TABLE 121-12 SYMPTOMATIC TREATMENT FOR NEUROPATHIC PAIN

TRICYCLIC ANTIDEPRESSANTS	Topiramate 150-200 mg bid (Level U)
Amitriptyline 10-150 mg qhs (Level B)	Duloxetine 60-120 mg qd (Level B)
Nortriptyline 10-150 mg qhs (Level U)	Pregabalin 150-600 mg qd (Level A)
Imipramine 10-150 mg qhs (Level U)	Sodium valproate 250-500 mg BID (Level B)
Desipramine 10-150 mg qhs (Level U)	**ALTERNATIVE TREATMENTS**
Venlafaxine 75-225 mg qd (Level B)	Tramadol 50-100 mg qid (Level B)
ANTICONVULSANTS	Lidoderm patches (Level C)
Gabapentin 300-1200 mg tid (Level B)	Capsaicin cream (Level B)
Carbamazepine 100-200 mg tid (Level U)	Transcutaneous nerve stimulation
	Acupuncture

of demyelinating and vasculitic neuropathies is extremely variable, depending on the cause.

COMMON MONONEUROPATHIES

Common mononeuropathies are explored in Table 121-7.

Carpal Tunnel Syndrome

Carpal tunnel syndrome results from compression of the median nerve at the wrist as it passes beneath the flexor retinaculum. Precipitating factors include activities that require repetitive wrist movements, such as mechanical work, gardening, house painting, and typing. Predisposing causes include pregnancy, diabetes, acromegaly, rheumatoid arthritis, chronic renal failure, thyroid disorders, and primary amyloidosis.

Symptoms usually begin in the dominant hand but commonly involve both hands over time. Patients typically report numbness, tingling, and burning sensations in the palm and in the fingers supplied by the median nerve: the thumb, index finger, middle finger, and medial one half of the ring finger. Some patients report that all fingers become numb. Pain and paresthesias are most prominent at night and often interrupt sleep. The pain is prominent at the wrist but may radiate to the forearm and occasionally to the shoulder. Shaking the hand relieves both pain and paresthesias. Percussion of the median nerve at the wrist provokes paresthesias in a median nerve distribution in 60% of patients (Tinel sign), and flexion of the wrist for 30 to 60 seconds provokes pain or paresthesias in 75% of cases (Phalen sign).

The diagnosis is based on clinical symptoms and signs. Electrodiagnostic studies may demonstrate prolongation of the sensory or motor latencies across the wrist in up to 85% of patients. In more severe cases, EMG may demonstrate evidence of denervation in the abductor pollicis brevis.

Treatment initially includes avoidance of repetitive wrist activities and the use of a neutral wrist splint. If these conservative measures fail, injections of lidocaine and methylprednisolone can be given into the carpal tunnel or surgical treatment by section of the transverse carpal ligament can effectively decompress the nerve. Indicators that have been shown to predict failure with conservative management include age greater than 50 years, disease duration greater than 10 months, constant paresthesias, and a positive Phalen sign in less than 10 seconds.

Ulnar Palsy

The ulnar nerve may become entrapped at the elbow because of external compression in the condylar groove. Injury may also occur years after a malunited supracondylar fracture of the humerus with bony overgrowth. Contrary to the findings in carpal tunnel syndrome, muscle weakness and atrophy characteristically predominate over sensory symptoms and signs. Patients notice atrophy of the first dorsal interosseous muscle and difficulty performing fine manipulations of the fingers. Numbness of the little finger, the contiguous one half of the ring finger, and the ulnar border of the hand may be present. Ulnar nerve compression can be confirmed with electrodiagnostic studies demonstrating slowed motor conduction velocity across the elbow. Treatment includes the use of elbow pads to avoid compression or surgical procedures including transposition of the ulnar nerve or decompression of the cubital tunnel.

Peroneal Neuropathy

The peroneal nerve can become compressed as it wraps around the fibular head and passes into the fibular tunnel between the peroneus longus muscle and the fibula. Compression may occur as a result of habitual leg crossing, prolonged bedrest, knee casts, prolonged squatting, anesthesia, or profound weight loss. The nerve can also be compressed as a result of Baker cysts, fibular fractures, blunt trauma, tumors, or hematomas at the knee. Symptoms include "footdrop" with selective weakness of the ankle dorsiflexors and evertors as well as the toe extensors. Reflexes remain normal, and sensory loss generally involves the anterolateral leg and dorsum of the foot. Electrodiagnostic studies demonstrate slowing of the peroneal conduction velocity across the fibular head and may demonstrate denervation if axonal injury is present. Compressive injuries usually resolve spontaneously within weeks to months. Magnetic resonance imaging (MRI) and surgical exploration should be considered if symptoms are progressive.

SPECIFIC ACQUIRED POLYNEUROPATHIES

Guillain-Barré Syndrome: Acute Inflammatory Demyelinating Polyneuropathy

Since the advent of polio vaccination, Guillian-Barré (GBS) has become the most frequent cause of acute flaccid paralysis throughout the world. GBS is an immune-mediated disorder that follows an identifiable infectious disorder in approximately 60% of patients. The best-documented antecedents include infection with *Campylobacter jejuni,* infectious mononucleosis, CMV, herpesvirus, and mycoplasma. *C. jejuni* is often associated with more severe axonal cases.

The initial symptoms of GBS often consist of tingling and pins-and-needles sensations in the feet and may be associated with dull low back pain. By the time of presentation, which occurs hours to 1 to 2 days after the first symptoms, weakness has usually developed. The weakness is usually most prominent in the legs, but the arms or cranial musculature may be involved first. Muscle stretch reflexes are lost early, even in regions where strength is retained. Cutaneous sensory deficits (loss of pain and