Current Approach to Radial Nerve Paralysis

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Learning Objectives: After studying this article, the participant should be able to: 1. Identify all potential points of radial nerve compression and other likely causes of radial nerve injury. 2. Accurately diagnose both surgical and nonsurgical causes of radial nerve paralysis. 3. Define a safe and effective approach to the surgical release and reconstruction of the radial nerve.

Radial nerve paralysis, which can result from a complex humerus fracture, direct nerve trauma, compressive neuropathies, neuritis, or (rarely) from malignant tumor formation, has been reported throughout the literature, with some controversy regarding its diagnosis and management. The appropriate management of any radial nerve palsy depends primarily on an accurate determination of its cause, severity, duration, and level of involvement. The radial nerve can be injured as proximally as the brachial plexus or as distally as the posterior interosseous or radial sensory nerve. This article reviews the etiology, prognosis, and various treatments available for radial nerve paralysis. It also provides a new classification system and treatment algorithm to assist in the management of patients with radial nerve palsies, and it offers a simple, five-step approach to radial nerve release in the forearm. (Plast. Reconstr. Surg. 110: 1099, 2002.)

The radial nerve is the most frequently injured major nerve in the upper extremity.1 Radial nerve paralysis generally can be divided into either open or closed injuries. All open injuries require exploration, whereas most closed injuries can usually be observed. The most common associated cause of radial nerve injury is a fracture to the shaft of the humerus. Iatrogenic injuries to the radial nerve may occur during complex and routine procedures of the upper extremity. Acute trauma from lacerations, missiles, injections, or traction can also result in radial nerve paralysis. Chronic or acute radial nerve compression can initiate a wide range of clinical symptoms, from weakness in wrist extension to complete radial nerve paralysis. Rarely, a neuritis or tumor of the radial nerve at levels as high as the brachial plexus will present with radial nerve palsy.

Treatment options for radial nerve paralysis are dependent upon the primary cause and level of injury. A radial nerve paralysis without an associated laceration or penetration is considered “closed.” Fractures of the humerus may result in a closed radial nerve injury that is typically observed for a period of 3 months before surgical exploration. Idiopathic causes of radial nerve paralysis can be treated conservatively after treatable causes such as tumors have been excluded. The surgical options for open radial nerve injuries that result in a loss of nerve continuity include primary repair, nerve grafts, or tendon transfers. Recently, nerve transfer has been reported as a potential alternative to tendon transfer after the complete loss of radial nerve function or a significant delay in treatment.2

RELEVANT ANATOMY

The radial nerve exits the brachial plexus from the posterior cord with contributions from C5, C6, C7, C8, and T1, traveling dorsal to the axillary artery and vein and closely abutting the shaft of the humerus near the spiral groove. Whitson3 has shown that the nerve actually lies several centimeters distal to the spiral groove, separated from the bone by a thin layer of the medial head of the triceps. It then runs posterolaterally, in proximity to the deep brachial artery beneath the lateral head of the
The triceps, traveling along the anterior surface of the lateral intermuscular septum.

The radial nerve gives off branches to the extensor carpi radialis longus and brachioradialis as it enters the antebrachial fossa between the biceps and brachialis medially and the brachioradialis laterally. As it passes over the elbow joint, it divides into a terminal motor and sensory branch at the level of the radiohumeral joint, but the exact site may vary by as much as 5 cm.\textsuperscript{4,5} The motor branch is the posterior interosseous (or deep radial) nerve, and the sensory branch is the superficial radial nerve.

The superficial branch of the radial nerve runs into the forearm under the brachioradialis before innervating the radial aspects of the dorsal wrist and hand. The posterior interosseous nerve travels a short distance over the radiohumeral joint, passing dorsolaterally around the radial head before entering the substance of the supinator.\textsuperscript{6} The nerve then winds around the neck of the radius to travel on the dorsal surface of the interosseous membrane.

The posterior interosseous nerve supplies the majority of forearm and hand extensors (including the extensor carpi radialis brevis, supinator, extensor digitorum communis, extensor digiti quinti, extensor carpi ulnaris, abductor pollicis longus, extensor pollicis longus and brevis, and extensor indicis proprius), with the exception of the extensor carpi radialis longus and brachioradialis. The normal course of the posterior interosseous nerve is through the supinator brevis muscle. Two anomalous courses of the nerve have been described by Woltman and Learmonth.\textsuperscript{7} One anomaly occurs in substance of the supinator, and the other involves a branch traveling superficial to the supinator brevis.

**HISTORICAL PERSPECTIVE**

In 1863, Agnew\textsuperscript{8} explored the forearm of a patient with flexor and extensor weakness and found a mass compressing the posterior interosseous and median nerves. The patient reportedly recovered after removal of the solid mass. Weinberger quoted Nancrede’s observation of a patient with a bursa between the common extensor muscles and the extensor carpi ulnaris compressing the posterior interosseous nerve.\textsuperscript{9–11} In 1905, Guillain and Courtellemont\textsuperscript{12} reported a case of radial nerve palsy that they believed to be related to repeated pronation and supination in a musical conductor. Grigoresco and Iordanesco\textsuperscript{13} reported a case of posterior interosseous nerve palsy after a minor injury was made worse by the patient sleeping with his head on his forearm.

At the Mayo Clinic in 1934, Woltman and Learmonth\textsuperscript{7} reported five cases of idiopathic posterior interosseous nerve paralysis that did not improve with observation or surgery. The exploration of one patient demonstrated an anatomic abnormality, with the radial nerve lying entirely superficial to the supinator muscle. Hobhouse and Heald\textsuperscript{14} reported an isolated case of idiopathic posterior interosseous paralysis in 1936, and Otenasek\textsuperscript{15} reported a case of posterior interosseous nerve palsy in 1947 that seemed to result from swelling around the nerve at exploration.

Various authors throughout the early 1900s reported injury to the radial nerve after elbow trauma.\textsuperscript{16,17} Richmond\textsuperscript{18} and Hustead et al.\textsuperscript{19} reported a case of radial nerve paralysis related to soft-tissue masses, and Kruse\textsuperscript{20} reported a case of radial nerve palsy in a swimmer that resolved within 3 months without surgical intervention. By the middle of the 20th century, the symptoms associated with radial nerve palsy had been fully described, but its specific pathogenesis remained unclear.

**PATHOGENESIS OF RADIAL NERVE PALSY**

Radial nerve paralysis may not always have a clear origin. The radial nerve may be paralyzed at any point along its course from the brachial plexus to the hand, resulting in a similar clinical presentation regardless of the cause. Determining the level of injury often assists the physician in identifying the origin of the disorder. Several distinct causes of radial nerve palsy have been clearly identified in the literature. A review of these potential causes will assist the physician in determining the most appropriate surgical option.

**Orthopedic Injury**

The radial nerve is injured through orthopedic trauma more than any other major nerve.\textsuperscript{21,22} It is estimated that over 237,000 humeral fractures occur each year in the United States, with 75 percent of these fractures involving the humeral shaft.\textsuperscript{21} Approximately 12 percent of humeral shaft fractures are complicated by a radial nerve paralysis.\textsuperscript{1,23,24} Spontaneous recovery within 8 to 16 weeks has
been reported in over 70 percent of the cases.\textsuperscript{21,23} Rarely, the radial nerve can become entrapped in the bony fragments or callous after humeral fractures. In particular, spiral fractures of the distal shaft of the humerus with radial angulations have been associated with radial nerve paralysis.\textsuperscript{25} Bateman\textsuperscript{26} reported that radial nerve paralysis occurred more commonly after open reduction of humeral fractures. Radial nerve paralysis associated with dislocation of the radial head is believed to result from traction on the nerve within the substance of the supinator, but the paralysis is usually transient.\textsuperscript{26} It is important to understand the vulnerability of the posterior interosseous nerve during surgical exploration of the elbow and its susceptibility to compression in this area.

There is a prognostic difference between primary (occurring after trauma) and secondary (occurring after treatment) radial nerve paralysis following humeral fractures. Shaw and Sakellarides\textsuperscript{27} reported spontaneous recovery in only 40 percent of patients with primary paralysis, but in all patients with secondary paralysis after closed or open reduction internal fixation. Garcia and Maeck\textsuperscript{28} reviewed over 226 patients with fractures of the humeral shaft. They found that immediate radial nerve palsy occurred in 11.7 percent of patients with humeral shaft fractures. Twenty-three percent of the patients explored developed secondary radial nerve paralysis, with 10 percent of these patients experiencing persistent symptoms.

Monteggia fractures involve dislocation of the radial head and fracture of the ulna. These fractures may be complicated by a radial nerve palsy at the level of the posterior interosseous nerve.\textsuperscript{29-31} A radial nerve injury is more common in humeral fractures that are associated with a radial head dislocation because of the intimate anatomic relationship between the nerve and the radial head.\textsuperscript{29,32,33} In this type of injury, the radial nerve may be stretched, compressed, or lacerated. Lichter and Jacobsen\textsuperscript{34} reported a delayed posterior interosseous nerve palsy that occurred over 30 years after a Monteggia fracture.

The radial nerve may be inadvertently divided during any surgical procedures performed in proximity to the nerve. Most postoperative radial nerve paralysis results from traction during exposure. Forearm pronation causes the posterior interosseous nerve to move medially, thereby protecting it during radial head exposure.\textsuperscript{1,37} Special care must be taken when using a posterolateral approach to the proximal radial shaft because of the proximity of the radial nerve to this area. Of course, the superficial branch of the radial nerve is especially susceptible to injury with transverse incision of the dorsoradial wrist and hand.

**Tumor and Inflammation**

Radial nerve compression can occur as the result of either a benign tumor or a malignancy. The radial nerve is more susceptible to external compression near bones or joints where ganglions can form. Benign tumors arising from the elbow or at the upper end of the radius have been reported to cause posterior interosseous nerve paralysis.\textsuperscript{36,37} Lipomas are the most common tumors reported in the literature to cause radial nerve palsy.\textsuperscript{18,38} Sharrard\textsuperscript{39} reported a case of posterior interosseous nerve palsy related to a fibroma, Bowen and Stone\textsuperscript{40} a case of radial nerve paralysis caused by a ganglion at the level of the elbow, and Dharapak and Nimberg\textsuperscript{41} a case of posterior interosseous nerve compression resulting from a mass effect of a traumatic aneurysm.

In 1966, Capener\textsuperscript{38} published a case report and anatomic study of the vulnerability of the posterior interosseous nerve in the forearm. Although the case involved a posterior interosseous nerve paralysis related to a tumor, in his discussion the author referred to an association between radial nerve symptoms and tennis elbow. The decompression of the radial nerve with a release of the septum of the extensor digitorum communis and supinator in 10 patients with tennis elbow resulted in complete resolution of clinical symptoms. Roles and Maudsley\textsuperscript{42} also reported significant improvement in clinical symptoms in patients with resistant tennis elbow who underwent simultaneous release of the radial nerve.

Weinberger\textsuperscript{9} described a case of posterior interosseous nerve palsy believed to result from inflammation of the bicipitoradial and interosseous bursa of the forearm. Posterior interosseous nerve palsy has also been reported in patients with a history of rheumatoid arthritis involving the elbow.\textsuperscript{43} Marmor et al.\textsuperscript{44} reported a case of radial nerve paralysis caused by rheumatoid arthritis that resulted in dislocation of the radial head.

Radial nerve palsy may also result from nerve...
Peripheral nerve tumors can be classified into neoplasms of the nerve sheath, neoplasms nerve cell origin, metastatic tumors, neoplasms of nonneural origin, and neuromas. Unless they are associated with significant compression, benign nerve tumors such as schwannomas, neurofibromas, or intraneural lipomas rarely result in complete nerve paralysis. Nerve tumors that do result in paralysis should be fully evaluated because, although rare, they are more likely to be malignant (as with malignant schwannomas or nerve sheath fibrosarcomas). An unusual benign surgical entity of the nerve also worth noting is hypertrophic neuropathy (or “onion whorl disease”), which is characterized as a localized enlargement of the nerve resulting in the progressive loss of nerve function.

Anatomic Compressions

Nontraumatic radial nerve palsy is rare compared with paralysis resulting from orthopedic trauma. The cause of spontaneous paralysis of the posterior interosseous nerve or the radial nerve is often unknown. Nevertheless, acute nerve paralysis requires investigation to rule out treatable causes of the disease such as neuritis, tumors, or compression. Neuritis, for example, is usually associated with several weeks of severe pain.

Many authors have sought an explanation for radial nerve paralysis without discovering a clear etiology. In 1905, Gullain and Courtellemont reported a case of posterior interosseous nerve paralysis in a patient who denied any history of trauma, and Whitely and Alpers presented a case of posterior interosseous nerve palsy associated with a spontaneous neuroma. Sharrard reported the improvement of an idiopathic posterior interosseous neuritis after the release of a crossing fibrous band, and Capener likened radial nerve compression to carpal tunnel syndrome. Recently, there has been increased interest in the association of this disease with idiopathic compressive neuropathies and the entity of hereditary neuropathy, in which some individuals may have a particular sensitivity to the development of compressive neuropathies.

The radial nerve can be compressed at multiple points along its course. At the elbow, for example, it can be compressed by the fibrous bands proximal to the radial tunnel, the vascular leash of Henry (the radial recurrent artery), the tendinous margin of the extensor carpi radialis brevis, and the arcade of Frohse (the tendinous superficial head of the supinator). Posterior interosseous nerve syndrome is a radial nerve paralysis that is believed to result from compression of the deep branch of the radial nerve at the level of the arcade of Frohse.

Arcade of Frohse. Spinner’s classic description of the arcade of Frohse as a mechanism to posterior interosseous nerve paralysis succeeded in providing a reasonable explanation for the syndrome. Spinner noted that radial nerve paralysis resulted from a narrowing at the leading edge of the superficial head of the supinator caused by adjacent structures, neoplasms, or inflammation. The arcade of Frohse was noted in approximately 30 percent of adults, but was not identified in any fetal dissections. Spinner theorized that repeated rotary movement of the forearm resulted in a thickening of the superficial head of the supinator.

Spinner’s theory of radial nerve compression by the arcade of Frohse has been confirmed over the years. Goldman et al. reported compression of the posterior interosseous nerve at the arcade of Frohse, which was identified by electromyographic examination. Although Nielsen described four cases of posterior interosseous nerve paralysis that were improved by releasing the fibrous band at the supinator muscle, Bryan et al. found no improvement in a patient with radial nerve paralysis 10 months after the release of the arcade of Frohse.

The size of the opening of the arcade of Frohse is variable. The posterior interosseous nerve travels beneath the proximal edge of the superficial supinator and can be further narrowed by the tendinous medial border of the extensor carpi radialis brevis. A full release of the fascial edge of the extensor carpi radialis brevis will not only improve exposure, it will also help to decrease compression of the nerve at this level and relieve any associated symptoms of lateral epicondylitis.

Triceps compression. Reports of transient palsies of the radial nerve after strenuous muscle activity have been attributed to compression of the nerve by the lateral head of the triceps muscle. Lotem et al. were the first to describe a radial nerve palsy resulting from a fibrous arch formed by the lateral head of the triceps. This arch was reportedly located approximately 2 cm distal to the deltoid insertion or the lateral border of the humerus. Although the injury was...
thought to be a radial neuropraxia that did not require surgical intervention, Manske later reported a permanent paralysis of the radial nerve in this area after strenuous activity, requiring surgical exploration and release. As far back as 1945, Sunderland reported radial nerve paralysis that had resulted from nerve compression at the edge of the latissimus dorsi tendon and long head of the triceps from using crutches.

Open Wounds

The radial nerve is seldom divided in traumatic lacerations because of the deep position of the motor component of the nerve. Nevertheless, penetrating wounds associated with radial nerve palsy require surgical exploration to rule out axonotmesis. When the radial nerve is sharply divided, the clinical results after primary repair have been good to fair in most instances, if the nerve is explored early and repaired without tension. It is of particular interest that the radial nerve has been found to be transected in up to 50 percent of patients following gunshot wounds associated with humeral fractures.

The treatment of postoperative radial nerve palsy requires a great deal of consideration. To identify nerve injuries in advance and to avoid any questions of origin after surgery, it is important to do a complete upper extremity nerve exam before any invasive procedure is performed in proximity to major nerves. The radial nerve can be injured inadvertently during orthopedic or vascular access procedures. A previous surgical scar or an exploration of the arm without the aid of a tourniquet often prevents a clear identification of nerves during dissections. Radial nerve paralysis may occur as the result of a traction injury or compressive neuropraxia after surgery. A thorough and honest reflection by the surgeon is required when a radial nerve paralysis is noted after surgery. If there is any chance that the nerve may have been severed or partially transected, immediate re-exploration is indicated to ensure the best clinical outcome.

Other Causes

Radial nerve paralysis has been reported in association with the use of a tourniquet or with injection injuries. The radial and sciatic nerves are those most commonly injured by injections. The high percentage of injuries to these nerves can be attributed to their proximity to common intramuscular injection sites. Tourniquets are used routinely in the operating room to provide a bloodless field, and nerve paralysis after tourniquet use is 2.5 times more common in the upper extremity than in the lower extremity. Upper extremity nerve paralysis is estimated to occur in one out of 13,000 applications. The radial nerve is the nerve most susceptible to tourniquet trauma, with its involvement present to some extent in up to 96 percent of upper extremity tourniquet injuries.

Postoperative radial nerve palsies can also be related to patient positioning or to blood pressure cuffs. Radial nerve paralysis may develop during sleep, especially when the patient is intoxicated, such as in a "Saturday night palsy." Sunderland described a variety of radial nerve injuries resulting from compression caused by local trauma and ischemia. He reported seven cases of radial nerve palsy, five of which occurred during sleep with compression noted at the level of the lateral intermuscular septum. All seven patients experienced complete resolution of paralysis with time. Traction injuries after motor vehicle accidents or other blunt traumas may result in prolonged paralysis of the radial nerve. Most of these nerve lesions can be treated conservatively if they are "closed" and if axonotmesis is not suspected. Patients who develop a spontaneous neuropathy may also have a susceptibility to other compressive neuropathies (i.e., hereditary neuropathy).

Radial nerve paralysis has been reported as the result of pentazocine-induced fibrous myopathy. Repeated pentazocine injections to the deltoid region was noted to induce triceps fibrosis, causing radial nerve compression. The patient developed a persistent wrist drop that required surgical decompression. Radial mononeuritis has also been related to alcohol, lead, arsenic, typhoid, and serum sickness. Idiopathic radial neuritis has been described throughout the literature, but its specific cause has yet to be clearly defined.

Clinical Evaluation

Clinical presentation of radial nerve paralysis is dependent upon the cause and level of nerve involvement. Some patients have symptoms of chronic compression that progress to complete paralysis, whereas other patients experience
the acute onset of paralysis. All lesions of the radial nerve must first be distinguished from lesions affecting the nerve roots (C5 through T1) or the brachial plexus. The clinical symptoms of radial nerve paralysis are often straightforward, with patients primarily demonstrating a motor deficit of wrist and finger extension. Radial nerve injuries usually result in a decrease in power grip and pinch primarily related to the loss of wrist extension.

The level of injury can often be determined by physical examination of the motor and sensory components. Anesthesia after radial nerve palsy may vary to include the dorsal surface of the proximal half of the thumb, index, and middle fingers and is usually limited to a small, triangular area on the dorsum of the first and second metacarpal web spaces. Because the sensory deficit is not on the tactile surface of the hand, the sensory deficit is usually trivial, but can be painful.

Loss of motor function depends on the level of the radial nerve lesion. Although loss of the anconeus muscle is not clinically noticeable after proximal radial nerve paralysis, the other muscles in the upper arm can be more clearly identified on physical examination. These muscles include the triceps, brachioradialis, and extensor carpi radialis longus and brevis. Loss of triceps function reflects an injury at the level of the brachial plexus. If the brachioradialis or extensor carpi radialis longus are not functional, then the injury is most likely at the level of the humeral shaft. Proximal radial nerve injuries result in a complete loss of extension at the wrist and metacarpophalangeal joints along with a loss in extension and abduction of the thumb.

Posterior interosseous nerve paralysis typically involves a more distal injury to the radial nerve. Patients with classic posterior interosseous nerve palsy experience radial deviation of the wrist with dorsiflexion because of the preservation of the extensor carpi radialis longus. These patients are usually unable to extend their fingers or thumb at the metacarpophalangeal joints, and they have no sensory deficit because the superficial radial nerve is preserved. The presence of active contractions of the brachioradialis and wrist extension in radial deviation allows for localization of the radial nerve injury to a point distal to the origin of the posterior interosseous nerve.

**Diagnostic Studies**

A complete patient history and a physical examination are often all that is needed to determine the level of injury and the suspected cause of radial nerve paralysis. A plain film of the involved area should be obtained if a fracture, dislocation, or foreign body is suspected. Plain x-rays of the elbow can be especially useful in ruling out more complicated orthopedic injuries or disorders of the radial head. Magnetic resonance imaging should be obtained if a mass is suspected at any level along the course of the radial nerve. Nevertheless, studies such as magnetic resonance imaging that are used for diagnostic purposes in patients with radial nerve paralysis may also be misleading without clinical confirmation.

All patients experiencing neural compromise after penetrating injury in proximity to nerves should be explored without the need for preoperative electrodiagnostic studies. Plain films, magnetic resonance imaging, or arteriograms may be indicated before exploration in some situations, but electrodiagnostic studies are rarely helpful within the first several weeks after nerve injury because these studies cannot differentiate between nerve injuries that will or will not recover spontaneously with time. Standard electrodiagnostic studies will, however, help to determine the level of injury or its distribution if the physical examination is unclear. Patients with nerve paralysis that persists beyond 6 to 8 weeks should be examined with electrodiagnostic studies. By 12 weeks, motor unit potentials will be present and will help to differentiate between recoverable injuries and those that will require surgery.

Intraoperative nerve-to-nerve studies are an excellent adjunct in the care of patients with peripheral nerve injuries. The results of these studies can assist the surgeon in the operative plan as long as the injury has been allowed to fully mature. The level and extent of a radial nerve lesion can be determined in the operating room using this technology. Intraoperative nerve-to-nerve studies can be used to examine the nerve proximal and distal to a suspected lesion. If the radial nerve demonstrates conduction across a lesion, spontaneous recovery is possible. If conduction is absent proximal to a lesion, a nerve repair or graft may not be indicated because either a more proximal injury must be present or the nerve is without function.
CLASSIFICATION

A conservative approach to the restoration of hand function after radial nerve paralysis has long been advocated in the literature. In the middle of the 20th century, many authors performed operations to remove offending factors causing radial nerve paralysis, but no one reported functional improvement after surgery.7,11,15 Sharrard39 reported one case of radial nerve palsy that required up to 4 years to recover. Clearly, there were no established guidelines at this time for the treatment of idiopathic radial nerve palsy. In 1968, however, Spinner6 began to advocate surgical exploration of patients with radial nerve palsy 6 to 8 weeks after clinical presentation of the disease.

Currently, most surgeons agree that once the diagnosis of radial nerve palsy is made, close observation is indicated. All patients require a wrist splint and hand therapy to prevent joint stiffness or permanent loss of function; however, controversy still exists as to the length of time a patient should be observed before surgical intervention.4,37,45 The confusion surrounding the appropriate clinical treatment of radial nerve palsy often stems from the lack of a clear and relevant classification system. Clearly, it is important to categorize all radial nerve lesions based on the type of injury and the level of involvement. A complete history and physical are often all that is required for complete classification, but electrophysiologic or other studies may be needed in some situations. Once the radial nerve injury is classified, the appropriate treatment options can be applied based on the information available.

In general, all nerve injuries can be classified as first- through sixth-degree injury.45,70 First-degree injury is a neurapraxia that results from a segmental demyelination without loss of nerve continuity or Wallerian degeneration. Second-degree injury is axonotmesis with injury to the axon, but intact endoneurial tissue and Schwann cell tubes. Third-degree injury involves additional injury to the endoneurium, but the perineurium remains intact. Fourth-degree injury involves a neuroma in continuity with complete scar block of nerve function. Fifth-degree injury is a transected nerve. Sixth-degree injury has been described and popularized by Mackinnon and Dellon45 as a combination of any of the above injuries.

After determining the degree of nerve injury, we developed a classification system for radial nerve paralysis to further assist with its clinical diagnosis and intervention (Table I). This new classification system takes into account other relevant factors critical to the successful management of patients with radial nerve palsy. All radial nerve injuries must first be classified as either open or closed. It is important to make a distinction between open and closed injuries because the management is quite different based on the degree of nerve injury most likely associated with each disorder. Closed injuries include radial nerve paralysis associated with orthopedic trauma, compression, neuritis, or idiopathic causes. Open injuries include lesions associated with penetrating wounds, lacerations, or surgical explorations in proximity to the radial nerve. If there is even the slightest concern that the nerve may be lacerated by bone fragments or surgical trauma, then the nerve should be explored.

The approximate level of radial nerve injury should be determined next. The radial nerve may be injured or paralyzed at different points along its course, but there are several specific points at which it is more susceptible to injury. The nerve may be injured along the spiral groove, near the radial head, or at the arcade of Frohse.71 We have defined the level of radial nerve injury to be a high injury when it occurs above the level of insertion of the pectoralis major muscle to the humerus, an intermediate injury when it occurs between the insertion of the pectoralis major and the posterior interosseous nerve, and a low injury when it involves the posterior interosseous nerve. The level of involvement can usually be determined by physical examination or electrophysiologic studies.

Radial nerve paralysis can also be classified as

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TABLE I

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<tr>
<th>Injury Classification</th>
<th>Explanation of Classification</th>
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<tr>
<td>Open versus closed</td>
<td>Injury is distinguished as either penetrating or nonpenetrating</td>
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<tr>
<td>High</td>
<td>Injury occurs above the distal insertion of the pectoralis major</td>
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<tr>
<td>Intermediate</td>
<td>Injury occurs between the insertion of the pectoralis major and</td>
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<td>the posterior interosseous nerve</td>
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<tr>
<td>Low</td>
<td>Injury involves the posterior interosseous or radial sensory nerve</td>
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<tr>
<td>Compressive</td>
<td>Injury involves chronic or acute anatomic compression</td>
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<tr>
<td>Delayed presentation</td>
<td>Injury involves a delay in presentation that significantly limits the available treatment options</td>
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either acute or chronic compression injuries. There are several important areas in which the radial nerve is particularly susceptible to either acute or chronic compressive neuropathy. For instance, the posterior interosseous nerve can be compressed by the fascia of the radiocapitellar joint, the leash of Henry, the fascia of the extensor carpi radialis brevis, the arcade of Frohse, or the distal edge of the supinator. The radial nerve can be compressed above the elbow by the lateral head of the triceps or humeral exostosis. Radial nerve palsy may result from invasion by a malignant tumor or compression from a benign mass. If there is evidence of persistent compression at any anatomic point or by a nonanatomic mass, then immediate exploration may be indicated.

Finally, radial nerve paralysis should also be classified based on the time of presentation. If a patient presents a long time after the original injury, the clinical management will significantly alter based on the situation. The reconstructive options for a symptomatic patient presenting 1 year after a high radial nerve injury are limited. It is also extremely difficult to explore open radial nerve lesions more than 10 days after the injury because of significant inflammation and scarring. The time of presentation after radial nerve paralysis is particularly important because the motor endplates must be reinnervated within 1 year if motor function is to be restored. Electrodiagnostic studies should be obtained on all patients who fail to demonstrate improvement within 3 months of injury. The level and duration of the injury will ultimately be the primary factors that determine the reconstructive options available. Therefore, it is extremely important to involve a peripheral nerve surgeon early in the management of all patients presenting with radial nerve palsy to optimize care and recovery.

**MANAGEMENT OPTIONS**

An algorithm for the treatment of radial nerve palsy was developed to determine the need for surgical reconstruction based on a concise and clinically relevant classification system (Fig. 1). The first step in determining a treatment is to classify the lesion as either open or closed. All radial nerve palsies associated with open wounds should be explored surgically. If the nerve is found to be in continuity at the time of the exploration, it is treated as a closed injury. If the radial nerve has been sharply transected, but there is adequate nerve length and minimal soft-tissue injury, then it should be repaired primarily.

A primary nerve repair should be performed without tension by mobilizing the nerve both proximally and distally. To ensure complete removal of the lesion or zone of injury, only surgeons familiar with the technique should perform acute nerve grafting in this situation. The proximal and distal extent of the transected or injured nerve can be more clearly delineated if the surgery is delayed for 3 weeks. Nevertheless, it may be quite difficult to explore a nerve safely after a delay because of the progression of the surgical scar. Before 3 weeks, the extent of nerve injury can be deter-
mined with intraoperative electrodiagnostic studies and microscopic examination.

The management of closed radial nerve palsies is far less straightforward. Closed radial nerve injuries can be associated with a wide range of underlying pathology. Surgical exploration is indicated only when transection of the radial nerve is suspected, as might be the case after a comminuted humeral fracture. If a radial nerve transection is not suspected, then the patient should be observed closely for a period of 3 months. The clinical outcome is dependent upon an accurate determination of the degree of nerve injury based on a complete patient history and physical and a well-timed electrodiagnostic study (when indicated). Fortunately, most closed radial nerve palsies are associated with either a neurapraxia or a second- or third-degree injury that usually recovers spontaneously with time. A Tinel sign can be used to follow the progressive recovery of the nerve along its anatomic course in both second- and third-degree injuries.

A physical examination of the upper extremity will help to determine a clinically obvious mass causing radial nerve compression. However, magnetic resonance imaging may be needed in some situations to rule out a less obvious soft-tissue mass, although both false-negative and false-positive magnetic resonance imaging reports may be seen in this situation. It is important to prevent joint contractures with a wrist splint and aggressive hand therapy during the 3 months of observation. Patients who do not demonstrate clinical evidence of recovery within 2 to 3 months of observation or after a negative surgical exploration should undergo electrodiagnostic evaluation. Surgical intervention is indicated if no clinical or electrical evidence of reinnervation is seen within 3 months.

Electrodiagnostic studies will help to determine the level and extent of the radial nerve injury. High, intermediate, or low radial nerve injuries should be explored, compression points should be released, and the nerve should be repaired or reconstructed when conservative management fails. The level of the nerve injury can be further delineated by an intraoperative, nerve-to-nerve study, as mentioned earlier. Because the motor endplates are not out of reach for regenerating axons, patients with low and intermediate radial nerve injuries repaired primarily or grafted at 3 to 4 months have an excellent prognosis. Conversely, the axons have a long distance to travel after a high radial nerve injury. This has resulted in the recommendation for tendon or nerve transfer after high injuries that fail to recover within 3 months. Also, patients with complete loss of radial nerve function following neuritis or whose treatment has been significantly delayed should be considered for tendon or nerve transfer (Table II).

**Five-Step Radial Nerve Release**

The general approach to the posterior interosseous nerve has been described previously in the literature. The radial nerve at the forearm is approached using a standard, five-step approach in which: 1) The interval between the extensor carpi radialis longus and the brachioradialis muscle is palpated preoperatively by having the patient extend his or her wrist

<table>
<thead>
<tr>
<th>Level of Injury</th>
<th>Type of Injury*</th>
<th>Treatment Recommendation(s)</th>
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<tbody>
<tr>
<td>Low</td>
<td>Neurapraxia</td>
<td>Observation for 3 months</td>
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<tr>
<td></td>
<td>Axonotmesis (I, II, III, VI)</td>
<td>Observation for 3 months</td>
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<tr>
<td>Intermediate</td>
<td>Axonotmesis (IV)</td>
<td>Primary repair, nerve graft</td>
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<td>Neurapraxia</td>
<td>Observation for 3 months</td>
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<td>Axonotmesis (I, II, III, VI)</td>
<td>Observation for 3 months</td>
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<td></td>
<td>Neurapraxia</td>
<td>Nerve graft</td>
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<td></td>
<td>Axonotmesis (IV)</td>
<td>Nerve graft</td>
</tr>
<tr>
<td>High</td>
<td>Neurotmesis</td>
<td>Primary repair, nerve graft</td>
</tr>
<tr>
<td></td>
<td>Neurapraxia</td>
<td>Observation for 3 months</td>
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<tr>
<td></td>
<td>Axonotmesis (I, II, III, VI)</td>
<td>Observation for 3 months</td>
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<tr>
<td></td>
<td>Axonotmesis (IV)</td>
<td>Tendon transfer, nerve transfer</td>
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</tbody>
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* I, first-degree injury or neurapraxia; II, second-degree injury with axonotmesis involving the axon and myelin but intact endoneurium; III, third-degree injury with axonotmesis involving the axon, myelin, and endoneurium but intact perineurium; IV, fourth-degree injury is a neurona in-continuity; V, fifth-degree injury is a transected nerve or neurotmesis; VI, sixth-degree injury is a combination of any of the above injuries (Mackinnon, S. and Dellon, A. L. Surgery of the Peripheral Nerve. New York: Thieme, 1988.)
against resistance; 2) a longitudinal incision is made, and the interval between the two muscles is identified by a slight color difference and the intervening posterior cutaneous nerve of the forearm; 3) the nerve is protected, and the avascular plain between the two muscles is carefully dissected; 4) the fascial edge of the extensor carpi radialis brevis is identified and released, exposing the underlying supinator; and 5) the edge of the supinator is identified and released, exposing the underlying branches of the radial nerve.

The radial sensory nerve, the nerve to the extensor carpi radialis brevis, and the posterior interosseous nerve can be fully distinguished through the above-described approach. Specific areas of compression can now be addressed, such as the vascular leash of Henry, the arcade of Frohse, the superficial head of the supinator, and the fascia around the radiocapitellar joint. The posterior interosseous nerve can be approached in several different ways, depending on the surgeon’s preference or familiarity with the procedure.

We have found the approach to the radial nerve through the brachioradialis-extensor carpi longus interval to be particularly useful in ensuring a complete release of all potential points of compression at this level.

**Neurorrhaphy and Nerve Grafting**

The radial nerve is the most suitable for neurorrhaphy of all major nerves because the fascicles are largely motor and the most common site of injury is close to motor endplates. Several factors, such as age, level of injury, length of defect, associated injuries, and interval to surgery, have been noted to influence the recovery from nerve injury. Better results in children probably stem from better central adaptation to altered profiles after nerve repair. Excellent results ranging between 78 and 90 percent have been reported after primary radial nerve repair.

The results of radial nerve repair using nerve grafts have been comparable with primary repairs. Millesi et al. reported that 77 percent of patients with interfascicular radial nerve grafting obtained four out of five motor strength. Dolenc reported 14 cases of radial nerve transections that required nerve grafts of varying lengths. He determined that the time from injury to surgery and the surgical condition were more important than the length of the nerve graft. In other studies, good results have been noted in 80 percent of patients that required radial nerve grafts. Nerve grafting is indicated if the nerve defect is large or there is significant tension on the repair.

**Tendon Transfers**

Most authors agree that tendon transfers provide good results if nerve reconstruction fails in patients with radial nerve palsy. Sunderland recommended a tendon transfer if there were no signs of radial nerve recovery within 1 year. In 1916, Jones described a tendon transfer for radial nerve palsy that included the pronator teres to the wrist radial extensors, the flexor carpi radialis to the extensors comminins, and the flexor carpi ulnaris to extensor indicis and pollicis longus. The transfer of both wrist flexors has since been abandoned by most surgeons because of the excess morbidity from the loss in wrist flexion.

Currently, there is continued disagreement on the best combination of tendon transfers to use in treating patients with radial nerve paralysis. The level of the radial nerve injury and a patient’s overall function and anatomy often dictate the best surgical option available. Most authors agree that the extensor carpi radialis brevis and longus should be reconstructed using the pronator teres tendon. The extensor digitorum communis can be reconstructed using the flexor digitorum superficialis (III), the flexor carpi ulnaris, or the flexor carpi radialis. The rerouted extensor pollicis longus can be reconstructed using the palmaris longus or the flexor digitorum superficialis (IV), and, in some cases, the abductor pollicis longus and extensor pollicis brevis can be reconstructed with the flexor carpi radialis. We prefer to use the pronator teres to the extensor carpi radialis brevis, the flexor carpi ulnaris to the extensor digitorum communis, and the palmaris longus rerouted to the extensor pollicis longus (when available); otherwise, we use the flexor digitorum superficialis.

In the 1970s, Bevin advocated early tendon transfer in radial nerve transection. He reported an average recovery time from nerve repair to be 7.5 months, with 66 percent of patients achieving good or excellent function. In the tendon transfer group, all patients noted good to excellent results in 8 weeks. The pronator teres was transferred to the extensor carpi radialis longus and brevis, the palmaris longus was transferred to the thumb extensors and long abductor (when present), and the
flexor carpi ulnaris was transferred to the common digital extensors. When the palmaris longus was not present, the thumb extensors and abductor were motored by the flexor carpi ulnaris as well. However, it was difficult to fully determine from Bevin’s article the approximate level of the radial nerve injury in the patients reviewed.

Burkhalter also advocated early tendon transfer because he believed the transfer acts both as a substitute during regrowth of the nerve or when lesions are irreparable and also as a helper during reinnervation. In a recent article, Kruft et al. reported that irreversible radial nerve paralysis should be treated with early tendon transfer. They reported 43 patients who underwent tendon transfer, with 38 patients ultimately returning to their original jobs. The authors qualified their results by stating that tendon transfers “never fully replace an intact radial nerve for the purpose of controlling the hand.”

Elton and Omer observed that patients with radial nerve paralysis treated by tendon transfer often experienced extensor tightness, which prevented simultaneous flexion of the wrist and fingers. Barton described this as a “rather unnatural movement, seldom needed in ordinary life.” Several authors have thought that the greatest functional loss after radial nerve palsy was not the loss of finger extension, but instead the loss of power grip, which cannot be easily recreated with standard tendon transfers. As such, it is important to fully examine alternative approaches to treating radial nerve palsy to decrease the long-term morbidity associated with tendon transfers that clinically often appear “unnatural.”

**Nerve Transfer**

In 1948, Lurje described the use of nerve transfers for severe brachial plexus injuries when other options were not available. In the 1960s, the popularity of nerve grafting tended to overshadow such early pioneering work in nerve transfers. Currently, nerve transfers are typically performed under limited circumstances such as brachial plexus avulsions, when no other options are available. Although, we have had excellent experience with nerve transfers using nearby, expendable motor fibers for reconstruction, we believe that nerve transfers may have limited applications for certain types of high radial nerve palsies or in cases of radial neuritis.

The median nerve has a limited number of anatomic variations in the forearm; therefore, it provides several dependable sources for nerve transfer to the distal radial nerve. The median nerve supplies several redundant nerve branches to the flexor digitorum superficialis muscle groups that can be transferred in certain situations. The nerve branch to the palmaris longus can also be sacrificed if it is not required for future tendon transfers. Intraoperative sensorimotor topographical identification can be used to identify redundant branches of the median nerve that can be transferred to the radial nerve. We have successfully transferred redundant branches of the median nerve to the posterior interosseous nerve in the forearm of several radial nerve paralysis patients, with good long-term results; however, additional experience with this technique is needed before definitive recommendations regarding its indications and use can be made. Nevertheless, continued success with nerve transfers in patients with radial nerve paralysis may provide a useful alternative to tendon transfers in patients with delayed presentation or high proximal nerve injuries or in situations of complete loss of nerve function.

**CONCLUSIONS**

The optimal treatment of patients with radial nerve paralysis requires a thorough understanding of the specific anatomy, clinical presentations, and potential causes or origins of the disorder. A systematic approach to the examination and diagnosis of the disease is vital to ensure the appropriate treatment and future recovery of these patients. A clinically relevant classification system for radial nerve paralysis should encourage clinicians to address specific diagnostic dilemmas and injury types. Our classification system attempts to define both the key clinical issues that ultimately determine the need for surgical intervention and the most appropriate surgical reconstruction. Recommendations for the management of radial nerve paralysis are based on the level and degree of injury confirmed by history and physical and by electrodiagnostic studies (when indicated). Finally, the algorithm provides a general guide for both the surgical and nonsurgical options available based on the time of presentation and the type of injury. A full integration of the above material on radial nerve paralysis should assist in the overall care and
management of patients who present with this often complex and challenging clinical problem.

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REFERENCES


Self-Assessment Examination follows on the next page.
Current Approach to Radial Nerve Paralysis
by James B. Lowe, III, M.D., Subhro K. Sen, M.D., and Susan E. Mackinnon, M.D.

1. THE POSTERIOR INTEROSSEOUS NERVE SUPPLIES ALL THE FOLLOWING MUSCLES OF THE UPPER EXTREMITY EXCEPT:
   A) Extensor carpi radialis brevis
   B) Supinator
   C) Extensor carpi ulnaris
   D) Abductor pollicis longus
   E) Brachioradialis

2. RADIAL NERVE COMPRESSION HAS BEEN REPORTED AT ALL POINTS LISTED BELOW EXCEPT:
   A) Triceps tendon
   B) Vascular leash of Henry
   C) Arcade of Frohse
   D) Tendinous margin of the extensor carpi radialis brevis
   E) Tendinous margin of the extensor carpi radialis longus

3. UPPER EXTREMITY NERVE PARALYSIS FOLLOWING Tourniquet Application Is Estimated To Occur At Approximately What Ratio?
   A) One out of 130
   B) One out of 1300
   C) One out of 13,000
   D) One out of 130,000
   E) One out of 1,300,000

4. DURING SURGICAL EXPLORATION OF THE RADIAL HEAD AT THE ELBOW, WHAT MANEUVER WOULD PROTECT THE RADIAL NERVE DURING A STANDARD EXPOSURE?
   A) Forearm supination
   B) Forearm pronation
   C) Elbow distraction
   D) Elbow extension
   E) Elbow flexion

5. COMPRESSION OF THE POSTERIOR INTEROSSEOUS NERVE BY THE ARCADE OF FROHSE IS CAUSED BY:
   A) Dislocation of the radial head
   B) Enlargement of the deep head of the supinator
   C) Thickening of the superficial head of the supinator
   D) Congenital deformity of the arcade of Frohse
   E) Malposition of the extensor radialis carpal tendon

6. NERVE TRANSFER IS MOST LIKELY INDICATED IN WHAT SITUATION?
   A) Closed injury
   B) Acute sharp nerve transection
   C) Injuries also involving the median nerve
   D) Delayed diagnosis with no identifiable healthy proximal nerve segment
   E) Loss of range of motion at the elbow

To complete the examination for CME credit, turn to page 1210 for instructions and the response form.