Upper trunk lesions may be due to trauma and idiopathic brachial plexitis (see later discussion). Lower trunk lesions may result from malignant tumor invasion, thoracic outlet syndrome, or as a complication of sternotomy. If the entire plexus is involved, radiation injury, trauma, and late metastatic disease are the most common causes.

Acute Autoimmune Brachial Neuritis

Acute autoimmune brachial neuritis is characterized by the abrupt onset of severe pain, usually over the lateral shoulder, but at times extending into the neck or entire arm. The acute pain generally subsides after a few days to a week; by this time, weakness of the proximal arm becomes apparent. The serratus anterior, deltoid, and supraspinatus are the most commonly affected muscles, but other muscles of the shoulder girdle may also be affected. In rare cases, most of the patient's arm and even the ipsilateral diaphragm are involved. Sensory loss is usually slight and generally involves the axillary nerve distribution. Weakness lasts weeks to months and be accompanied by severe atrophy of the shoulder girdle. No therapy has been shown to alter or shorten the clinical course, although steroids and analgesics may reduce pain. Most patients recover within several months to 3 years. The disorder frequently follows an upper respiratory infection or an immunization, but in many instances no antecedent illness occurs. It is bilateral in one third of cases but is always asymmetrical; it may recur in 5% of patients. Recurrent brachial plexopathies that are painless may be related to an autosomal dominant disorder, hereditary neuropathy with liability to pressure palsies (HNPP), caused by a deletion, or point mutation of PMP-22 protein (chromosome 17p).

Lumbosacral Plexopathy

The lumbosacral plexus is formed from the ventral rami of spinal nerves T12 to S4. These divide within the plexus into ventral and dorsal branches that form the femoral, sciatic, and obturator nerves. The plexus is located within the substance of the psoas major muscle. Clinical features include proximal pain and weakness in anterior thigh muscles (femoral) or posterior thigh muscles and the buttocks. Bowel and bladder dysfunction may also occur. Diabetes, malignant invasion, radiation therapy, infection (herpes zoster), psoas abscess, trauma, and retroperitoneal hemorrhage are common causes. An autoimmune form is much less frequent than brachial neuritis.

DISORDERS OF THE PERIPHERAL NERVES

Definition and Epidemiology

Peripheral neuropathy refers to a large group of disorders that can produce focal (mononeuropathy or multiple mononeuropathies) or generalized nerve dysfunction (polyneuropathies) (Table 121-6). Peripheral neuropathies are prevalent neurologic conditions, affecting 2% to 8% of adults, with the incidence increasing with age. They range in severity from mild sensory abnormalities, found in up to 70% of patients with long-standing diabetes, to fulminant, life-threatening paralytic disorders such as Guillain-Barré syndrome (GBS).

Mononeuropathies are disorders in which only a single peripheral nerve is affected. The most common cause is nerve entrapment such as median nerve compression resulting in carpal tunnel syndrome or peroneal nerve injury causing footdrop (Table 121-7). When more than one peripheral nerve is involved, the term *mononeuropathy multiplex* or *multiple mononeuropathies* is often used. Multiple mononeuropathies are most commonly seen in diabetes mellitus and vasculitis but also occur in leprosy, vasculitis, sarcoidosis, hereditary neuropathy with predisposition to pressure palsies, and amyloidosis.

TABLE 121-6 CLASSIFICATION AND CAUSES OF PERIPHERAL NEUROPATHY

TYPE OF NEUROPATHY	EXAMPLES
MONONEUROPATHIES	
Compressive	Carpal tunnel syndrome, ulnar palsy
Hereditary	Hereditary neuropathy with predisposition to pressure palsies
Inflammatory	Bell's palsy
Multiple mononeuropathies	Vasculitis (mononeuritis multiplex), diabetes, leprosy, sarcoidosis, amyloidosis
POLYNEUROPATHIES	
Hereditary	Charcot-Marie-Tooth disease
Endocrine	Diabetes, hypothyroidism
Metabolic	Uremia, liver failure
Infections	Leprosy, diphtheria, human immunodeficiency virus, Lyme disease
Immune mediated	Guillain-Barré syndrome, chronic inflammatory demyelinating polyneuropathy
Toxic	Lead, arsenic, alcohol, drug induced
Paraneoplastic	Lung cancer

Polyneuropathies are a group of disorders affecting the motor, sensory, and autonomic nerves. These disorders may predominantly affect the nerve axon (axonal neuropathies), myelin sheath (demyelinating neuropathies), or the small- to medium-sized blood vessels supplying the nerves (vasculitic neuropathies). The clinical features of the polyneuropathies reflect the pathology of the underlying process.

Pathology

In the symmetrical *axonal* polyneuropathies, the underlying pathology is usually a slowly evolving type of axonal degeneration that involves the ends of long nerve fibers first and preferentially. With time, the degenerative process involves more proximal regions of long fibers, and shorter fibers are affected. This pattern of distal axonal degeneration or *dying back* of nerve fibers results from a wide variety of metabolic, toxic, and endocrinologic causes.

In the *demyelinating* polyneuropathies, the underlying pathology involves the myelin sheath.

Demyelination of a peripheral nerve at even a single site can block conduction, resulting in a functional deficit identical to that seen after axonal degeneration. In contrast to repair by regeneration, however, repair by remyelination can be rapid. Autoimmune attack on the myelin sheath occurs in the inflammatory demyelinating neuropathies (GBS and chronic inflammatory demyelinating polyneuropathy [CIDP]) and some neuropathies associated with paraproteinemias (see later discussion). Inherited disorders of myelin such as Charcot-Marie-Tooth (CMT) disease comprise the other major category of demyelinating neuropathies. Other causes include toxic, mechanical, and