Although trauma is the leading cause of brachial plexus neuropathy, a number of diseases of the neuromuscular and endocrine systems produce forelimb signs. Generalized neuropathies and myopathies are characterized, in most instances, by the appearance of hindlimb and forelimb weakness, with the hindlimbs affected more severely. Exception, such as brachial plexus neuropathy resulting from immunization with phenolized rabies vaccine, is uncommon. Caudal cervical spinal cord disease may produce upper motor neuron signs in the hindlimbs and lower motor neuron signs in the brachial plexus. The signs are usually bilateral, although not necessarily symmetric.

Unilateral neuropathy of named nerves of the brachial plexus maybe caused by trauma or neoplasia and presents a challenge to the clinician to accurately diagnose, prognose, and treat. Each of the latter requires a thorough knowledge of functional anatomy, diagnostic techniques, and alternatives to and complications of therapy.

### Functional Anatomy

The brachial plexus is formed by the last three cervical and first two thoracic nerves. The contributions of C5 and T2 are variable in the dog. The potential contributors to the major named peripheral nerves are outlined below.

<table>
<thead>
<tr>
<th>Nerve</th>
<th>C6</th>
<th>C7</th>
<th>C8</th>
<th>T1</th>
<th>T2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Suprascapular nerve</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Subscapular nerve</td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Musculocutaneous nerve</td>
<td>X</td>
<td>?</td>
<td>?</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Axillary nerve</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Radial nerve</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
</tr>
<tr>
<td>Median nerve</td>
<td></td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ulnar nerve</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thoracodorsal nerve</td>
<td>X</td>
<td>X</td>
<td>X</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

The suprascapular nerve innervates the supraspinatus and infraspinatus muscles. The axillary nerve supplies the flexors of the shoulder, including the teres major, teres minor, deltoideus, and part of the subscapularis. The radial nerve is motor to all the extensors of the elbow, carpus, and digits and is therefore the primary nerve of support for the limb. The musculocutaneous nerve innervates the biceps brachii and brachialis and supplies cutaneous innervation to the medial side of the forelimb. The median and ulnar nerves are frequently considered as a unit, since they supply all of the flexor muscles of the
carpus and digits, but the areas of cutaneous sensation differ.

The cutaneous innervation was recently mapped using electrophysiologic techniques. The suprascapular, subscapular, lateralthoracic, thoracodorsal, and cranial and caudal pectoral nerves lacked cutaneous areas of innervation. The dorsal cutaneous branch of C6 had a cutaneous area, but dorsal cutaneous branches were not demonstrated for C7, C8, or T1. The cervical nerves had no lateral cutaneous branches, but thoracic nerves T2 to T4 had dorsal, ventral, and lateral cutaneous branches.

There was marked overlapping of the cutaneous areas of the brachiocephalic, axillary, musculocutaneous, radial, median, and ulnar nerves. The cutaneous zone innervated by only one of these nerves was considered smaller than that generally described in neurology texts and depicted in the clinical diagnosis section that follows.

Physical and Neurologic Examination

A thorough history and physical examination are essential. The interval between trauma and presentation may determine the likelihood of muscle atrophy, which is rare before 5 to 7 days, and the reliability of electrodiagnostic tests, which is best after 7 days. Bilateral brachial plexus neuropathy does not negate the possibility of trauma as the cause. Similarly, neurologic examination should follow or accompany physical examination. The orthopaedist who does not look for neurologic signs is likely to fail as often as the neurologist who presupposes the absence of fractures. Each endangers the animal if he is unaware of coexisting cardiomyopathy.

Although each clinician develops a technique for abbreviating neurologic examinations, major functional nerve groups should be routinely tested. Flexor and extensor thrust reflexes test for peripheral nerve and segmental integrity but may be supplemented by myotactic reflexes. The specific changes observed in brachial plexus lesions will be discussed. If segmental signs are normal, long-tract signs are elicited by extensor postural thrust offront and rear limbs, placing and hopping reactions, and tests for proprioception. Tonic neck reflexes limit the long-tract area examined to C1 and areas caudal in the spinal cord. Testing for the panniculus reflex is rarely useful in brachial plexus neuropathy in spite of the proximity of the origin of the efferent arc at T2. Cutaneous sensation testing is a valuable adjunct to localization in forelimb paralysis. (4, 8, 11, 13, 15)

In paralysis of the median nerve, the gait is normal, but cutaneous sensation is absent on the palmar surface of the medial three digits. Selective atrophy of the pronator muscles is difficult to assess clinically, as is the myotactic reflex. Atrophy of the flexors of the carpus and digits may be apparent in ulnar or ulnar and median paralysis. These flexors are dually innervated except near the ulna (ulnar nerve) and radius (median nerve). The carpus sags to a slightly overextended angle on weight bearing in ulnar paralysis, and cutaneous sensation is absent on the caudal and caudolateral sides of the antebrachium, carpus, metacarpus, palmar surface of digits IV and V, and the dorsal and lateral surfaces of digit V.

Loss of radial nerve integrity produces paralysis and atrophy of the extensors of the elbow, carpus, and digits. Myotactic tests on the triceps and long and lateral digital extensors may be useful. Cutaneous sensation will be absent on the cranial surface of the antebrachium and paw except for the fifth digit. Inability to extend the carpus and the elbow is observed in low and high radial paralysis, respectively. In low radial paralysis, the animal may learn to flip the foot forward in gaiting. After 7 to 10 days of high radial paralysis, the animal swings the limb forward and touches the toe to the ground but is usually unable to extend the carpus or the elbow sufficiently for weightbearing.

The musculocutaneous nerve innervates the flexor of the elbow and some flexors of the carpus. Palpation and myotactic reflex testing of the biceps and brachialis muscles are most useful. Loss of cutaneous sensation on the medial surface of the antebrachium is difficult to recognize because of overlaps from the radial nerve cranially and the ulnar nerve caudally.

Isolated axillary nerve deficit is rare. The primary flexor muscles of the shoulder atrophy, and cutaneous sensation is deficient on the lateral surface of the brachium.

Paralysis of the suprascapular nerve results in atrophy of the supraspinatus and infraspinatus muscles (sweeny) and no loss in cutaneous sensation.

The thoracodorsal nerve is distributed mainly to the latissimus dorsi, accompanying the thoracodorsal vessels toward their peripheral distribution.
Electrodiagnostic Testing

Involvement of the nerves of the brachial plexus is rarely complete in either summation of nerves or in damage to individual named nerves. The clinical testing described above affords adequate diagnosis in traumatic radial-brachial paralysis but limited information for prognosis. Electrodiagnostic testing increases the specificity of diagnosis and prognosis. (1,3,6)

Insertion of a needle into normal muscle results in a burst of electrical activity followed by electrical silence. When the needle and accompanying reference and ground electrodes are connected to an oscilloscope by appropriate means, a display of the insertion potential and subsequent testing and stimulated electrical activity may be observed. Simultaneous production of sounds based on the duration, frequency, and amplitude of the individual electrical displays presents the examiner with the visual and auditory display used in electromyography.

Space does not permit a thorough discussion of the equipment, technique, and criteria used in electromyography. Suffice to say that high-amplitude, short-duration spontaneous spikes occurring after cessation of insertion potentials are indicative of denervation. Positive sharp waves, downward deflection of lesser amplitude and slightly longer duration may appear in neuropathic myopathy. Variable muscle action and motor unit potentials and bizarre high-frequency wave forms are indicative of myopathies and myositis. In addition, the insertion potential may be decreased in amplitude and duration in denervated skeletal muscles that have become fibrotic and increased induration in metabolic diseases such as Cushing's syndrome.

The continuity and conduction ability of motor and sensory nerves may similarly be established by stimulating and recording the response at some point distant using standard or averaging techniques. The information gained by nerve conduction testing and by electromyography is an integral part of peripheral nerve examination at most veterinary institutions. Those wishing to further their abilities are referred to these institutions and to texts on electrodiagnostic testing. (3,8)

Practical Differential Diagnosis

The differential diagnosis of forelimb weakness or paresis requires coordination of all the methods discussed above and, less commonly, special techniques such as nerve and muscle biopsy, provocative testing with drugs, myelography, and clinical laboratory testing for endocrine disorders. (1,6)

The first step in diagnosis of forelimb paresis is to confirm by physical and neurologic examination that the weakness is restricted to the forelimbs and is not associated with paraparesis (Fig. 66-1). Quadriplegia may be acute, progressive, or episodic. Trauma, intervertebral disk herniation, or cartilaginous thromboembolism may cause acute quadriplegia with bilateral but asymmetric lower motor neuron signs in the forelimbs and upper motor signs to the rear. Neurologic testing is usually sufficient to localize the caudal cervical spinal cord lesion and suggest auxiliary tests to define the cause. Progressive quadriplegia with forelimb lower motor neuron signs may be more difficult to diagnose. Careful testing should be performed to exclude signs of cranial or multifocal central nervous system (CNS) disease, as might occur in toxoplasmosis, reticulosis, or toxicity. Electroencephalography (EEG) and cerebrospinal fluid (CSF) examination and cultures are aids to diagnosis. General or patchy distribution of muscle weakness, atrophy, or exaggerated contraction suggests muscular or neuromuscular disease. Electromyography and nerve and muscle biopsies are necessary for diagnosis. (1,6)

Similar diagnostic tests are indicated in episodic weakness. Cataplexy, the sudden and transient loss in appendicular muscle tone, may be recognized by the appearance of the attack and confirmed by electrodiagnostic testing or challenge with drugs. Myasthenia gravis may similarly be exacerbated by physostigmine but is diagnosed more definitively by decremental muscle action response to repetitive stimulation of the motor nerve. The episodic weakness of Cushing's syndrome may be masked by other clinical signs. Electromyographic examination shows that insertion potentials in the awake animal are prolonged by bizarre but myotonic potentials. Cellular and chemical analyses of blood and endocrine function determinations are indicated for diagnosis.

Neuropathy of the forelimb may be bilateral or unilateral. Progressive bilateral brachial plexus neuropathy has been reported in the dog. The condition is characterized by onset over 2 to 4 days and slow recovery. Motor nerve conduction velocity is slowed, and the muscle action potential is polyphasic. (5) Involvement of the brachial plexus by a
cervical rib has also been observed in the dog but is usually characterized by caudal cervical pain and minimal paresis. (10)

Progressive unilateral forelimb paresis is most often caused by neoplasia, although neoplasms of peripheral nerve are uncommon. (9, 14) The pattern of hypalgesia and progressive muscle atrophy is characteristic in dogs 5 to 7 years of age and occasionally in older dogs and may permit an accurate localization to the named nerves involved. Electromyography is useful in specific localization, particularly if the contributing spinal nerves are involved in the neoplasm. The prognosis is less favorable when there are signs of spinal nerve involvement, although surgical exploration, as discussed below, is generally indicated.

Forelimb Paralysis Following Trauma

Acute-onset forelimb paralysis concurrent with trauma is the most common form of forelimb paralysis in the dog and cat. (4, 8, 10, 13, 15) Injury to a single radial nerve in the form of neuropraxia, axonotmesis, or neurotmesis most often accompanies oblique or comminuted fractures of the humerus and is located at the junction of the middle and distal thirds of that bone. The animal frequently refuses to extend the carpus when the humerus is fractured regardless of whether there is radialis paralysis. Loss of cutaneous sensation on the cranial surface of the forelimb in association with humeral fracture is sufficient indication for visualization of the radial nerve during fracture repair. Severed nerves should be repaired (19) (see Chapter 65). There is little evidence that tagging and delayed surgical approach for nerve repair is more successful than primary repair concurrent with fracture fixation.

Injury to the musculocutaneous nerve distal to the midshaft of the humerus causes minimal clinical signs in the dog. Damage to the median and ulnar nerves is uncommon in spite of the proximity to the humerus. A medial approach is necessary for exploration and repair.

Unfortunately, trauma to the forelimb frequently causes damage to multiple nerves of the brachial plexus rather than a single named peripheral nerve. Theories equating the extent of damage to the abnormal position of the limb caused by trauma have merit. Those nerves with origins in the caudal cervical and cranial thoracic spinal nerves are easily injured by forcible retraction of the limb caudally and outward, stretching these spinal nerves around the first rib. Nevertheless, multiple root damage has resulted from abduction alone, and spinal nerves C5-7 have been damaged in animals. In any event, the probability that forelimb paralysis due to trauma is the result of avulsion of multiple nerve roots of the brachial plexus is great. Physical and neurologic examination must be complete. Radiographic examination of the limb or caudal cervical spine is indicated by the deficits observed and by evidence of instability or crepitation.

Exploration of the Brachial Plexus

Avulsion of spinal nerves resulting in brachial paralysis is a theoretic indication for exploration of the brachial plexus. The practical value is lessened by the fact that the neurotmesis is almost always located at or within the spinal column. Anastomosis at this point would require extraordinary equipment, time, and expertise and would still offer limited success.

Exploration of the brachial plexus is indicated in compression by extraneural neoplasia or other pathology. (14)

Following adequate preparation for surgery, double-layer stockinette is placed on the limb and affixed to four-corner drapes. The limb is retracted caudally and an incision made from the caudal aspect of the jugular furrow caudally, medial to the greater tubercle of the humerus, to the axilla. Subcutaneous tissue and sparse cutaneous muscle are dissected to expose the brachiocephalicus muscle. Incision along the medial edge of the brachiocephalicus (cleidobrachialis) muscle will reveal a tributary of the cephalic vein, which is isolated with umbilical tape. The cranial end of the superficial pectoral muscle is
severed near its insertion on the humerus. The brachial plexus is exposed by retraction of the limb laterally and blunt dissection of the soft tissues around the nerves and the axillary artery (Fig. 66-2).

Closure is achieved following exploration and treatment by suturing the pectoral muscle, the brachiocephalicus to the adjacent fascia, the subcutaneous tissue, and skin.

Other Treatments for Radial-Brachial Paralysis

The other alternatives for treatment include coaptation splintage, tendon transposition, nerve transposition, ankylosis, and amputation. There are indications for each treatment method and combinations of these methods. Unfortunately, all except coaptation splintage have been used prematurely or have been considered too late to be effective. A scheme for staging treatment is proposed. (See Fig. 66-1.)

COAPTATION SPLINTS

Determination of neuropraxia versus neurotmesis in an animal presented with acute forelimb paralysis is not possible without exploratory surgery. An thorough neurologic examination of the limb should follow appropriate emergency treatment of the patient. The pattern of motor and cutaneous sensory loss should be observed and recorded exactly. Radiographs of the limb should be made if indicated. If neither wound nor fracture indicates the potential for severance at a specific site, the limb should be splinted. A coaptation splint is indicated for the lower limb if the animal can extend the elbow, if not, a Schroeder-Thomassplint should be applied with the elbow in a normal flexed position. The purposes of splintage are to prevent trauma to the dorsum of the foot, which might result from dragging, and to prevent contracture of flexor tendons. If the leg is not dragged and the patient and owner cooperate, passive flexion and extension and massage of the limb and minimal protective bandage are recommended. The physical therapy should be done four or five times daily. Anti-inflammatory drugs may be given in the hope of reducing neuronal swelling. Injection of 2.5 mg triamcinolone in the area around epineural repair of severed nerves enhances the histologic and functional regeneration of peripheral nerves, possibly by reducing fibroblastic activity and scarring.

Clinical improvement following reversible injury to peripheral nerves is slow. Electrodiagnostic methods will reveal neuropathy by the presence of abnormal fibrillation potentials and positive sharp waves on electromyography and absence or decreased response to motor nerve stimulation 7 to 10 days (in some instances 5 days) after injury. If electrodiagnostic equipment is available, it should be used in one week when the animal is returned for splint evaluation. Definitive treatment, such as amputation, may be indicated by denervation potentials in the radial, ulnar, musculocutaneous, and median nerves. We prefer to record the results of electromyography/motor nerve conduction velocity accurately and return the animal 2 weeks later (3 weeks post trauma) for further evaluation.

After 3 weeks, the splint is removed and motor and sensory cutaneous functions observed and compared with the original findings. If electrodiagnostic equipment is available, electromyography and motor nerve conduction velocity are repeated. Clinical signs of improvement include improved extensor and flexor function of the muscles, improved myotactic reflexes, minimal muscle atrophy, and increased areas of cutaneous sensation. In 3 weeks, one might expect cutaneous sensation to extend approximately 1 cm to 1.5 cm distally. Improvement is sufficient indication to delay other forms of therapy subject to the findings at subsequent 3-week examinations.

TENDON TRANSPOSITION

Muscle and tendon transpositions have been described in humans and animals for radial paralysis. The principles of transposition are described elsewhere. (See Chapter 71.) Inability to extend the carpus, digits, and elbow and therefore to support weight results from radial paralysis. Extension of the carpus and digit will result if the flexor carpi radialis tendon is severed distally, transposed laterally, and anastomosed to the distal severed end of the tendon of the common digital extensor. A longitudinal, S-shaped incision is made from the craniomedial surface of the forelimb to the dorsal surface of the carpus. The tendons are sutured with stainless steel suture, and a coaptation splint is applied in extension for 3 weeks after surgery. The median nerve must be functional for active contraction of the
flexor Carpi radialis.

Extension of the elbow may be accomplished by transposition of the insertion of the biceps brachii (11) or the brachialis muscle (13, 15) to the olecranon if innervation to both of these flexors is intact. In each procedure, the muscle is transposed by dissection to preserve the blood and nerve supply beneath the appropriate medial or lateral portion of the triceps muscle (Fig. 66-4). Holes are drilled in the olecranon with a Kirschner wire. Stainless steel suture is passed through one hole, continued in a Bunnell pattern in the muscle-tendon end, and returned through the other hole in the olecranon. In small dogs, the wire is placed through the hole and returned lateral or medial to the olecranon. The ends of the wire are then pulled tight and tied. The outside of the muscle tendon is sutured to the deep surface of the adjacent triceps muscle with continuous or interrupted absorbable sutures. The incision is closed in a routine manner. A Schroeder-Thomas splint is applied for 3 weeks after surgery. Transposition of the brachialis muscle is preferred, even though it has minimal tendon of insertion, because of the increased ease of the lateral approach over the medial approach to the elbow needed for biceps transposition.

ARTHRODESIS
Arthrodesis of the carpus is indicated if there is sensation to the foot, the triceps is functional, or the musculocutaneous nerve is intact, permitting transposition of a flexor to the olecranon, and the flexor Carpi radialis is denervated or the flexor tendons contracted.

NERVE-MUSCLE TRANSPOSITION/TRANSPLANTATION
A branch of the musculocutaneous nerve was transposed and anastomosed to the radial nerve by end-to-end or side-to-side techniques in four dogs in whom the radial nerve was severed at the midhumerus. In each animal, return of cutaneous sensation reached approximately 5 cm distally in the next 5 weeks and reached the carpus in 4 months. Motor function returned to permit the animal to flip the foot and support weight normally. Nevertheless, two dogs mutilated the digits of the affected limb by chewing such that amputation was required.

Self-mutilation is a common occurrence in reinnervation. It occurs consistently when cutaneous sensation reaches the digits but has been observed to march distally as cutaneous sensation returns to any part of the limb, a phenomena recognized as Tinel's sign.

The results of transplantation of frozen allografts of nerves varies with the author and technique. Failure related to immune reaction, failure of neuronal recannulation, and surgical techniques decrease the rate of success. Hopefully the future will bring microsurgical techniques into common use in veterinary medicine. Transplantation of muscles with neurovascular pedicles intact should replace the use of antagonist muscles and nerves.

AMPUTATION
Amputation of the forelimb is a satisfactory salvage procedure in avulsion of the contributing nerve roots of the brachial plexus in cats and dogs.

Complications
The complications of treatment for radial-brachial paralysis are many and varied. Inadequate diagnosis of the specific site of injury is common without electrodiagnostic testing. Alignment is maintained without coaptation splintage, but atrophy is common. Erosion of the skin on the dorsolateral surface of the foot occurs frequently unless the limb is bandaged.
The two primary complications are reflexor tendon contracture with degenerative joint disease in the carpus and erosion of the foot pads. The former is nearly always the result of poor physical therapy or splinting. As has been mentioned, physical therapy with only enough bandage to protect the foot is preferred if the therapy is done well and continued. Unfortunately, the continued use of good physical therapy is rare in home care of animals. External splinting is therefore indicated more frequently.

Erosion of the foot pads on the lateral side is common in dogs with tendon transpositions. The erosion results when the ulnar nerve is damaged. The animal cannot feel the foot touch the ground and, in bearing weight, the foot slides laterally. In such cases the willingness of the owners to care for the foot by frequent application and removal of a fabricated leather shoe and the willingness of the dog to permit use of such an appliance are essential to success with tendon transposition. If the denervated pads are likely to bear weight without protection, osteomyelitis will result. Amputation is indicated in these patients.

References


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