of the small vessels and capillaries and this progresses to further compression, scarring and increased weakness.¹ In the

presence of trauma, which may lead to Volkmann’s contracture or with the loss of power as a result of severe injury, surgery and decompression is the only option.^{6,1,7} Stewart⁹ cautions us as to these “causes” of pronator teres syndrome and cites cadaver dissection studies that have shown many of these so-called abnormalities occur in normal persons.

Many investigators propose a dynamic compression of the median nerve during supination or elbow extension. Pronator teres syndrome appears to be caused by repetitive use of the arm, minor trauma or muscular hypertrophy associated with anatomical abnormalities.^{6,7,8,9}

The anterior interosseous nerve is vulnerable to injury or compression by several means including (8):

- A tendinous origin of the deep head of the pronator teres
- A tendinous origin of the flexor digitorum superficialis to the index finger
- A thrombosis of crossing ulnar collateral vessels
- An accessory muscle and tendon from the flexor digitorum superficialis to the flexor pollicis longus
- An aberrant radial artery
- A tendinous origin of variant muscles, the palmaris profundus or flexor carpi radialis brevis
- An enlarged bicipital bursa encroaching on the median nerve near the origin of the anterior interosseous nerve

Trauma to the arm and nerve occurs as a rare complication of fractures and deep penetrating wounds. There are reports of anterior interosseous syndromes following excessive exercise, with rapid improvement when the exercise is stopped.⁹ Prolonged pressure applied to the area during sleep, leaning over a beam,⁶ or plaster cast application³ have been implicated. Strenuous exercise and weight lifting have also lead to paralysis.⁶ Stewart⁹ believes that spontaneous anterior interosseous neuropathy is occasionally the only finding in patients with severe shoulder and arm pain that is characteristic of brachial plexus neuropathy. Stewart⁹ also indicated that partial median nerve lesions may mimic anterior interosseous nerve neuropathy as would nerve damage due to improper venipuncture. He reports cases of rupture of flexor digitorum profundus and flexor pollicis longus due to rheumatoid arthritis that mimicked anterior interosseous neuropathy.⁹

Treatment

Conservative management is the recommended initial

treatment of choice. However the definition of conservative varies from, reduction of physical activity, forearm immobilization for a limited time and local corticosteroid injection,⁷ to NSAIDS and avoiding activities that exacerbate the condition,⁶ to conservative management from two to three months,¹ to spontaneous recovery within 18–24 months.³ Dawson et al.⁶ mentioned that spontaneous recovery indicates that a neuritis was present. All agreed that in the presence of trauma or impending Volkmann’s contracture surgical decompression is immediately required. After the failure of conservative care, surgical intervention is necessary. However, Dawson⁶ found, from personal experience, that the patients operated on did not demonstrate faster recovery than those treated conservatively. Stewart⁹ found that surgical decompression was beneficial with patients when objective evidence of nerve dysfunction was not found. Those with the best evidence of nerve dysfunction improved by stopping the repetitive pronation and local anesthetic or corticosteroid injections.

With the above two cases, treatment started as did the examination, by assessing all the dysfunctional aspects of the upper extremity kinetic chain.⁵ Liebenson⁴ shows a guideline to follow for the improvement of the entire kinetic chain linking key muscle or joint pathologies when addressing spinal rehabilitation. This rationale can also be applied to any extremity and joint rehabilitation protocol (Table 1).

When assessing the upper extremity, Janda’s⁴ examination for muscular imbalances can be used: push up test, head flexion and shoulder abduction tests. Lewit¹⁷ points out that failure in treatments may be due to the inability to improve the patient’s habits rather than ineffective methods of treatment. He found that with pain in the elbow area, the underlying cause of the condition is a cramped way of

Table 1
Addressing Functional Pathology
in the Kinetic Chain

Relax/stretch overactive/tight muscles
Mobilize/adjust stiff joints
Facilitate/strengthen weak muscles
Re-educate movement patterns on reflex, subcortical basis

using the hand and therefore the restoration of proper kinematics is crucial.¹⁷

Successful treatment relies on finding the key functional pathologies that are biomechanically or kinesiology-related to the symptomatic area.⁴ Liebenson⁴ also states that such findings are only qualifiable. Quantifiable tests, such as functional testing or electrodiagnostics give us a baseline of a measurable, objective deficit but they do not tell us what motor system dysfunction is related to the patient's symptoms or "pain generator."⁴ Functional testing performs two basic tasks. First, it provides a baseline of functional capacity and secondly, it identifies the targets for functional restoration.⁴ Looking at the scapulothoracic rhythm, upper body position including cervicothoracic positioning, elbow and wrist movement patterns gives us information as to the arthrokinematics of the upper limb. The information gathered from the analysis of the upper limb kinetic chain allows us to determine why the area around the median and anterior interosseous nerves is being over-stressed, over-irritated or compressed.

With the above patients, muscular relaxation was achieved with electrical muscle stimulation applied to the upper thoracics. Postisometric relaxation techniques were used on the cervicothoracic, the scapulothoracic and distal extremity flexors/extensors. Proprioceptive neuromuscular facilitation was also utilized on the distal musculature. These patients were instructed on dynamic flexibility exercises as outlined by Murphy.¹¹ The stretches included, scapular retraction/protraction, scapular abduction/adduction, shoulder rotation and shoulder flexion/extension.¹¹

Diversified chiropractic manipulative therapy was applied to the restricted extremity articulations as well as to the restricted spinal vertebrae to restore joint mobility with resultant muscular relaxation.¹⁷ Mobilization techniques were utilized to regain proper joint play in the proximal and distal radioulnar articulation.³ The use of joint mobilization as originally described by Mennell¹⁸ and later used by Kessler and Hertling¹⁹ or joint manipulation as described by Lewit¹⁷ help restore the axis of rotation in the involved articulations and are the first step towards proper arthrokinematics.

The patients were also instructed on cervicothoracic postural exercises and on scapular stabilization exercises to strengthen the lower and middle trapezius, rhomboids and external shoulder rotators. Some of the exercises utilized were as follows: isometric cervical exercises espe-

cially for longus colli, rhomboid and middle trapezius facilitation, Thera-tube scapular retraction and external rotation as well as Thera-tube forearm flexion/extension and pronation/supination. The Neck ball was also used to restore proper cervical movement patterns. When exercising, correct posture was stressed and Brugger's position¹⁷ was maintained.

Conclusion

Careful consideration should be made to the signs and more importantly, the presenting symptoms with patients presenting with forearm and hand pain. Differentiating median nerve entrapment syndrome and anterior interosseous nerve entrapment syndrome from carpal tunnel syndrome is clinically important as to the direction of the treatment protocol and therefore the cessation of the presenting complaint. Provocative testing and understanding the presenting symptoms as they relate to the anatomy is important in arriving at a diagnosis. As described, an accurate method of assessment is by direct pressure with resultant tenderness and reproduction of symptomology with possible radiations. Electrodiagnostics may be inconclusive in some cases. Once the area of entrapment has been determined, proper functional restoration should be utilized involving the entire closed kinetic chain. In the two cases outlined, spinal and joint manipulation combined with active rehabilitation were the treatments of choice without the use of invasive procedures. The resolution of the presenting complaints was rapid.

We must not mistake the pain for the problem but must identify and then treat the dysfunction responsible for the pain.⁴ Unfortunately there remains a strong reluctance within the medical profession to accept altered function as an important cause of disease and suffering.⁴ Locomotor dysfunction has remained a medical no man's land, lost between such specialties as rheumatology, orthopaedics, neurology and rehabilitation medicine.⁴ A clear distinction between structural and functional pathology (dysfunction) is fundamental, for the diagnosis and management as well as for classification. As Lewit⁴ states, it can be compared only to the distinction between hardware and software.

Symptomatic treatments for pain control are important, however they should be used to promote active rehabilitation rather being the conclusion to treatment.⁴ Liebenson⁴ states that the pathoanatomical approach emphasizes treatment of the injured tissue while functional restoration

looks at the patient in a biopsychosocial context. Rehabilitation is more than exercise, it is a comprehensive management approach incorporating patient education, physical training and the identification of complicating (psychosocial) issues.⁴

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