Uncommon Nerve Compression Syndromes of the Upper Extremity

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Abstract

Nerve compression syndromes are a common cause of pain, sensory disturbance, and motor weakness in both the upper and the lower extremities. Although carpal tunnel syndrome is frequently diagnosed and treated surgically with success, other compression syndromes are less common and are often best treated nonsurgically. Understanding the anatomy of the major peripheral nerves with respect to intermuscular septa, fibrous bands, muscle margins, and internervous planes is crucial to understanding how and where peripheral nerve compression can occur. Some conditions, such as anterior interosseous nerve syndrome, respond well to nonoperative treatment; others, such as posterior interosseous nerve syndrome, are better treated by surgical intervention. The authors discuss the anatomic and pathologic causes for compression syndromes, as well as guidelines for treatment and outcomes.

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Compression syndromes of peripheral nerves have a number of possible causes. Pressure on a nerve may disrupt either the local blood flow or the axoplasmic flow to the nerve. Low blood pressure may diminish the blood supply of peripheral nerves and cause the familiar dysesthesias, paresthesias, and occasional motor weakness about which patients frequently complain. Direct pressure of 500 mm Hg or more may cause internal disruption of the axons.[1] Epineurial scarring may spontaneously form in peripheral nerves, with resultant symptoms of partial or complete compression (Fig. 1).[2,3]

Motor nerves, such as the posterior interosseous branch of the radial nerve and the anterior interosseous branch of the median nerve, contain stretch receptors, sensory fibers, and motor fibers. Therefore, pressure on a motor nerve may be interpreted as a painful stimulus by the brain. To describe the extent of compression and/or injury to a nerve, classification systems have been developed by Sunderland[4] (Table 1) and by Seddon[5] (Table 2). Peripheral nerve dysfunction secondary to viral illness or exposure to toxins, such as heavy metals, can mimic compression syndrome. Patients with systemic diseases, such as diabetes, may be more susceptible to nerve compression. Lifestyle and behavioral patterns may also influence the occurrence of nerve compression, as in the "Saturday night palsy" seen in alcoholics.

Radial Tunnel Syndrome

Pathoanatomy

The radial nerve is composed of fibers from the sixth, seventh, and eighth roots of the brachial plexus. These fibers course through the posterior divisions of the upper, middle, and lower trunks, forming the posterior cord and, subsequently, the radial nerve arising from the posterior cord. The nerve passes anterior to the subscapularis, teres major, and latissimus dorsi muscles, where the first potential site of compression may occur. Although rare, an anomalous muscle, the accessory subscapularis-teres-latissimus, has been reported to cause compression of the radial nerve at this level.[6] Spinner[7] has described penetration of the nerve directly by the subscapular artery more distally in the axilla, forming a neural loop and potentially resulting in compression. Exiting the axilla, the radial nerve courses laterally, passing through the triangular space and then proceeding through the lateral head of the triceps, where Lotem et al[8] and other

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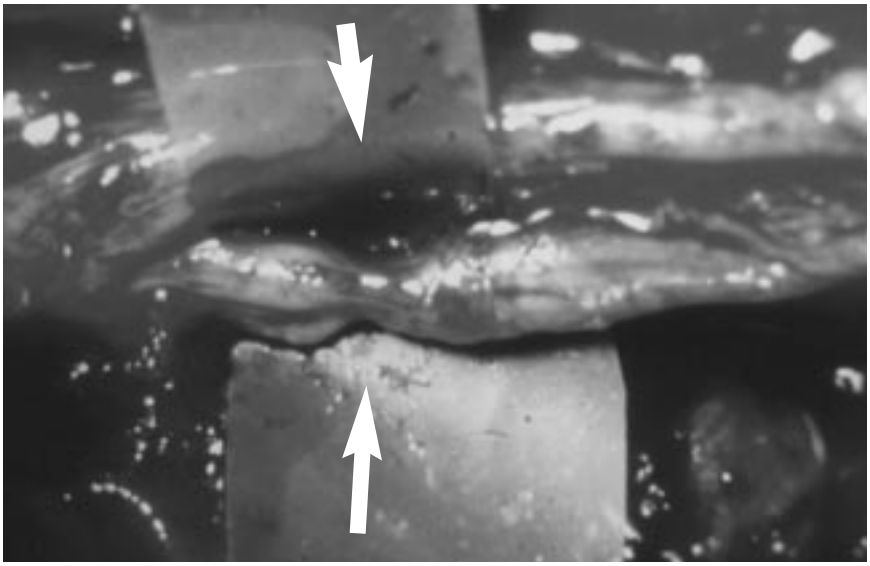

Fig. 1 Compressive lesion in the radial nerve. The constriction was intraepineurial, and the patient was treated with epineurolysis. Recovery was slow, and tendon transfers were performed. The lesion resolved over the course of 5 years, with electromyographic evidence of return of normal function. (Courtesy of Graham D. Lister, MD, Vero Beach, Fla.)

researchers have reported compression. (Lotem et al correlated the data obtained in cadaveric studies with findings from clinical studies. Surgery was not performed to verify the exact cause of the compression.)

Familial radial nerve entrapment has been reported secondary to compression at the lateral head of the triceps.[9] A genetic defect in Schwann cell myelin metabolism has also been postulated as a cause of radial tunnel syndrome.[10] Although this disorder may be asymptomatic, it can predispose the nerve to intermittent compression.

The nerve then courses distally along the humerus and passes from the posterior to the anterior compartment of the arm, where yet another potential site of compression, the lateral intermuscular septum, is found. Following the deep surface of the brachioradialis and the extensor carpi radialis longus muscles, the radial nerve bifurcates into a superficial branch and a deep branch. The superficial branch contains sensory fibers and continues beneath the brachioradialis into the forearm, passing between the brachioradialis and the flexor carpi radialis in the distal third of the forearm to lie superficial and subcutaneous. The deep branch of the radial nerve (the posterior interosseous branch) passes through the so-called radial tunnel, where it once again becomes subject to compression.

The radial tunnel is composed of the anatomic structures between the radiohumeral joint and the distal extent of the supinator muscle. Potential sites of compression causing radial tunnel syndrome include the fibrous margin of the extensor carpi radialis brevis muscle, fibrous bands at the level of the radiocapitellar joint, the radial recurrent artery, the arcade of Frohse proximally as the nerve passes distally through the supinator muscle, and a fibrous band at the distal margin of the supinator muscle.[11,12] Once through the supinator, the deep branch of the radial nerve divides into superficial and deep components. The superficial branch courses medially, innervating the extensor digito-

Table 1
Sunderland's Classification of Nerve Compression[4]

Grade	Description
1	Interruption of axial conduction at the site of injury. The axon remains in continuity; some segmental demyelinization may be present but not Wallerian degeneration. The condition is reversible.
2	The axon itself is no longer in continuity. The axon does not survive distal to the level of the injury and for a short distance proximal. The endoneurium is preserved. Full recovery may be expected.
3	The axon is severed, and Wallerian degeneration develops. The endoneurial tube is lost, and the fascicular anatomy is disturbed. Recovery is incomplete.
4	Total destruction of the internal architecture of the nerve. The trunk is intact, but a neuroma will form. Spontaneous recovery is rare. Surgical repair is indicated.
5	Loss of continuity of the nerve trunk. Surgical repair is mandatory.

Table 2
Seddon's Classification of Nerve Compression[5]

Type	Definition
Neurapraxia	Pressure on the nerve with resultant dysesthesias but no loss of continuity
Axonotmesis	The neural tube is intact, but the internal axons have been disrupted
Neurotmesis	The nerve itself has been completely divided

rum, the extensor digiti minimi, and the extensor carpi ulnaris muscles. The deep branch continues distally to supply the abductor pollicis longus, the extensor pollicis brevis, the extensor indicis proprius, and the extensor pollicis longus.

Clinical History and Symptoms

Pain is the most common primary presenting symptom in radial tunnel syndrome. There is some controversy concerning the existence of this syndrome because it is based essentially on the presence of localized pain without objective findings. It is the only nerve compression syndrome in which the signs and symptoms are not based on the nerve distribution.[13] A point of maximal tenderness is present at the site of compression, usually located over the anterior radial neck, in contrast to tennis elbow, in which pain is at the origin of the extensor carpi radialis brevis muscle. Compression of the "mobile wad" may also cause pain, as can resistance to active extension of the middle finger. Roles and Maudsley[14] were the first to describe radial tunnel syndrome in a patient with resistant tennis elbow.

At any of the potential sites, compression of the radial nerve is most significant with the elbow extended, the forearm pronated, and the wrist flexed. Active wrist extension and forearm supination against resistance may also reproduce the pain.[11] However, this maneuver (among others) will also cause pain with lateral epicondylitis. Forearm pain may be produced by resisted supination with the elbow extended or by resisted extension of the middle-finger metacarpophalangeal joint with the elbow extended and the forearm supinated. These maneuvers purportedly produce compression of the nerve by the fibrous arch of the supinator and extensor carpi radialis brevis muscles, respectively. Electrodiagnostic evaluations are negative in most cases of radial tunnel syndrome, but are positive in the presence of posterior interosseous nerve syndrome.

Management

Nonoperative treatment is important, and every effort should be made to modify patient activity to avoid provocative positioning of the arm. For example, if the patient's job requires elbow extension, forearm pronation, and wrist flexion repetitively or for long periods of time, an ergonomic evaluation should be completed, and every effort should be made to modify the task or change the job.

Symptomatic treatment should be attempted in all nerve compression syndromes, including radial tunnel syndrome, although the efficacy of such management is not well established. Treatment may include rest, stretching exercise, and splinting.[14,15] If symptoms have not improved after 6 to 12 weeks, a corticosteroid injection carefully placed adjacent to, but not within, the nerve is an acceptable therapeutic option.[14,15]

Surgical intervention may be considered if the symptoms are not relieved by rest, activity modification, nonsteroidal medication, or a cortisone injection. Before considering surgical treatment, precise localization of the pain to the region directly over the radial nerve within the radial tunnel must be confirmed. Lister et al[11] have recommended decompression of the radial nerve through a transverse incision at the level of the supinator when the surgeon is absolutely certain that the site of compression is the supinator. If doubt exists because of tenderness proximally over the radial nerve, a more extensile, bayonet-shaped incision beginning at the level of the lateral epicondyle and extending in a curvilinear fashion distally across the supinator muscle is necessary. Care should be taken to identify all potential sites of compression and to release the entire supinator, including its distal edge.[12]

Postoperative management includes use of a long-arm posterior splint with the wrist in neutral position. A gradual range-of-motion exercise program is begun at 1 week, with stretching exercise for the extensor muscles of the forearm. Return to unlimited activities can take 6 to 12 weeks, depending on job requirements. Patients who are receiving worker's compensation should be managed proactively with job modification. When time off work or operative treatment is required, it is important to establish agreement between patient and employer on a gradual return-to-work program.

Posterior Interosseous Nerve Syndrome

Pathoanatomy

Posterior interosseous nerve syndrome is the result of pressure on the nerve with secondary loss of motor function. Typical causes are elbow synovitis caused by rheumatoid arthritis and benign tumors, such as ganglions (Fig. 2) and lipomas. When compression within the radial tunnel is sufficient to cause paralysis but there is no palsy, the condition is termed posterior interosseous nerve syndrome, rather than radial tunnel syndrome.

Clinical History and Symptoms

Partial lesions occur when only one nerve branch is involved. Compression of the superficial or medial branch causes paralysis of the extensor carpi ulnaris, extensor digiti quinti, and extensor digitorum communis. Compression of the lateral branch causes paralysis of the abductor pollicis longus, extensor pollicis brevis, extensor pollicis longus, and extensor indicis proprius. Compression of the superficial branch affects the extensor communis, extensor digiti minimi, and extensor carpi ulnaris. Extensor carpi radialis longus function is preserved even in a complete palsy. The wrist extends and deviates radially because it is not opposed by the extensor carpi ulnaris and extensor carpi radialis brevis, which insert at more ulnar sites in the base of the small-finger and long-finger metacarpals, respectively.

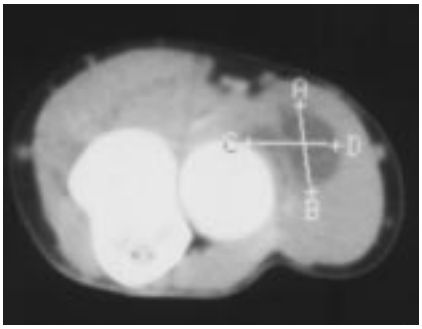

Fig. 2 This ganglion cyst in the proximal forearm resulted in posterior interosseous nerve palsy.

Posterior interosseous nerve compression may coexist with lateral epicondylitis. As part of the clinical history, other potential causes of peripheral neuritis, such as polyarteritis, rheumatologic disorders, and post–systemic illness angioneuropathy, should be considered.

Management

Initial nonoperative treatment should include rest, activity modification, and use of a wrist cock-up splint. A cortisone injection should also be considered. Regular gentle stretching of the wrist extensor muscles with the elbow held in full extension is begun after a spontaneous recovery.

If no improvement is seen within 90 days, spontaneous recovery is unlikely, and surgery should be performed. If the condition persists for 18 months or more, muscle fibrosis occurs, creating an irreversible condition.[16] In late cases, tendon transfers will be necessary.

There have been no long-term prospective outcome studies comparing operative and nonoperative treatment for either radial tunnel or posterior interosseous nerve entrapment. Lister et al[11] reported improvement in 19 of 20 patients with radial tunnel syndrome who were followed up 9 months to 4 years after surgical release. The outcome was particularly dependent on the correct preoperative diagnosis. Therefore, the surgeon's familiarity with the diagnosis and treatment of radial tunnel syndrome, lateral epicondylitis, and posterior interosseous nerve syndrome is critical in patient selection for surgical treatment and may influence the predictability of a successful outcome. A recent Mayo Clinic study cited a 51% success rate for surgery for radial tunnel syndrome and cited difficulty in differentiating radial tunnel syndrome from lateral epicondylitis as the reason for their mediocre results.[17] In our experience, while considerable improvement is often noted in selected patients, few return to work at their preoperative level, and none return to physically demanding jobs.

Late return of function was a feature of the patients in the initial description of the condition. With early diagnosis (within 3 months of onset of symptoms), a higher rate of spontaneous recovery can be expected. When an underlying cause, such as a lipoma or ganglion, is suspected, a magnetic resonance imaging study (Fig. 2) may confirm the diagnosis, and surgical decompression of the nerve, with removal of the lesion, is indicated. When no obvious anatomic lesion exists, decompression of the posterior interosseous nerve by release of the supinator and the arcade of Frohse may be considered if spontaneous return of function does not occur by 6 months. In the case of older patients and patients for whom recovery is less likely (those with no return of function after 18 months or more), tendon transfer should be performed.

Pronator Syndrome

Pathoanatomy

The median nerve is composed of fibers from the roots of the fifth, sixth, seventh, and eighth cervical nerves and the first thoracic nerve. To reach the median nerve, fibers from these nerve roots must pass through the anterior divisions of the upper, middle, and lower trunks and the lateral and medial cords of the brachial plexus. The

median nerve is formed anterior to the third portion of the axillary artery. It enters the upper arm behind the pectoralis major muscle, lateral to the brachial artery. The nerve passes with relative freedom through the upper portion of the arm, with the only potential sites of compression being the pectoralis minor muscle, anomalous muscles or blood vessels, and the deltopectoral fascia.[18]

Distally in the arm, a supracondyloid process (located medial and proximal to the medial epicondyle) and associated ligament of Struthers is a potential compression site. In 1% of upper extremities, a supracondyloid process (Fig. 3) exists where the ligament of Struthers originates.[16] More commonly, the ligament of Struthers originates from the humerus at the site where the supracondyloid process may occur and inserts in the medial epicondyle. There are no branches of the median nerve in the arm, except possibly a separate fascicular bundle that may leave the main trunk of the median nerve and innervate the pronator teres.[19]

Entering the forearm, the nerve is subject to potential compression by the lacertus fibrosus. The median nerve then passes beneath the humeral head of the pronator teres. In 6% of patients, the ulnar head is absent[20]; however, compression from the humeral head is still possible. The nerve then passes beneath the proximal fibrous arch of the two muscular heads of the flexor digitorum superficialis, where compression is also possible. The median nerve continues distally in the forearm between the flexor digitorum sublimis and the flexor digitorum profundus. The anterior interosseous nerve, which is the last major branch of the median nerve in the proximal forearm, supplies the flexor pollicis longus, the flexor digitorum profundus to the index and long fingers, and the pronator quadratus.

Additional sites of compression of the median nerve in the forearm include anomalous muscles (e.g., Gantzer's muscle, which is an anomalous flexor pollicis longus), a palmaris profundus muscle, and a flexor carpi radialis brevis muscle.[16] Anomalous arteries, such as an aberrant radial artery, or an enlarged bicipital bursa, may also cause pressure on the nerve.

Pronator syndrome is the result of compression of the median nerve between the two heads of the pronator teres muscle. Pronator syndrome commonly occurs with strenuous activities such as weight lifting and in occupations requiring repetitive pronation of the forearm with the elbow extended.

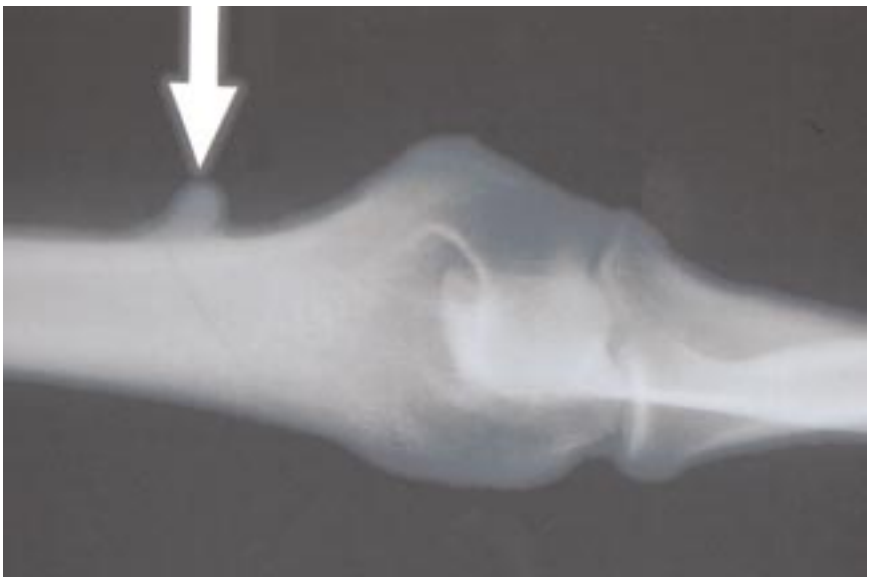

Fig. 3 Anteroposterior radiograph shows a characteristic supracondyloid process (arrow). When this appearance is seen in a symptomatic patient, removal of the supracondyloid process and the associated ligament of Struthers should be considered.

Clinical History and Symptoms

Pronator syndrome can be confused with carpal tunnel syndrome, as both may cause numbness and paresthesias in the median nerve–innervated digits, weakness of the thenar muscles, and pain in the wrist and forearm. Unlike carpal tunnel syndrome, there is no Tinel sign at the wrist. Dysesthesias are present in the palmar triangle or in the skin overlying the thenar eminence, as this is innervated by the palmar cutaneous branch of the median nerve, which originates proximal to the transverse carpal ligament. Furthermore, pronator syndrome does not produce nocturnal symptoms.[21] However, carpal tunnel syndrome and pronator syndrome may coexist, and the examiner should carefully evaluate the patient for the simultaneous presentation of both conditions.

Management

Although the diagnosis is rarely made, once it is established, surgical intervention is usually not necessary. The condition is typically treated with activity modification.

When nonoperative treatment fails or when space-occupying lesions exist, surgery may be indicated. In a review of the long-term results of surgical treatment of

pronator syndrome in 5 patients, Johnson et al[22] noted relief of pain in 4 (80%). In a Mayo Clinic study of 36 patients treated surgically, 8 had excellent results, 20 had a good outcome, and 5 had only fair results; the condition of the remaining 3 patients was unchanged.[21]

Anterior Interosseous Nerve Syndrome

Pathoanatomy and Diagnosis

Anterior interosseous nerve syndrome was first described by Tinel in 1918 and was further delineated by Kiloh and Nevin in 1952. In contrast to pronator syndrome, pain may be elicited by resisted flexion of the flexor digitorum sublimis of the long finger and may also be present at rest and on local palpation of the nerve. When the syndrome is complete, denervated muscles include the flexor pollicis longus, the two radial profundus tendons, and the pronator quadratus. No sensory changes occur, and the hand assumes a characteristic posture such that the patient is unable to position the thumb and index finger in the shape of a six (Fig. 4). The profundus tendon to the long finger is not always solely innervated by the median nerve, and the posture of the fingers in making a fist may resemble that seen with an isolated flexor digitorum profundus avulsion or laceration of the index profundus.

Association with absent flexor pollicis longus function establishes the diagnosis of anterior interosseous nerve syndrome. The pronator quadratus may be tested with the elbow held in a flexed position to neutralize the humeral head of the pronator teres muscle. Electromyographic (EMG) and nerve-conduction studies are often helpful in establishing the diagnosis. If the findings from electrodiagnostic studies are not consistent with anterior interosseous nerve syndrome, the surgeon should consider a more proximal cause of nerve compression, such as pronator syndrome, brachial plexopathy, or a tendon rupture, as occurs in patients with rheumatoid arthritis.

Management

Reports in the neurologic literature indicate that anterior interosseous nerve syndrome resolves spontaneously. In one series,[23] all patients recovered without surgical intervention. Miller-Breslow et al[24] believe that the condition is a neuritis. Regardless of the cause and management of the neuropathy, if motor function does not recover, tendon transfers will restore function satisfactorily.

Anterior interosseous nerve syndrome usually resolves with time, particularly if the lesion is secondary to neuritis. Observation for 3 to 6 months is favored before surgical treatment. Strengthening of remaining muscles and, occasionally, modalities such as heat and stretching are useful in most cases. If no improvement is noted, or if a space-occupying lesion is present, surgical release is recommended. If no improvement occurs after decompression, tendon transfer should be performed.[25]

Surgical exposure of the median nerve and its anterior interosseous branch is through an S-shaped incision that extends proximal to the elbow to allow exposure of the median nerve at the ligament of Struthers if necessary.[26] The nerve is then traced distally, passing beneath the lacertus fibrosus[27] and then between the humeral and ulnar heads of the pronator teres. The humeral head is taken down and tagged for later lengthening or reattachment. The median nerve may actually penetrate the pronator teres muscle. The safer approach to the median nerve is from the radial side, as nearly all branches of the median nerve arise on its ulnar side at this level, the most notable exception being the anterior interosseous branch, which originates on the radial side. In severe cases, neurolysis and pronator teres lengthening may be required.[28]

Fig. 4 Patients with anterior interosseous nerve palsy are asked to position their hand as shown. Those with absent profundus and flexor pollicis longus activity flex only the interphalangeal joint of the index finger and the metacarpophalangeal joint of the thumb.

Quadrilateral Space Syndrome

Pathoanatomy and Diagnosis

The axillary nerve originates from the C5-6 nerve roots and proceeds through the posterior division of the upper trunk, coursing to the posterolateral aspect of the posterior cord. In approximately 72% of cases, the axillary nerve separates from the posterior cord at the level of the coracoid, and the posterior cord becomes the radial nerve.[29] The axillary nerve then travels with the posterior circumflex humeral artery through the quadrilateral space, which is bounded by the long head of the triceps medially, the proximal humerus laterally, the teres major inferiorly, and the teres minor superiorly.

Idiopathic quadrilateral space syndrome is very uncommon.[29,30]

Vague shoulder discomfort and pain with fatigue occur when the patient holds the arm above shoulder level. The pain of axillary nerve compression is poorly localized to the shoulder. Paresthesias are present in a nondermatomal pattern. Discrete tenderness to palpation in the quadrilateral space and deltoid weakness are present. Objective evidence of compression is demonstrated by an arteriogram indicating compression of the posterior circumflex humeral artery during abduction of the shoulder.[31] Doppler studies may obviate the need for an arteriogram. The EMG findings are also diagnostic. From Erb's point distally, distances of 15 to 18 cm should have an average latency of 4.3 msec.[32] Any latency longer than 5 msec should be considered abnormal. The differential diagnosis includes thoracic outlet syndrome, suprascapular nerve entrapment, rotator cuff disease, and C5, C6, and C7 radiculopathies.

Management

Initial treatment is conservative, with muscle relaxants, nonsteroidal anti-inflammatory medication, rest, and cortisone injections. If there is no improvement after 3 to 6 months, operative treatment may be considered. A positive EMG, Doppler, or arteriographic study is also an indication for operative treatment.

Three surgical approaches—anterior, axillary, and posterior—are available. The anterior approach through the deltopectoral interval is not helpful in compressive cases because only the anterior portion of the space can be visualized. The anterior approach is useful when there has been penetrating trauma and when space-occupying lesions are present anteriorly. The axillary approach is excellent for exposure; however, care must be taken to avoid the intercostobrachial cutaneous nerve, as injury can produce considerable postoperative pain.[30] The posterior approach is made under the lower edge of the deltoid. Both transverse and vertical incisions have been described. This approach exposes the main area of compression in the posterior aspect of the space and is the preferred approach. Fibrous bands or an anomalous head of the triceps is usually responsible for the compression.[31]

The patient may begin active range-of-motion exercises as early as postoperative day 7. According to Dellon and Mackinnon,[20] this promotes axillary nerve gliding and prevents scarring. In one study, Cahill and Palmer[30] found that 89% of patients showed improvement of symptoms postoperatively.

Suprascapular Nerve Entrapment

Pathoanatomy

The suprascapular nerve is a mixed motor and sensory nerve that originates from the upper trunk of the brachial plexus. It leaves the trunk 3 cm above the clavicle and passes deep to the trapezius and omohyoid muscles on its way to the suprascapular notch. In the notch, the suprascapular nerve passes beneath the superior transverse scapular ligament, and the suprascapular artery and vein pass above the ligament. The nerve supplies one or two branches to the supraspinatus muscles and then passes into the infraspinatus fossa by proceeding around the lateral margin of the scapular spine.[33,34]

Suprascapular nerve entrapment is a cause of shoulder pain to be considered in the differential diagnosis with rotator cuff disease, impingement syndrome, acromioclavicular joint arthritis, and cervical radiculopathy.

Clinical History and Symptoms

Patients normally complain of a dull aching pain over the posterior or lateral aspect of the shoulder. This pain may radiate up the neck or down the lateral aspect of the arm. The etiology of suprascapular nerve entrapment is varied and may include sports activities such as weight lifting, volleyball, and baseball. Other causes include soft-tissue growths, such as ganglion cysts, and iatrogenic injury during rotator-cuff surgical mobilization for tears greater than 3 cm.[35]

Clinical diagnosis is difficult when muscle atrophy is not evident. Tenderness on palpation of the notch, differential injections, and the cross-body adduction test of the arm may help establish the diagnosis. Electromyographic and nerve-conduction studies are required and will be diagnostic. Magnetic resonance imaging will help rule out a rotator-cuff disorder and may reveal a ganglion cyst in the notch.[36]

Management

Treatment usually begins by eliminating the activity associated with the problem. Nonsteroidal anti-inflammatory drugs, analgesic agents, and trapezius-strengthening exercises are encouraged. Cortisone injections are also used. If 3 to 6 months of conservative therapy fails or if the initial EMG study is positive, demonstrating muscle fibrillation, operative intervention is warranted.[29] Three operative approaches to the suprascapular nerve are available. The posterior approach is generally used in cases of muscle atrophy or underdeveloped muscle. Otherwise, the muscle covers the notch and is difficult to retract.[37]

The cranial approach exposes the notch well, but distal dissection is difficult. Hadley et al[34] described a third approach midway

between the clavicle and the spine of the scapula through the trapezius muscle. The upper border of the scapula is palpated, and the notch is identified. Although exposure is difficult, the omohyoid muscle leads directly to the medial margin of the notch. Care must be taken to avoid the suprascapular artery and vein above the notch. The transverse scapular ligament is then transected, and the nerve is explored. Resection of the notch is controversial. Murray[37] has stated that osseous overgrowth may occur if the notch is resected, but Vastamäki and Göransson[38] recommend bone resection if the notch is narrow.

Postoperatively, the pain should be greatly diminished or completely relieved.[38] Muscle atrophy and weakness improve very slowly with physical therapy. The patient should begin active motion within 2 weeks after surgery.

Summary

The ability to identify potential causes of appendicular pain in the musculoskeletal system is crucial to the practicing orthopaedist. Although radicular pain from the cervical spine is a common cause of extremity pain and dysfunction, peripheral nerve compression with secondary dysfunction in the extremity should be considered in the differential diagnosis.

References

1. Ochoa J, Fowler TJ, Gilliat RW: Anatomical changes in peripheral nerves compressed by pneumatic tourniquet. *J Anat* 1972;113:433-455.
2. Burns J, Lister GD: Localized constrictive radial neuropathy in the absence of extrinsic compression: Three cases. *J Hand Surg [Am]* 1984;9:99-103.
3. Haussmann P, Patel MR: Intraepineurial constriction of nerve fascicles in pronator syndrome and anterior interosseous nerve syndrome. *Orthop Clin North Am* 1996;27:339-344.
4. Sunderland S: *Nerves and Nerve Injuries*, 2nd ed. Edinburgh: Churchill Livingstone, 1978, p 127.
5. Seddon HJ: *Surgical Disorders of the Peripheral Nerves*. Baltimore: Williams & Wilkins, 1972, pp 68-88.
6. Kameda Y: An anomalous muscle (accessory subscapularis-teres-latissimus muscle) in the axilla penetrating the brachial plexus in man. *Acta Anat (Basel)* 1976;96:513-533.
7. Spinner M: Management of nerve compression lesions of the upper extremity, in Omer GE Jr, Spinner M (eds): *Management of Peripheral Nerve Problems*. Philadelphia: WB Saunders, 1980, pp 569-587.
8. Lotem M, Fried A, Levy M, Solzi P, Najenson T, Nathan H: Radial palsy following muscular effort: A nerve compression syndrome possibly related to a fibrous arch of the lateral head of the triceps. *J Bone Joint Surg Br* 1971;53:500-506.
9. Lubahn JD, Lister GD: Familial radial nerve entrapment syndrome: A case report and literature review. *J Hand Surg [Am]* 1983;8:297-299.
10. Mayer RF, Garcia-Mullin R: Hereditary neuropathy manifested by pressure palsies: Schwann cell disorder? *Trans Am Neurol Assoc* 1968;93:238-240.
11. Lister GD, Belsole RB, Kleinert HE: The radial tunnel syndrome. *J Hand Surg [Am]* 1979;4:52-59.
12. Sponseller PD, Engber WD: Double-entrapment radial tunnel syndrome. *J Hand Surg [Am]* 1983;8:420-423.
13. Verhaar J, Spaans F: Radial tunnel syndrome: An investigation of compression neuropathy as a possible cause. *J Bone Joint Surg Am* 1991;73:539-544.
14. Roles NC, Maudsley R: Radial tunnel syndrome: Resistant tennis elbow as a nerve entrapment. *J Bone Joint Surg Br* 1972;54:499-508.
15. Eaton CJ, Lister GD: Radial nerve compression. *Hand Clin* 1992;8:345-357.
16. Spinner M: *Injuries to the Major Branches of Peripheral Nerves of the Forearm*, 2nd ed. Philadelphia: WB Saunders, 1978, p 234.
17. Ritts GD, Wood MB, Linscheid RL: Radial tunnel syndrome: A ten-year surgical experience. *Clin Orthop* 1987; 219:201-205.
18. Mayfield FH: Compression syndromes of the shoulder girdles and arms, in Vinken PJ, Bruyn GW (eds): *Diseases of the Nerves*. New York: North-Holland Publishing, 1970, pp 441-444. Handbook of Clinical Neurology series, vol 7, pt 1.
19. Inglis AE: Surgical exposure of the peripheral nerves, in Omer GE Jr, Spinner M (eds): *Management of Peripheral Nerve Problems*. Philadelphia: WB Saunders, 1980, pp 317-331.
20. Dellon AL, Mackinnon SE: Musculoaponeurotic variations along the course of the median nerve in the proximal forearm. *J Hand Surg [Br]* 1987;12:359-363.
21. Hartz CR, Linscheid RL, Gramse RR, Daube JR: The pronator teres syndrome: Compressive neuropathy of the median nerve. *J Bone Joint Surg Am* 1981;63:885-890.
22. Johnson RK, Spinner M, Shusberry MM: Median nerve entrapment syndrome in the proximal forearm. *J Hand Surg [Am]* 1979;4:48-51.
23. Nakano KK, Lundergran C, Okihiro MM: Anterior interosseous nerve syndromes: Diagnostic methods and alternative treatments. *Arch Neurol* 1977; 34:477-480.
24. Miller-Breslow A, Terrono A, Millender LH: Nonoperative treatment of anterior interosseous nerve paralysis. *J Hand Surg [Am]* 1990;15:493-496
25. Burkhalter WE: Median nerve palsy, in Green DP, Hotchkiss RN (eds): *Operative Hand Surgery*, 3rd ed. New York: Churchill Livingstone, 1993, vol 2, p 1442.
26. Eversmann WW Jr: Entrapment and compression neuropathies, in Green DP, Hotchkiss RN (eds): *Operative Hand Surgery*, 3rd ed. New York: Churchill Livingstone, 1993, vol 2, pp 1343-1344.
27. Swiggett R, Ruby LK: Median nerve compression neuropathy by the lacertus fibrosus: Report of three cases. *J Hand Surg [Am]* 1986;11:700-703.
28. Dawson DM, Hallet M, Millender LH: Radial nerve entrapment, in Dawson DM (ed): *Entrapment Neuropathies*, 2nd ed. Boston: Little Brown, 1990, p 217.
29. Narakas A: Compression syndromes about the shoulder including brachial plexus, in Szabo RM (ed): *Nerve Compression Syndromes: Diagnosis and Treatment*. Thorofare, NJ: Slack, 1989, pp 227-246.

30. Cahill BR, Palmer RE: Quadrilateral space syndrome. *J Hand Surg [Am]* 1983;8:65-69.
31. Francel TJ, Dellon AL, Campbell JN: Quadrilateral space syndrome: Diagnosis and operative decompression technique. *Plast Reconstr Surg* 1991;87: 911-916.
32. Gassel MM: A test of nerve conduction to muscles of the shoulder girdle as an aid in the diagnosis of proximal neurogenic and muscular disease. *J Neurol Neurosurg Psychiatry* 1964;27:200-205.
33. Bigliani L, Dalsey RM, McCann PD, April EW: An anatomical study of the suprascapular nerve. *Arthroscopy* 1990;6:301-305.
34. Hadley MN, Sonntag VKH, Pittman HW: Suprascapular nerve entrapment: A summary of seven cases. *J Neurosurg* 1986;64:843-848.
35. Warner JJP, Krushell RJ, Masquelet AJ, Gerber C: Anatomy and relationships of the suprascapular nerve: Anatomical constraints to mobilization of the supraspinatus and infraspinatus muscles in the management of massive rotator-cuff tears. *J Bone Joint Surg Am* 1992;74:36-45.
36. Fritz RC, Helms CA, Steinbach LS, Genant HK: Suprascapular nerve entrapment: Evaluation with MR imaging. *Radiology* 1992;182:437-444.
37. Murray JWG: A surgical approach for entrapment neuropathy of the suprascapular nerve. *Orthop Rev* 1974;3:33-35.
38. Vastamäki M, Göransson H: Suprascapular nerve entrapment. *Clin Orthop* 1993;297:135-143.