CHAPTER 68
PERIPHERAL AND CRANIAL NERVE LESIONS

Dale J. Lange

Werner Trojaborg

Lewis P. Rowland

INJURY TO CRANIAL AND PERIPHERAL NERVES

The peripheral and cranial nerves are subject to trauma, infections, tumors, toxic agents, and vascular or metabolic disorders. Trauma is the most common cause of localized injury to a single nerve (mononeuropathy). Toxic and metabolic disorders usually affect many nerves (mononeuropathy multiplex or symmetric polyneuropathy).

Pathology

After nerve damage, the pathologic changes depend on the nature of the injury, which also affects the regenerative response and the prognosis for recovery. According to Seddon (1954), mechanical nerve injuries are classified as follows: (1) complete severing of a nerve (*neurotmesis*), (2) axonal interruption with distal degeneration but an intact endoneurium (*axonotmesis*), or (3) conduction block at the site of the lesion but normal distal conduction without degeneration of distal fibers (*neurapraxia*).

Within the first 24 hours of injury, focal swelling occurs adjacent to the damaged site with fragmentation of endoplasmic reticulum, neurotubules, and neurofilaments, and accumulation of organelles. The axolemma becomes discontinuous; axons swell at some sites and narrow at others to give a beaded appearance. This process begins between the nodes of Ranvier and appears first in smaller fibers. Changes in myelin sheaths lag behind those in axons but progress in a similar way along the entire distal stump, again affecting small fibers first. The myelin surrounding the fragmented axons breaks up to form rows of elliptoids. Finally, Schwann cells and macrophages degrade the axon and myelin debris. In addition to these distal nerve changes, a *retrograde axon reaction* or *chromatolysis* is seen, with retraction of axons proximal to the lesion and alterations in the somata of neurons, such as cell body swelling, disruption of Nissl substance, migration of the cell nucleus, and increase in the size of the nucleolus. Presynaptic terminals gradually withdraw from the soma and dendrites; synaptic transmission is reduced until dorsal root stimulation fails to excite the motor neuron and evoke a reflex discharge in the ventral root. The pathologic distal changes of degeneration and retrograde axon reaction are similar in crush injury or complete nerve transection.

If a nerve has been completely severed, the orderly process just described is interfered with in proportion to the length of the discontinuity between proximal and distal ends. If this distance is great, regeneration is not possible, unless the ends are apposed at operation. If the distance is small, the fine processes of the axon penetrate the fibrin and connective tissue in the scar and enter the distal end of the nerve. Some of these may be deflected

from the proper path by the scar and become entangled to form a *neuroma*.

Clinical Manifestations

The symptoms and signs of nerve injury depend on the type of nerve affected. If the nerve is mainly motor, the result is flaccid paralysis with wasting of the muscles innervated by the nerve. If the nerve contains sensory fibers, the result is loss of sensation in an area that is usually smaller than the anatomic distribution of the nerve. Vasomotor disorders and “trophic disturbances” are more common when a sensory or mixed type of nerve is injured than when a motor nerve is damaged. Partial injury or incomplete division of a nerve may be accompanied by pain that may be stabbing in character, by dysesthesia in the form of a pins-and-needles sensation, or, rarely, by severe burning pain (*causalgia*). Complete or incomplete interruption of a nerve may be followed by changes in the skin, mucous membranes, bones, and nails (trophic changes).

Diagnosis

The diagnosis of injury to one or more peripheral nerves can usually be made clinically by the distribution of the motor and sensory abnormalities. These patterns are considered later in connection with the description of isolated peripheral nerve lesions. The differentiation between lesions of the spinal roots and one or more peripheral nerves can be made by determining whether the muscular weakness and sensory loss are segmental rather than in the pattern of a nerve distribution. Electromyography (EMG) can be used to study the patterns of denervation and later reinnervation; nerve conduction studies can ascertain the site and the nature of the injury.

The differential diagnosis between polyneuropathy and other causes of generalized weakness is reviewed in Chapter 105.

Prognosis

The prognosis after injury of peripheral nerves is related to the degree of axonal injury and, to some extent, to the site of the injury. As a rule, the nearer the injury is to the central nervous system (CNS), the lower the probability will be that a completely severed nerve will regenerate, particularly cranial nerves, which are part of the CNS.

When injury to a peripheral nerve involves no loss of axons (i.e., conduction block) or little axonal loss, recovery is complete within a few days or weeks. If axonal loss is severe, recovery is slow, because axonal regeneration is required for recovery of function. If the nerve is severed or the damage is so great that axons regrow along appropriate tubules, recovery may not be complete or may fail to occur at all. In this circumstance, the neuronal dysfunction is permanent.

P.437

Treatment

When a peripheral nerve is severed by trauma, the ends should be surgically anastomosed. There is no agreement about the best time to explore and repair lesions of peripheral

nerves if it cannot be determined whether there has been anatomic or physiologic interruption of the nerve. Most clinicians believe that surgery should be performed as soon as possible if there is any doubt about the state of the nerve.

After surgical therapy, or in patients who do not need operative therapy, rehabilitation measures should commence immediately with passive range-of-motion exercises for paralyzed muscles and reeducative exercises for weak muscles. Electrical stimulation is of unproven value in preventing permanent weakness. Splints, braces, and other corrective devices should be used when the lesion produces a deformity, but should be removable for the regular application of physiotherapy.

CRANIAL NEUROPATHIES

Olfactory Nerve and Tract

The ability to smell is a special quality relegated to the olfactory cells in the nasal mucosa. The molecular biology of smell is uncertain, but transcription-activating factors, such as Olf-1, found exclusively in neurons with olfactory receptors, probably direct cellular differentiation. Smell may be impaired after injury of the nasal mucosa, the olfactory bulb or its filaments, or CNS connections. Lesions of the nerve cause diminution or loss of the sense of smell. Injury to the CNS connections usually is not accompanied by any detectable loss of olfactory sense. Occasionally, olfactory hallucinations of a transient and paroxysmal nature may occur with lesions in the temporal lobe. Loss of the sense of smell is often accompanied by impaired taste, depending on the volatile substances in the food and beverages.

The sense of smell may be temporarily impaired in connection with the common cold. Inflammatory or neuritic lesions of the bulb or tract are uncommon, but these structures are sometimes affected in meningitis or in multiple peripheral neuritis. Patients with diabetes mellitus may have impaired sensation of smell. Hyposmia or anosmia is common early in Refsum disease. The olfactory bulb or tract may be compressed by meningiomas, metastatic tumors, or aneurysms in the anterior fossa or by infiltrating tumors of the frontal lobe. The filaments of the olfactory nerve may be torn from the cribriform plate, or the olfactory bulb may be contused or lacerated in head injuries. Leigh and Zee (1991) reported altered olfactory sense in 7.2% of 1,000 patients with head injuries observed at a military hospital. The loss was complete in 4.1% and partial in 3.1%. Recovery of smell occurred in only 6 of 72 patients. *Parosmia* (perversion of sense of smell) was present in 12 patients. In a study of head injuries in civilians, Friedman and Merritt (1944) found that the olfactory nerve was damaged in 11 (2.6%) of 430 patients. In all patients, the anosmia was bilateral. In three, the loss was transient and disappeared within 2 weeks of injury.

Parosmia is not accompanied by impairment of olfactory acuity and is most commonly caused by lesions of the temporal lobe, although it has been reported when the injury was probably in the olfactory bulb or tract. Hallucinations of smell may occur in psychotic persons or may be an aura in patients with convulsive seizures (hippocampal or uncinate gyrus fits). The aura in such cases is usually an unpleasant odor that is described with difficulty.

Increased sensitivity to olfactory stimuli is rare, but cases have been reported in which the sense of smell is so acute that it is a source of discomfort. Such a symptom is usually psychogenic.

Optic Nerve and Tract

The retina, optic nerve, and optic tract are subject to injury from many causes with resulting loss of vision, impairment of pupillary light reflexes, and abnormalities in pupil size (Table 68.1).

TABLE 68.1. EFFECTS OF LESIONS OF THE OPTIC, OCULOMOTOR, AND SYMPATHETIC PATHWAYS ON THE PUPILS

Changes in the retina or optic nerve may result from direct trauma, damage by toxins, systemic diseases (e.g., chronic renal failure, diabetes mellitus, leukemia, anemia, polycythemia, nutritional deficiencies, syphilis, tuberculosis, the lipodystrophies,

P.438

giant cell arteritis, or generalized arteriosclerosis), demyelinating hereditary diseases, local conditions (e.g., chorioretinitis, glaucoma, tumors, congenital anomalies, or thrombosis or embolism of the veins or arteries of the retina), infiltration or compression of the nerve (e.g., by glioma, meningioma, pituitary tumor, craniopharyngioma, metastatic tumor, or aneurysm), or increased intracranial pressure. Most of these conditions are considered elsewhere in this volume. Disorders of vision are also discussed in Chapter 7.

Optic neuritis is a term used loosely to describe lesions of the optic nerve accompanied by diminution in visual acuity with or without changes in the peripheral fields of vision and caused by inflammatory, degenerative, demyelinating, or toxic disorders (Fig. 68.1). On ophthalmoscopic examination, the disc may appear normal at first, or swelling and congestion of the nerve may be apparent. Later, the disc is pale and smaller than normal.

FIG. 68.1. Chart of visual fields in a patient with retrobulbar neuritis indicates large central scotoma in left eye. Visual acuity: OD 15/15, OS 1/400. (Courtesy of Dr. M. Chamlin.)

The optic nerve or retina may be injured by many toxic substances, including methyl alcohol, ethyl alcohol, tobacco, quinine, pentavalent arsenicals, thallium, lead, or mercury.

Alcohol-Tobacco Amblyopia

This term is used to describe the optic neuritis that is attributed to long and continued use of both tobacco and ethyl alcohol. The lesion could be an interstitial neuritis with destruction of the papillomacular bundle. A more reasonable hypothesis, however, is that the ganglion cells in the macular region of the retina are damaged. The neuritis is most common in middle-aged men who smoke a pipe and drink alcohol in large quantities. It usually affects both eyes. At the onset, a central or paracentral scotoma for colors exists

that progresses to a complete central scotoma. The peripheral fields of vision are normal. Alcohol-tobacco amblyopia has been noted in association with pernicious anemia; malabsorption of vitamin B_{12} may be a factor in causing alcohol-tobacco amblyopia. Many authorities believe that the condition is primarily a nutritional disorder in alcoholic persons who are not eating properly. Absolute withdrawal of all forms of alcohol and tobacco may improve vision, unless the disease has progressed to the point of complete atrophy of the retinal cells of the optic nerve.

Oculomotor, Trochlear, and Abducens Nerves

Injury to the nerves or nuclei that innervate the ocular muscles causes diplopia, deviation of the eyeball, and impairment of ocular movements (Table 68.2).

TABLE 68.2. CAUSES OF THIRD AND SIXTH CRANIAL NERVE PALSIES

Complete lesions of the third nerve or its nucleus produce paralysis of the extrinsic muscles of the eye supplied by this nerve (medial rectus, superior rectus, inferior rectus, inferior oblique, and levator palpebrae superior), as well as the constrictor of the ciliary muscles. There is ptosis of the lid with loss of the ability to open the eye; the eyeball is deviated outward and slightly downward; the pupil is dilated, does not react to light, and loses the power of accommodation. Partial lesions of the third nerve or its nucleus produce fragments of the above picture according to the extent of involvement of the nerve fibers or neurons.

Lesions of the fourth nerve or nucleus cause paralysis of the superior oblique muscle with impairment of the ability to turn the eye downward and inward. Deviation of the eyeball is slight, and diplopia is prevented by inclination of the head forward and to the side of the normal eye.

Injury to the sixth nerve causes paralysis of the lateral rectus muscle. The eyeball is deviated inward, and diplopia is present in almost all ranges of movement of the eye, except on gaze to the side opposite the lesion. Lesions in the brainstem that involve the sixth nerve nucleus are accompanied by a paralysis of lateral gaze. On attempts to look toward the affected side, neither eyeball moves beyond the midline. An intact third nerve on the opposite side can be demonstrated by the ability of the patient to move the internal rectus muscle of that eye in accommodation–convergence movements.

Paralysis of the ocular muscles may result from injury to the corresponding motor nerves or cells of origin by many conditions, including trauma, neurosyphilis, multiple sclerosis (MS) and other demyelinating diseases, tumors or aneurysms at the base of the skull, acute or subacute meningitis, thrombosis of intracranial venous sinuses, encephalitis, acute anterior poliomyelitis, diphtheria, diabetes mellitus, syringobulbia, vascular accidents in the brainstem, lead poisoning, botulism, alcoholic polioencephalitis (Wernicke encephalitis), osteomyelitis of the skull, and following spinal anesthesia or simple

lumbar puncture. Intraorbital lesions may cause ophthalmoplegia, proptosis, and local pain;

retroorbital lesions may cause similar symptoms.

Intracavernous inflammation is held to be responsible for a form of painful ophthalmoplegia known as the *Tolosa-Hunt syndrome*, but the pathology has been documented in few cases; most would also be considered examples of *orbital myositis* or *orbital pseudotumor*, in which swelling of the muscles within the orbit can be demonstrated by computed tomography (CT). Ocular palsies are frequently seen in myasthenia gravis, ocular myopathy, and, rarely, polyneuropathy. Discussion in this section is restricted to the disturbance of eye movements in patients with increased intracranial pressure.

Paralysis of Eye Muscles Associated with Increased Intracranial Pressure

The sixth nerve has a long course from its point of emergence from the brainstem to the lateral rectus muscle in the orbit. Although it lies in a fluid-cushioned channel for a portion of this course, it is peculiarly subject to injury by compression against the floor of the skull when intracranial pressure is increased from any cause. Thus, unilateral or bilateral paralysis of the lateral rectus muscle may develop in patients with increased intracranial pressure. In these patients, the paralysis is of no value in localizing the site of the lesion (see Table 68.2).

Rarely, the third nerve is injured by increased intracranial pressure. The nerve may be damaged when the increase in pressure develops slowly, as with tumors of the brain, but it is more likely to be injured when the increased pressure is of sudden onset, with herniation of the uncinate gyrus through the tentorial notch and compression of the nerve. It is most commonly seen in patients with massive intracerebral hemorrhage or with extradural or subdural hematomas. Patients are usually comatose, and thus it is impossible to test eye movements, except by doll's eye or caloric tests, which may not suffice to show paresis of muscles innervated by the third cranial nerve. However, compression of the third nerve may be manifest by a dilated pupil nonresponsive to light ipsilateral to the herniation.

Fifth (Trigeminal) Nerve

Injury to the fifth cranial nerve causes paralysis of the muscles of mastication with deviation of the jaw toward the side of the lesion; loss of ability to appreciate soft tactile, thermal, or painful sensations in the face; and loss of the corneal and sneezing (*sternutatory*) reflexes.

The dorsal root ganglion for sensory fibers in the trigeminal nerve is the trigeminal (gasserian) ganglion in the middle cranial fossa. Sensory fibers pass into the brainstem at the midpons level and either ascend to terminate in the main sensory nucleus (subserving light touch) or descend to terminate in the nucleus of the spinal tract (pain and temperature sensation) or in the mesencephalic nucleus (subserving proprioception for jaw muscles). Lesions in the pons usually involve the motor and main sensory nuclei, causing paralysis of the muscles of mastication and loss of sensation of light touch in the face. Lesions in the medulla affect only the descending tract and cause loss of the sensation of light touch in the face.

The fifth nerve may be injured by trauma, neoplasms, aneurysm, or meningeal infections.

Occasionally, it may be involved in poliomyelitis and generalized polyneuropathy. The sensory and motor nuclei in the pons and medulla may be destroyed by intramedullary tumors or vascular lesions. In addition, an isolated lesion of the descending tract may occur in syringobulbia or MS.

If no accompanying neurologic signs facilitate localization, isolated facial numbness (*idiopathic trigeminal neuropathy*) may be a difficult problem to solve. Common causes of facial numbness are dental trauma, herpes zoster, cranial trauma, head and neck tumors, intracranial tumors, and idiopathic trigeminal neuropathy. Systemic sclerosis, mixed connective tissue diseases, amyloidosis, MS, and sarcoidosis are less common causes of facial numbness. Although restricted loss of sensation over the chin (*numb-chin syndrome*) may result from dental trauma or even poorly fitting dentures, it may be the only manifestation of systemic malignancy, such as lymphoma, metastatic breast carcinoma, melanoma, or prostatic cancer. Magnetic resonance imaging (MRI) or CT of the mandible often identifies the disorder.

Painful facial numbness may herald the presence of nasopharyngeal carcinoma or metastatic carcinoma. Severe facial pain in the absence of numbness or other objective findings (tic douloureux) is caused by fifth nerve dysfunction.

Trigeminal Neuralgia (Tic Douloureux)

This disorder of the sensory division of the trigeminal nerve is characterized by recurrent paroxysms of sharp, stabbing pains in the distribution of one or more branches of the nerve. The cause is unknown. In most cases, no organic disease of the fifth nerve or CNS can be identified. Degenerative or fibrotic changes in the gasserian ganglion have been found but are too variable to be considered causal. Some investigators believe that most patients with idiopathic trigeminal neuralgia have anomalous blood vessels that compress the nerve.

Pain typical of trigeminal neuralgia occasionally affects patients with lesions in the brainstem as a result of MS or with vascular

P.440

lesions that involve the descending root of the fifth nerve. Usually, trigeminal neuralgia follows other symptoms of MS. Of all patients with MS, however, 10% have facial pain first, and other symptoms of MS may not appear for 6 years.

The attacks of facial pain in trigeminal neuralgia are attributed to discharges in the descending nucleus of the nerve, presumably because of excessive inflow of impulses to the nucleus. In support of this hypothesis is evidence that typical attacks of trigeminal neuralgia may be relieved by section of the greater auricular or occipital nerves or that an episode of trigeminal neuralgia can be interrupted by intravenous injection of phenytoin sodium (Dilantin).

Trigeminal neuralgia is the most common of all neuralgias. Onset is usually in middle or late life but may occur at any age. Typical trigeminal neuralgia occasionally affects children but rarely occurs before age 35. The incidence is slightly greater in women than in men.

The pain occurs in paroxysms. Between attacks, the patient is free of symptoms, except for

fear of an impending attack. The pain is searing or burning, coming in lightning like jabs. A paroxysm may last 15 minutes or more. The frequency of attacks varies from many times a day to a few times a month. The patient ceases to talk when the pain strikes and may rub or pinch the face; movements of the face and jaw may accompany the pain. Sometimes, ipsilateral lacrimation is prominent. No objective loss of cutaneous sensation is found during or after the paroxysms, but the patient may complain of facial hyperesthesia.

A characteristic feature is the *trigger zone*, stimulation of which sets off a typical paroxysm of pain. This zone is a small area on the cheek, lip, or nose that may be stimulated by facial movement, chewing, or touch. The patient may avoid making facial expressions during conversation, may go without eating for days, or may avoid the slightest breeze to prevent an attack. The pain is limited strictly to one or more branches of the fifth nerve and does not spread beyond the distribution of that nerve. The second division is involved more frequently than the third. The first division is primarily affected in less than 5% of patients. Pain may spread to one or both of the other divisions. In cases of long duration, all three divisions are affected in 15%. The pain is occasionally bilateral (5%) but rarely occurs at the same time. Bilateral trigeminal neuralgia is encountered most often in patients with MS.

The physical findings in patients with trigeminal neuralgia are normal. Hemifacial spasm, however, may accompany trigeminal neuralgia. The patient may be undernourished or emaciated if attacks are provoked by eating. There is no objective sensory loss, and motor functions are normal. The results of laboratory examinations are normal.

The diagnosis of trigeminal neuralgia is usually made from the history without difficulty. Also characteristic is the method patients use to demonstrate the site of origin and mode of spread of the pain. They do not touch the area but hold the tip of the index finger a short distance from the face to point to areas of origin and spread.

Trigeminal neuralgia must be differentiated from other types of pain that occur in the face or head, particularly from infections of the teeth and nasal sinus. The pains of dental and nasal sinus disease differ from those of trigeminal neuralgia in that they are usually steady and throbbing and persist for many hours. Nevertheless, many patients with trigeminal neuralgia have had numerous operations on the sinuses, and most of their teeth have been removed before the diagnosis is established. Conversely, many patients with diseased teeth are referred to neurologists with the diagnosis of trigeminal neuralgia. In these patients, the role of the diseased tooth in the production of pain can be demonstrated by syringing it and the surrounding gum with ice water. Patients with temporomandibular joint disease may have symptoms similar to those of trigeminal neuralgia, but the pain is not paroxysmal, is exacerbated by eating, and has no trigger point. Cluster headaches may be confused with trigeminal neuralgia, especially when the ipsilateral eye is red and watery. The pain of cluster headaches, however, is not paroxysmal, does not conform to the trigeminal distribution, and is accompanied by nasal stuffiness with Horner syndrome on the affected side.

Atypical facial pain occurs in the territory of the trigeminal nerve, but the characteristics are different from those of trigeminal neuralgia. The pain may be as excruciating, but the individual paroxysm always last longer than a few seconds—usually minutes or even continuously. The pain itself is dull, aching, crushing, or burning. Surgical treatment is not

effective in atypical facial pain, which may sometimes be a manifestation of depression.

Treatment of trigeminal neuralgia with carbamazepine (Tegretol) is often successful. The effective dose is usually 200 mg four times daily. Larger doses may be needed, with monitoring of serum levels and clinical signs of toxicity. Toxicity is manifested as drowsiness, dizziness, unsteady gait, and nausea. A rare but serious complication is aplastic anemia. Unfortunately, tolerance to this medication frequently develops. Baclofen (Lioresal), 50 to 60 mg daily, also relieves symptoms. Phenytoin in a dose of 300 to 400 mg per day may help some patients unresponsive to carbamazepine or baclofen, but it is more commonly used as an adjunct to these medications.

Surgical procedures to control pain are used in common practice. Techniques include microvascular decompression, radiofrequency and chemical gangliolysis using stereotactic techniques, and rhizotomy. One of the more popular procedures is radiofrequency surgery, which selectively interferes with pain-conducting small fibers but spares the large-diameter motor fibers. Some studies report 90% to 97% partial or complete relief. The rate of recurrence is uncertain.

Increasing evidence indicates that many patients with trigeminal neuralgia have compression of the trigeminal nerve by arterial loops. Posterior fossa exploration is therefore recommended for patients whose symptoms are difficult to control. Other chronic masses, such as arteriovenous malformation, aneurysm, and cholesteatoma, may be found.

Seventh (Facial) Nerve

As the facial nerve leaves the brainstem, it has two divisions: the motor root and the nervus intermedius. The functions of the intermedius are much like those of the glossopharyngeal nerve. It conducts taste sensation from the anterior two-thirds of the tongue and supplies autonomic fibers to the submaxillary and sphenopalatine ganglia that innervate the salivary and lacrimal

P.441

glands. Whether the seventh nerve has any somatic sensory function is debatable. It is thought to carry proprioceptive impulses from the facial muscles and cutaneous sensation from a small strip of skin on the posteromedial surface of the pinna and around the external auditory canal. In fact, however, sensory loss is only rarely detected in patients with lesions of the seventh nerve. Similarly, hearing is seldom impaired, although the ear may become more sensitive to low tones when the stapedius is paralyzed.

Injuries to the facial nerve cause paralysis of the facial muscles with or without loss of taste on the anterior two-thirds of the tongue or altered secretion of the lacrimal and salivary glands, depending on the portion of the nerve involved. Lesions near the origin or in the region of the geniculate ganglion are accompanied by a paralysis of the motor, gustatory, and autonomic functions. Lesions between the geniculate ganglion and the origin of the chorda tympani produce the same dysfunction as that resulting from injury in the region of the geniculate ganglion, except that lacrimal secretion is not affected. Lesions near the stylomastoid foramen result only in facial paralysis.

Lesions of the facial nucleus in the brainstem cause paralysis of all facial muscles. Lesions of the motor cortex or the connections between the cortex and the facial nucleus are

accompanied by partial paralysis, usually most severe in muscles of the lower half of the face (supranuclear palsy). Asymmetric facial movements may follow voluntary or emotional stimuli.

Because they are superficial, the peripheral branches of the seventh nerve are subject to injury by stab and gunshot wounds, cuts, and birth trauma. The nerve is occasionally injured in operations on the mastoid and parotid gland and in acoustic neuromas or trigeminal neuralgia. Damage to the seventh nerve is often found with fracture of the temporal bone and is usually evident immediately after injury. Occasionally, however, facial paralysis is delayed for several days after the accident. The mechanism of this delayed paralysis is not clear. Improvement is the rule when the nerve damage is associated with head trauma, but recovery may not be complete.

Within the skull, the nerve may be affected by tumors, aneurysms, meningeal infections, leukemia, osteomyelitis, herpes zoster, Paget disease, and sarcomas or other tumors of bone. Occasionally, it is affected in the course of generalized polyneuritis, which is common in leprosy, Guillain-Barré syndrome, or diphtheritic polyneuropathy, but seldom in diabetic or alcoholic neuropathy. The peripheral portion of the nerve may be compressed by tumors of the parotid gland. Facial palsy is rare in mumps but is common in sarcoidosis. Bilateral facial palsy may be caused by many of the conditions that produce unilateral paralysis, but is most often seen in sarcoidosis, Guillain-Barré syndrome, leprosy, leukemia, and meningococcal meningitis. The facial nucleus may be damaged by tumors, inflammatory lesions, vascular lesions, acute poliomyelitis, and MS.

Bell Palsy

Paralysis of the seventh nerve may occur without any known cause. Bell palsy often follows exposure to cold (e.g., riding in an open car) and is thought to be caused by swelling of the nerve within the facial (fallopian) canal. It occurs at all ages but is slightly more common in the third to fifth decades. The frequency of involvement on the two sides is approximately equal. Paralysis occasionally recurs either on the same or on the opposite side. Familial occurrence of Bell palsy is occasionally seen.

The onset of facial paralysis may be accompanied by a feeling of stiffness of the muscles. Pain is rare, however, except in the *Ramsay Hunt syndrome*, which is caused by herpes zoster and includes pain in the ear ipsilateral to the facial paralysis.

The signs of complete paralysis of the seventh nerve can be divided into motor, secretory, and sensory. When the damage is severe, facial paralysis is obvious, even when the face is at rest. Muscles of the lower half of the face sag. The normal folds and lines around the lips, nose, and forehead are ironed out, and the palpebral fissure is wider than normal. Absence of voluntary and associated movements of the facial and platysmal muscles is complete. When the patient attempts to smile, the lower facial muscles are pulled to the opposite side. This distortion of the facial muscles may give the false appearance of deviation of the protruded tongue or the open jaw. Saliva and food are likely to collect on the paralyzed side. The patient cannot close the eye, and with attempts to do so, the eyeball can be seen to divert upward and slightly inward (the Bell phenomenon). When the lesion is peripheral to the ganglion, the lacrimal fibers are spared and the collection of

tears in the conjunctival sac is excessive because the tears are not expressed into the lacrimal duct by lid movements. The corneal reflex is absent as a result of paralysis of the upper lid; preservation of corneal sensation and the afferent portion of the reflex is manifested by blinking of the other lid. Secretion of tears is diminished only if the lesion is proximal to the geniculate ganglion. Decrease in salivary secretion and loss of taste in the anterior two-thirds of the tongue are found when the chorda tympani is affected.

Although the seventh nerve presumably transmits proprioceptive sense from the facial muscles and cutaneous sensation from a small area of the pinna and the external auditory canal, loss of these sensations is rarely detected.

Partial injury to the facial nerve causes weakness of the upper and lower halves of the face. Occasionally, however, the lower half is more severely affected than the upper half; rarely, the opposite is seen. Recovery from facial paralysis depends on the severity of the lesion. If the nerve is anatomically sectioned, the chances of complete or even partial recovery are remote. In most patients, especially those with Bell palsy, partial or complete recovery occurs. With complete recovery, no apparent difference can be detected between the two sides of the face at rest or in motion. When recovery is partial, “contractures” may develop on the paralyzed side; superficial inspection seems to reveal weakness of muscles on the normal side. The inaccuracy of this impression becomes obvious as soon as the patient smiles or attempts to move the facial muscles.

Abnormal movement of facial muscles and lacrimation may follow facial palsy. A slight twitch of the labial muscles may occur whenever the patient blinks (*synkinesis*), or an excess secretion of tears may result when the salivary glands are activated during eating. Paroxysmal clonic contractions of all facial muscles may simulate focal jacksonian seizures. These spasms are occasionally seen in patients who have never had any obvious lesion of the facial nerve. The cause of these sequelae is not known.

P.442

They are attributed by some researchers to misdirection of the regenerated fibers or to the spread of impulses between fibers within the nerve (*ephaptic conduction*).

The differential diagnosis between facial paralysis caused by a cortical lesion and that resulting from a lesion of the nucleus or nerve can be made without difficulty, except when weakness is barely evident. Other signs of supranuclear cortical involvement include sparing of the muscles of the forehead and upper lid and preservation of electrical reactions. In addition, the weakness of a peripheral lesion is equal for all movements, whereas in supranuclear lesions volitional contractions may be greater or less than those in the emotional responses of smiling or laughing.

The differentiation between lesions of the nucleus and those of the nerve is made by associated findings. Lesions in the tegmentum of the brainstem are accompanied by paralysis of lateral gaze because of concomitant injury to the sixth nerve nucleus and parapontine gaze center. Lesions in the basal part of the brainstem are accompanied by corticospinal signs. Lesions of the nerve as it emerges from the brainstem may be caused by tumors, meningitis, or other infections, resulting in concomitant paralysis of the facial nerve with abnormalities of the eighth, sixth, and, possibly, the fifth nerves.

Attempts should be made to remove the lesion that causes the facial paralysis. Without

formal therapeutic trials, some clinicians recommend massage or electrical stimulation of the paralyzed muscles to preserve tone. Surgical procedures may help when spontaneous recovery does not occur. Neurolysis or end-to-end suture may be indicated in extracranial lesions of the nerve or its branches. When the nerve is damaged proximal to the stylomastoid foramen, end-to-end suture is not possible, and innervation of the facial muscles can be restored only by suturing the distal portion of the seventh nerve to the central portion of the eleventh or the twelfth nerves. If the eleventh nerve is used, paralysis of the sternocleidomastoid and upper fibers of the trapezius is permanent. The resultant deformity is slight, but the facial muscles contract whenever the patient attempts to turn the head or elevate the shoulder. Sooner or later, a new motor pattern develops in the cerebral cortex, and movements of the facial muscles are dissociated from those of the shoulder. Conversely, anastomosis of the twelfth nerve with the seventh nerve is followed by atrophy and paralysis of one-half of the tongue. This outcome causes little discomfort, and control of the facial muscles returns without adventitious movement of other muscles.

Anastomosis of the facial nerve with either the eleventh or the twelfth nerve should be performed as soon as possible if the nerve is cut in mastoid surgery or in removal of an acoustic neuroma. In other types of peripheral facial paralysis, surgery should be delayed for 6 months or more to determine whether spontaneous regeneration occurs.

Steroid therapy has been recommended for Bell palsy to relieve edema in the nerve. Reports of this therapy have not been convincing; therapeutic trials have not been adequately controlled, because improvement is seen spontaneously in almost all cases. Acyclovir (Zovirax) therapy is also unproven. Decompression of the nerve in the canal is recommended by some otologists to expedite and enhance return of function in Bell palsy. No available evidence suggests that this treatment changes the course of the disorder, and the surgery itself is not without risk. Therefore, many surgeons have stopped performing this operation.

Surgery may be necessary to alleviate the facial spasm that occurs spontaneously or after partial regeneration of the injured nerve. The nerve or one of its branches can be injected with alcohol or partially sectioned when the spasms are localized. These operations occasionally give permanent relief from the spasms, but the spasms usually recur when the nerve regenerates. Permanent relief can be obtained by anastomosing the seventh nerve with the eleventh or twelfth cranial nerve.

Blepharospasm, Myokymia, and Hemifacial Spasm

Blepharospasm is a state of forceful closure of the eye. Unilateral and repeated brief blepharospasm is a focal dystonia and may be part of hemifacial spasm. Bilateral blepharospasm may be seen in basal ganglia disorders, especially parkinsonism. The combination of blepharospasm and oromandibular dyskinesia is the Meige syndrome. Injections of botulinum toxin are effective and safe in treating of blepharospasm.

Facial myokymia is characterized by fine rippling movements of facial muscles. EMG shows bursts of rapidly firing motor units that tend to recur in a regular fashion. Persistent facial myokymia is sometimes a manifestation of MS, brainstem glioma, or some other disorder of the brainstem. These CNS lesions presumably interrupt descending inhibitory impulses that

act on motor neurons in the facial nucleus, thereby releasing the involuntary activity. Myokymia also can arise peripherally, especially in the acute phase of the Guillain-Barré syndrome.

Hemifacial spasm is characterized by clonic spasms of the facial muscles, usually starting around the eye and often spreading to other muscles of one side of the face. It increases in intensity during stress and may occur in sleep. The characteristic EMG findings include bursts of muscle action potentials that occur either regularly or irregularly at 5 to 20 per second. Synkinetic motor responses in muscles innervated by the facial nerve follow stimulation of the ipsilateral fifth nerve (blink reflex). Hemifacial spasm does not have the ominous implications of myokymia, but the cosmetic effects may be distressing. The cause is usually obscure, but it may follow facial nerve trauma. Treatment with anticonvulsant medication, such as carbamazepine, may be effective. Jannetta (1977) reported relief of the involuntary movements by exposing the facial nerve in the posterior fossa and decompressing vessels at the root entry zone. Botulinum toxin is also effective.

Eighth (Acoustic) Nerve

Eighth nerve disorders are described in Chapter 6.

Ninth (Glossopharyngeal) Nerve

The ninth cranial nerve contains both motor and sensory fibers. The motor fibers supply the stylopharyngeus muscle and the constrictors of the pharynx. Other efferent fibers innervate secretory glands in the pharyngeal mucosa. The sensory fibers carry general sensation from the upper part of the pharynx and the special sensation of taste from the posterior one-third of the tongue.

Isolated lesions of the nerve or its nuclei are rare and are not accompanied by perceptible disability. Taste is lost on the posterior

P.443

one-third of the tongue, and the gag reflex is absent on the side of the lesion. Injuries of the ninth nerve by infections or tumors are usually accompanied by signs of involvement of the neighboring nerves. The tractus solitarius receives taste fibers from both the seventh and the ninth nerves and may be destroyed by vascular or neoplastic lesions in the brainstem. Because the ninth, tenth, and eleventh nerves exit the jugular foramen together, tumors here produce multiple cranial nerve palsies (*jugular foramen syndrome*). The territory of the ninth nerve is the distribution also affected in glossopharyngeal neuralgia.

Glossopharyngeal neuralgia (tic douloureux of the ninth nerve) is characterized by paroxysms of excruciating pain in the region of the tonsils, posterior pharynx, back of the tongue, and middle ear. The cause of glossopharyngeal neuralgia is unknown, and no significant pathologic changes occur in most cases. Pain in the distribution of the nerve occasionally follows injury of the nerve in the neck by tumors.

Glossopharyngeal neuralgia is rare, with a frequency about 5% that of trigeminal neuralgia. The paroxysms are burning or stabbing in nature. They may occur spontaneously but are often precipitated by swallowing, talking, or touching the tonsils or posterior pharynx. The attacks last only a few seconds but sometimes last several minutes. The frequency of

attacks varies from many times daily to once in several weeks.

The diagnosis of glossopharyngeal neuralgia can be made from the description of the pain. The only differential diagnosis of any importance is neuralgia of the mandibular branch of the fifth nerve. The diagnosis of glossopharyngeal neuralgia is established when an attack of pain can be precipitated by stimulation of the tonsils, posterior pharynx, or base of the tongue or when the pain is relieved by spraying the affected area with local anesthetic. When the membrane becomes anesthetized, the pains disappear and cannot be precipitated by stimulation with an applicator. During this period, the patient can swallow food and talk without discomfort.

There may be long remissions. During a remission the trigger zone disappears. The pains almost always recur, unless they are prevented by medical therapy or the nerve is surgically sectioned. The disease does not shorten life, but affected patients may become emaciated because of the fear that each morsel of food will precipitate a pain paroxysm.

Carbamazepine, alone or in combination with phenytoin, is usually effective in producing a remission. If medical therapy is not effective, the nerve can be sectioned intracranially; the results of the operation are satisfactory. The patient is relieved of the pain, and there are no serious sequelae. The mucous membrane supplied by the ninth nerve is permanently anesthetized with loss of the gag reflex on that side. Taste is lost on the posterior one-third of the tongue. There are no motor symptoms, such as dysphagia or dysarthria, unless the tenth nerve is injured during surgery.

Tenth (Vagus) Nerve

The motor fibers of the tenth nerve arise from the nucleus ambiguus (to innervate the somatic muscles of the pharynx and larynx) and from the dorsal motor nucleus (to supply the autonomic innervation of the heart, lungs, esophagus, and stomach). The vagus nerve also carries sensory (visceral afferent) fibers from the mucosa in the oropharynx and upper part of the gastrointestinal tract; sensory fibers from the thoracic and abdominal organs send information into the tractus solitarius.

Unilateral lesions of the nucleus ambiguus in the medulla cause dysarthria and dysphagia. Because the nucleus has a considerable longitudinal extent in the medulla, lesions in the brainstem may produce dysarthria without dysphagia, or vice versa, according to the site of the lesion. Lesions confined to the lower portion of the nucleus cause dysphagia, whereas lesions of the upper portions produce dysarthria.

The dysphagia or dysarthria that follows unilateral lesions of the nucleus ambiguus is rarely severe. The voice may be hoarse, but speech is intelligible. Difficulty in swallowing solid food is usually only slight, but occasionally a transient aphagia necessitates the administration of food by tube for a few days or weeks. On examination, the palate on the affected side is lax, and the uvula deviates to the opposite side on phonation. The palatal reflex is absent on the affected side. Lesions of the nucleus ambiguus on both sides cause complete aphonia and aphagia. Bilateral destruction of this nucleus is rare, except in the terminal stages of amyotrophic lateral sclerosis (ALS). Selective destruction of cells in the nucleus ambiguus may occur in syringobulbia or intramedullary tumors, consequently causing paralysis of the vocal cords in adduction. The patient can talk and swallow without

difficulty, but inspiratory stridor and dyspnea may be severe enough to require tracheotomy.

Unilateral lesions of the dorsal motor nucleus are not accompanied by any symptoms of autonomic dysfunction. Bilateral lesions may be life-threatening. The nucleus of the tenth nerve may also be damaged by infections (especially acute poliomyelitis), intramedullary tumors, and vascular lesions. It may be involved in polyneuropathy, especially in the diphtheritic and Guillain-Barré forms.

Injury to the pharyngeal branches of the nerve results in dysphagia. Lesions of the superior laryngeal nerve produce anesthesia of the upper part of the larynx and paralysis of the cricothyroid muscle. The voice is weak and easily tires. Involvement of the recurrent laryngeal nerve, which is frequent with aneurysms of the aorta and occasionally occurs after operations in the neck, causes hoarseness and dysphonia as a result of paralysis of the vocal cords. Complete paralysis of both recurrent laryngeal nerves produces aphonia and inspiratory stridor. Partial bilateral paralysis may produce a paralysis of both abductors with severe dyspnea and inspiratory stridor; it does not cause any alteration in the voice, however.

Unilateral lesions of the vagus nerve do not produce any constant disturbance of the autonomic functions of the nerve. The heart rate may be unchanged, slowed, or accelerated. The respiratory rhythm is not affected, and no significant disturbance in the action of the gastrointestinal tract results.

Involuntary spasm of the vocal cords (*spastic dysphonia* or *laryngeal dystonia*) is of uncertain cause, but it interferes with speech. Injection of botulinum toxin is an effective treatment.

Eleventh (Spinal Accessory) Nerve

The spinal portion of the eleventh nerve innervates the sternocleidomastoid and part or all of the trapezius muscles. The fibers from the accessory portion of the nerve originate in the nucleus

ambiguus, travel with the tenth nerve through the jugular foramen, and eventually merge with the axons from motor fibers from the upper four cervical levels. Fibers from the nucleus ambiguus portion innervate the larynx. Fibers from the spinal portion pass along the carotid artery, penetrate and innervate the sternocleidomastoid muscle, and emerge in the middle of that muscle at its posterior border, crossing the posterior triangle of the neck to innervate the upper portion of the trapezius. Lesions of the spinal portion produce weakness and atrophy of the trapezius muscle, impairing rotary movements of the neck and chin to the opposite side and weakness of shrugging movements of the shoulder. Weakness of the upper portion of the trapezius results in winging of the scapula, which must be differentiated from that produced by weakness of the serratus anterior. Scapular winging from weakness of the trapezius is present at rest (arms at side) and becomes worse on abduction of the shoulder. Scapular winging from weakness of the serratus anterior is negligible at rest and worsens during flexion of the shoulder.

The nucleus of the eleventh nerve may be destroyed by infections and degenerative

disorders in the medulla, such as syringobulbia or ALS; the peripheral portion of the nerve may be involved in polyneuropathy, meningeal infection, extramedullary tumor (e.g., meningioma and neurinoma), or destructive processes in the occipital bone. Because of its passage through the posterior triangle of the neck, the nerve is susceptible to damage during lymph node biopsy, cannulation of the internal jugular vein, or carotid endarterectomy. The muscles supplied by this nerve are frequently involved in myotonic muscular dystrophy, polymyositis, and myasthenia gravis.

Twelfth (Hypoglossal) Nerve

The hypoglossal nerve is the motor nerve to the tongue. The nucleus in the medulla or the peripheral nerve portion may be injured by all the disorders mentioned in connection with the tenth and eleventh nuclei. Occlusions of the short branches of the basilar artery that nourish the paramedian area of the medulla cause paralysis of the tongue on one side and of the arm and leg on the opposite side (*alternating hemiplegia*).

Unilateral injury to the nucleus results in atrophy and paralysis of the muscles of one-half of the tongue. When the tongue is protruded, it deviates toward the paralyzed side, and while it is protruded, movement toward the normal side is absent or weakly performed. When the tongue lies on the floor of the mouth, it deviates slightly toward the healthy side, and movement of the tongue toward the back of the mouth on this side is impaired. Fibrillation of the muscles is seen in chronic processes involving the hypoglossal nucleus (e.g., syringobulbia, ALS). Bilateral paralysis of the nucleus or nerve produces atrophy of both sides of the tongue and paralysis of all movements, with severe dysarthria and resultant difficulty in manipulating food in the process of eating.

The tongue is only rarely affected by lesions in the cerebral hemispheres or corticobulbar connections. Homolateral weakness of the tongue may accompany severe hemiplegia. Such weakness appears as a slight deviation of the tongue to the paralyzed side when it is protruded. Moderate weakness of the tongue may accompany pseudobulbar palsy but is never as severe as the weakness seen with destruction of both medullary nuclei. Tremor of the tongue is seen in chronic alcoholism. Apraxia of the tongue (i.e., inability to protrude the tongue on command but preservation of the associated movements in eating or licking of the lips) frequently accompanies motor aphasia.

PERIPHERAL NERVES

The peripheral nerves are subject to injury by pressure, constriction by fascial bands, or trauma associated with injection of drugs, perforating wounds, fractures of the bones, or stretching of the nerves. Isolated or multiple nerve paralysis may also be associated with a reaction to the injection of serum or to some toxic or metabolic disorders.

The radial, common peroneal, ulnar, and long thoracic nerves are subject to damage by external pressure. The median nerve is most frequently affected by constriction by fascial bands at the wrist. The axillary nerve is commonly affected in an allergic reaction to injections of serum. The sciatic nerve is affected by direct injection of drugs. Any peripheral nerve may be damaged by perforating wounds or fractures of the bones. The frequency of involvement of the peripheral nerves by trauma is shown in Table 68.3.

TABLE 68.3. INCIDENCE OF PERIPHERAL NERVE LESIONS BY TRAUMA

Nerves of the Arm

Radial Nerve

The radial nerve arises from the posterior secondary trunk of the brachial plexus (C-5 to C-8). It is predominantly a motor nerve and innervates the chief extensors of the forearm, wrist, and fingers (Table 68.4).

TABLE 68.4. MUSCLES INNERVATED BY THE RADIAL NERVE

The radial nerve may be injured by cuts, gunshot wounds, callus formation after fracture of the humerus, pressure of crutches, or pressure against some hard surface, especially in sleep (*"Saturday night palsy"*). A complete lesion of the nerve in the axilla is characterized by paralysis of the triceps and abolition of the triceps reflex, in addition to the other signs of radial nerve palsy reviewed

in the following. Lesions of the radial nerve in the axilla are usually accompanied by evidence of injury to other nerves in this region. When the nerve is injured in the posteromedial surface of the arm, one or more of the branches to the triceps may be spared so that weakness of extension of the forearm is minimal.

The most common site of injury to the radial nerve is in the middle one-third of the arm proximal to the branch to the brachioradialis muscle. Lesions of the nerve at this level result in weakness of flexion of the forearm caused by paralysis of the brachioradialis muscle, which is a stronger flexor of the forearm than of the biceps, and paralysis of extension of the wrist, thumb, and fingers at the proximal joints. Extension at the distal phalanges is performed by the interosseus muscles. There is weakness of adduction of the hand as a result of loss of action of the extensor carpi ulnaris and loss of supination when the forearm is extended, because the supinating action of the biceps is evident only when the forearm is flexed. In addition, there is an apparent weakness of flexion of the fingers. This weakness is not real and is a result of faulty posture of the hand. When the wrist is passively extended, the fingers have normal power of flexion.

Sensory loss associated with lesions of the radial nerve is slight and is confined in most cases to a small area on the posterior radial surface of the hand and of the first and second metacarpals of the thumb and the index and middle fingers. Lesions of the nerve or its branches in the forearm or wrist are accompanied by fragments of the syndrome previously described, according to the site of the lesion.

Complete lesions of the radial nerve are followed by atrophy of the paralyzed muscles. Vasomotor or trophic disturbances are rare, unless there is an associated vascular lesion.

Causalgia rarely follows partial injury to the nerve.

Median Nerve

The median nerve comprises fibers from the sixth, seventh, and eighth cervical nerves and first thoracic roots. It arises in two heads (lateral and medial cords), derived from the upper and lower trunks of the brachial plexus. It has important motor and sensory functions. The following movements are controlled by this nerve: pronation of the forearm by the pronator quadratus and pronator teres, flexion of the hand by the flexor carpi radialis and palmaris longus, flexion of the thumb and the index and middle fingers by the superficial and deep flexors, and opposition of the thumb (Table 68.5). The sensory region of the median nerve comprises the radial side of the palm of the hand, the volar surface of the thumb and the index and middle fingers, the radial one-half of the ring finger, the dorsal surface of the distal phalanx of the thumb, and the middle and terminal phalanges of the index and middle fingers.

TABLE 68.5. MUSCLES INNERVATED BY THE MEDIAN NERVE

Injury to the median nerve in the arm is characterized by loss of ability to pronate the forearm, weakness of flexion of the wrist, paralysis of flexion of the thumb and the index finger, weakness of flexion of the middle finger, paralysis of opposition of the thumb, atrophy of the muscles of the thenar eminence, and loss of sensation in an area somewhat smaller than that of the anatomic distribution of the nerve. Lesions of the median nerve at the wrist cause paralysis and atrophy of the thenar muscles and sensory loss in the characteristic distribution.

There is absolute paralysis of few movements of the wrist or fingers in isolated lesions of the median nerve because of the compensatory action of unparalyzed muscles. Pronation can be accomplished by the action of the deltoid in holding the arm outward when the forearm is flexed and by rotation of the arm inward by the subscapularis when the arm is extended. Flexion of the wrist can be performed by the action of the flexor carpi ulnaris with deviation of the hand toward the ulnar side of the arm. There is absence of flexion in the index and the middle fingers, although the middle finger is usually influenced by movements of the ring finger and its deep flexor may be supplied by the ulnar nerve. In addition, flexion of the proximal phalanx of the fingers, including the index finger in association with extension of the distal phalanges, is possible through the action of the interosseus muscles. Although the opponens pollicis is paralyzed, feeble movements of opposition can be made by energetic contraction of the adductors that causes the thumb to move to the ulnar edge of the hand by pressing against the base of the fingers.

Partial lesions of the median nerve are more frequent than complete interruption, with dissociation in the degree of involvement of the various muscles supplied by the nerve and with little or no sensory loss. Flexion of the index finger and opposition of the thumb are the movements that are usually most affected in partial lesions.

Vasomotor disturbances are common with median nerve lesions, probably because of

associated lesions of blood vessels. The syndrome of causalgia is most commonly associated with lesions of the median nerve.

A slowly developing atrophy limited to the muscles of the outer radial side of the thenar eminence has been described by the term *partial thenar atrophy*. The atrophy is often bilateral and exceeds the motor weakness. Pain, paresthesia, and a mild degree of impairment of sensation in the distribution of the nerves with or without motor weakness are fairly common as a result of compression of the nerve by the transverse carpal segment (*carpal tunnel syndrome*). The pain is severe, often waking the patient

P.446

from sleep. The pain is usually in the thumb and the index finger but may spread to other fingers or up the arm to the axilla. These symptoms most commonly occur in middle-aged persons and are often associated with arthritis or other changes in the tendons and connective tissues of the wrists in amyloid disease, myxedema, gout, or acromegaly. Surgical division of the transverse ligament results in relief of the pain and paresthesia and gradual decrease of the weakness.

Ulnar Nerve

The ulnar nerve is the main branch of the lower cord of the brachial plexus. The fibers arise from the eighth cervical and first thoracic segments. The motor fibers innervate the muscles listed in Table 68.6. The sensory portion of the nerve supplies the skin on the palmar and dorsal surfaces of the little finger, the inner one-half of the ring finger, and the ulnar side of the hand.

TABLE 68.6. MUSCLES INNERVATED BY THE ULNAR NERVE

The ulnar nerve is frequently injured by gunshot wounds, stab wounds, and fractures of the lower end of the humerus, olecranon, or head of the radius. The nerve may be compressed in the axilla by a cervical rib. More frequently, it is compressed at the elbow in sleep or as an occupational neuritis in workers who rest their elbows on hard surfaces for prolonged periods.

Complete lesions of the ulnar nerve are characterized by weakness of flexion and adduction of the wrist and of flexion of the ring and the little fingers, paralysis of abduction and opposition of the little finger, paralysis of adduction of the thumb, and paralysis of adduction and abduction of the fingers. There is atrophy of the hypothenar muscles and the interossei. Atrophy of the first dorsal interosseous is especially obvious, seen on the dorsum of the hand between the thumb and the index finger. Sensory loss is greatest in the little finger and is present to a lesser extent on the inner side of the ring finger. There is clawing of the hand. Dissociated paralysis of the muscles supplied by the ulnar nerve may occur with partial lesions of the nerve in the arm or forearm.

Trophic and vasomotor symptoms are not prominent after complete lesions of the nerve. There may be some hyperkeratosis or changes in the palmar fascia. Irritative lesions may be accompanied by pain, but injuries to the ulnar nerve are only rarely accompanied by

causalgia.

The diagnosis of ulnar palsy can usually be made without difficulty by the posture of the hand, which is always clawed, by atrophy of the hypothenar eminence and the first dorsal interosseous, and by the characteristic distribution of paralysis. One diagnostic sign of ulnar palsy, the Froment sign, is flexion of the terminal phalanx of the thumb when the patient attempts to hold a sheet of paper between the thumb and the index finger (because the thumb cannot be adducted).

Musculocutaneous Nerve

The musculocutaneous nerve is the main branch of the upper trunk of the brachial plexus. Its fibers arise in the fifth and sixth cervical segments. The musculocutaneous nerve is a mixed nerve, innervating the coracobrachialis, biceps brachii, and brachialis muscles and transmitting cutaneous sensation from the anterior outer part and a small area on the posterior outer surface of the forearm. Isolated injuries of the nerve are rare. It may be involved in traumatic lesions of the brachial plexus.

Lesions of the musculocutaneous nerve produce weakness of flexion and supination of the forearm, a small area of hypesthesia or anesthesia on the anterior outer surface of the forearm, atrophy of the muscles on the anterior surface of the arm, and loss of the biceps reflex. Flexor movements of the forearm can still be vigorously performed by the brachioradialis muscle, which is innervated by the radial nerve. If flexion is performed against resistance, palpation reveals that the biceps muscle is inactive. If the forearm is kept in supination, forearm flexion is impossible. Because the biceps is the chief supinator of the forearm, this movement is paralyzed. Loss of function of the coracobrachialis muscle is compensated for by the action of other adductor muscles of the arm.

Axillary Nerve

The axillary nerve is the last branch of the posterior cord of the brachial plexus, including fibers from the fifth and sixth cervical segments. It innervates the deltoid muscle and transmits cutaneous sensation from a small area on the lateral surface of the shoulder.

Lesions of the axillary nerve caused by trauma or by fracture or dislocation of the head of the humerus are usually associated with injury to the brachial plexus. The axillary nerve may be involved alone or in combination with other nerves in the neuritis that follows serum (especially antitetanus) therapy. Lesions of the axillary nerve are characterized by loss of power in outward, backward, and forward movements of the arm because of paralysis of the deltoid muscle. The area of hypesthesia or anesthesia is inconstant and is much smaller than the anatomic distribution of the nerve.

Long Thoracic Nerve

The long thoracic nerve arises from the fifth, sixth, and seventh cervical roots. It is the motor nerve to the serratus anterior muscle.

Lesions of the long thoracic nerve are most common in men who do heavy labor. The nerve may be injured by continued muscular effort with the arm extended or by the carrying of

heavy sharp-cornered objects on the shoulder (“hod carrier's palsy”). Injury of the nerve following acute or chronic trauma is characterized by weakness in elevation of the arm above the horizontal plane. Winging of the scapula is a constant sign when the arm is fully abducted or elevated anteriorly (Fig. 68.2). Winging is usually absent when the arm is held at the side.

FIG. 68.2. Paralysis of the serratus magnus muscle with winging of the scapula.

P.447

Brachial Cutaneous and Antebrachial Cutaneous Nerves

The brachial and antebrachial cutaneous nerves, branches of the plexus (C8-T1), transmit sensory impulses from the inner surface of the arm and upper two-thirds of the forearm. These nerves are rarely affected, except in injuries of the medial cord of the brachial plexus. Lesions of these nerves produce hypesthesia on the inner surface of the arm and forearm.

Suprascapular Nerve

The suprascapular nerve arises from the upper trunk of the brachial plexus. Most of its fibers come from the fifth and sixth cervical roots. It is primarily motor and innervates the supraspinatus and infraspinatus muscles.

Isolated lesions of the nerve are rare. It may be wounded directly, injured in falls, or stretched by muscular overaction. It may be involved in association with the axillary nerve in serum reactions, or it may be injured in traumatic lesions of the brachial plexus. Lesions of the nerve produce an atrophic paralysis of the supraspinatus and infraspinatus muscles. Weakness of movements performed by these muscles (i.e., abduction and external rotation of the shoulder) is masked by the action of the deltoid and teres minor muscles.

Brachial Plexus

The fifth, sixth, seventh, and eighth cervical roots and the first thoracic root contribute to the formation of the brachial plexus (Fig. 68.3). The roots form three trunks: upper, middle, and lower. The upper is composed of fibers from C-5 and C-6, the middle receives fibers from C-7, and the lower receives fibers from C-8 and T-1. The redistribution of fibers from the trunks results in the formation of the cords (lateral, posterior, and medial) that contribute to the formation of the peripheral nerves as follows: lateral cord, the musculocutaneous and the lateral head of the median nerve; posterior cord, the axillary and radial nerves; the medial cord, the brachial and antebrachial cutaneous nerves, the ulnar nerve, and the medial head of the median nerve.

P.448

In addition to these major nerves, which are formed by fibers in the secondary trunks, collateral branches from the roots and trunks form nerves that innervate the shoulder and

scapular muscles and supply fibers to the interior cervical ganglion.

FIG. 68.3. Brachial plexus. (From Haymaker W, Woodhall B. *Peripheal nerve injuries*. Philadelphia: WB Saunders, 1945; with permission.)

The roots or trunks of the brachial plexus may be damaged by cuts, gunshot wounds, or direct trauma (Fig. 68.4). They may be compressed by tumors or aneurysms or stretched and torn by violent movements of the shoulder in falls, dislocations of the shoulder, the carrying of heavy packs on the shoulder ("rucksack paralysis"), and traction in delivery at birth.

FIG. 68.4. Traumatic avulsion of lower cervical nerve roots. Iophendylate cervical myelogram (anteroposterior view) demonstrates contrast in avulsed right C-8 and T-1 root pouches. (Courtesy of Dr. S.K. Hilal and Dr. J.A. Bello.)

Unilateral or bilateral disorders of the brachial plexus may follow respiratory infections. The combination of local pain, weakness, and wasting of muscle had led to the popular terms *neuralgic amyotrophy* and *brachial plexopathy*. Weakness is maximal within a few days. The cerebrospinal fluid is normal. A similar disorder may affect the lumbosacral plexus. Myelography excludes intraspinal lesions. Nerve conduction studies may localize lesions of the brachial plexus. The condition remains stable for days or weeks and then improves. Some patients recover completely; others are left with moderate or severe disability.

Various complex syndromes result from injuries to the plexus. Only the trunk syndromes are reviewed here. A minute examination of muscular and sensory disability must be made and studied in connection with anatomic charts of the plexus to determine the site of the lesion and whether the fibers have been injured at their point of emergence from the spinal cord or after the formation of the primary or secondary trunks (Tables 68.7, 68.8, and 68.9).

TABLE 68.7. INNERVATION OF THE MUSCLES OF THE SHOULDER GIRDLE

TABLE 68.8. INNERVATION OF MUSCLES OF THE ARM AND FOREARM

TABLE 68.9. INNERVATION OF THE MUSCLES OF THE HAND

Radicular Syndromes (Roots and Primary Trunks)

The syndromes of the roots and primary trunks are essentially those of the roots involved, but partial paralysis and incomplete sensory loss are common because many muscles of the arm receive innervation from two or more roots and there are extensive substitutions among the various roots.

Upper Radicular (Erb-Duchenne) Syndrome

Lesions of the upper roots (fourth, fifth, and sixth cervical roots or upper trunk) are characterized by paralysis of the deltoid, biceps, brachialis anticus, brachioradialis, pectoralis major, supraspinatus, infraspinatus, subscapularis, and teres major muscles. If the lesion is near the roots, the serratus magnus, rhomboids, and levator anguli scapulae are also paralyzed.

The motor disability resulting from lesions of the upper radicular group is essentially paralysis of flexion of the forearm and of abduction and internal and external rotation of the arm. There is also weakness or paralysis of apposition of the scapula and backward-inward movements of the arm. Sensory loss is incomplete and consists of hypesthesia on the outer surface of the arm and forearm. The biceps reflex is absent, and percussion of the styloid process of the radius produces flexion of the fingers instead of the normal flexion of the forearm.

Middle Radicular Syndrome

Injury to the seventh cervical root or the middle trunk causes paralysis of the muscles supplied by the radial nerve with the exception of the brachioradialis, which is spared entirely. Weakness is essentially similar to that seen in paralysis of the radial nerve below the origin of the fibers to the brachioradialis or in lead palsy. Sensory loss is inconstant and, when present, is limited to hypesthesia over the dorsal surface of the forearm and the external part of the dorsal surface of the hand.

Lower Radicular (Klumpke) Syndrome

Injury to the lower primary trunk or eighth cervical and first thoracic root is characterized by paralysis of the flexor carpi ulnaris, the flexor digitorum, the interossei, and the thenar and hypothenar muscles. The motor disability is similar to that of a combined lesion of the median and ulnar nerves with a flattened or simian hand.

The sensory disturbance is hypesthesia on the inner side of the arm and forearm and on the ulnar side of the hand. The triceps reflex is abolished. If the communicating branch to the inferior cervical ganglion is injured, there is paralysis of the sympathetic nerves, with resulting Horner syndrome.

Cord Syndromes of the Brachial Plexus

Lesions of the cords of the brachial plexus produce motor and sensory disturbances that

resemble those seen after injuries to two or more peripheral nerves. The syndrome of the *lateral cord* is a combination of the signs and symptoms caused by injury to the musculocutaneous nerve and the lateral head of the median nerve. These injuries are accompanied by a paralysis of the pronator teres, almost complete paralysis of the flexor carpi radialis, and weakness of the flexor pollicis and opponens. Injury to the *posterior cord* produces paralysis similar to that resulting from injury to the radial and axillary nerves. The syndrome of the *medial cord* is the same as that of the ulnar nerve combined with a paralysis of flexion of the fingers as a result of injury to the medial head of the median nerve.

Ischemic Paralysis of the Arm

Paralysis of arm muscles may follow injury to large arteries. Ischemic paralysis may follow ligation of the major vessels when the collateral circulation is inadequate, or it may follow prolonged constriction of the arm by plaster casts.

In the initial stages of ischemic paralysis, the distal part of the limb is cyanotic and edematous. Active movements of the finger and wrist muscles are possible but are of a limited range. There is diminution of cutaneous sensibility; all stimuli are poorly localized and have a painful quality. With the passage of time, the cyanosis and edema disappear, the skin becomes smooth and shiny, and the muscles undergo fibrotic changes; anesthesia extends in a glovelike distribution to the wrist or middle of the forearm. The hand is held extended, and the fingers are slightly flexed except when there are associated nerve lesions.

Ischemic paralysis can be differentiated from paralysis caused by lesions of the nerves by the absence of pulsations in the radial artery, the glovelike distribution of sensory loss, which does not correspond to that of any peripheral nerve, the fibrous consistency of the tissues, and, in some cases, persistence of feeble imperfect movements of some of the muscles.

Ischemic paralysis is frequently permanent. Improvement in some patients can be obtained by hot baths, massage, passive movements, and electrical stimulation.

NERVES OF THE LEG

Obturator Nerve

The obturator nerve is a mixed nerve that originates in the lumbar plexus from the second, third, and fourth lumbar roots. It transmits cutaneous sensation from a small area on the inner surface of the middle side of the hip, thigh, and knee joint. It innervates the obturator externus muscle, adductor longus, adductor brevis, gracilis, and adductor magnus muscles.

Lesions of the obturator nerve are uncommon. It may be injured by pressure within the pelvis by tumors, obturator hernias, or the fetal head in difficult labor. Injuries to the obturator nerve result in severe weakness of adduction and, to a lesser extent, internal and external rotation of the thigh. Pain in the knee joint is sometimes caused by pelvic involvement of the geniculate branch of the obturator.

Iliohypogastric Nerve

The iliohypogastric nerve is a mixed nerve that originates from the uppermost part of the lumbar plexus and is derived from the twelfth thoracic and first lumbar roots. It transmits cutaneous sensation from the outer and upper parts of the buttocks and the lower part of the abdomen and supplies partial innervation to the internal oblique and transversalis muscles. Lesions of the iliohypogastric

P.450

nerve are rare. It may be divided by incisions in kidney operations or together with the ilioinguinal nerve in operations in the inguinal region. Lesions of these nerves do not produce any significant motor loss, and there is only a small area of cutaneous anesthesia.

Ilioinguinal Nerve

The ilioinguinal nerve, a branch of the lumbar plexus, arises from the twelfth thoracic and first lumbar roots. It transmits cutaneous sensation from the upper inner portion of the thigh, the pubic region, and the external genitalia. Motor filaments are given off to the transversalis, internal oblique, and external oblique muscles. The ilioinguinal nerve is usually injured in connection with the iliohypogastric nerve.

Genitofemoral Nerve

This nerve originates from the second lumbar root and is primarily a sensory nerve. It transmits cutaneous sensation from an oval area on the thigh in the region of the Scarpa triangle and from the scrotum and the contiguous area of the inner surface of the thigh. Lesions of the genitofemoral nerve are rare. Irritative lesions of the nerve in the abdominal wall are accompanied by painful hyperesthesia at the root of the thigh and the scrotum.

Lateral Cutaneous Nerve of Thigh

This nerve is formed by fibers from the second and third lumbar roots. It crosses beneath the fascia iliaca to emerge at the anterosuperior iliac spine, descends in the thigh beneath the fascia lata, and divides into two branches. The posterior branch passes obliquely backward through the fascia lata and transmits cutaneous sensation from the superior external part of the buttocks. The anterior branch, which is more important clinically, pierces the fascia lata through a small fibrous canal about 10 cm below the ligament and transmits cutaneous fibrous sensation from the outer surface of the thigh.

The anterior portion of the nerve is occasionally the site of *meralgia paresthetica*, which is a sensory neuritis with dysesthesia in the nature of tingling, burning, prickling, or pins-and-needles sensations with or without sensory loss in the cutaneous distribution of the nerve. The long superficial course exposes it to various forms of trauma, but in most patients there is no history of trauma to explain the onset of symptoms. Various factors said to play a contributing role include pressure of tight belts or corsets and intermittent stretching by extensor movements of the thigh during walking. The involvement is unilateral in most cases. Men are affected about three times as frequently as women.

The diagnosis is not difficult when the dysesthesia is limited to the distribution of the

anterior division of the nerve. Pains in the lateral surface of the thigh caused by spinal lesions or pelvic tumors must be excluded by appropriate diagnostic studies.

The course of meralgia paresthetica is variable. Occasionally, symptoms spontaneously disappear after a few weeks. In most patients, they clear up by the removal of tight belts and avoidance of excessive walking. It is rarely necessary to split the fascia lata at the point of emergence of the nerve or correct the angulation of the nerve at the iliac spine.

Femoral Nerve

The femoral nerve arises from the second, third, and fourth lumbar nerves. It innervates the iliacus, psoas magnus, pectineus, sartorius, and quadriceps femoris muscles. It also transmits cutaneous sensation from the anterior surface of the thigh and, by its internal saphenous branch, from the entire inner surface of the leg and the anterior internal surface of the knee.

Traumatic lesions of the femoral nerve are uncommon. It may be compressed by tumors and other lesions in the pelvis, or it may be injured by fractures of the pubic ramus or femur. Often, there is no adequate explanation for the occurrence of an isolated femoral nerve palsy. In such cases, the nerve lesion is presumed to be a result of some toxic factor, such as diabetes, typhoid, or gout.

Injury to the femoral nerve produces paralysis of extension of the leg and weakness of flexion of the thigh. When the patient stands erect, the leg is held stiffly extended by contraction of the tensor fasciae femoris and the gracilis. Walking on level ground is possible as long as the leg can be kept extended, but if the slightest flexion occurs, the patient sinks down on the suddenly flexed knee. Climbing stairs or walking uphill is difficult or impossible. The quadriceps reflex is lost on the affected side, and cutaneous sensation is impaired in an area somewhat smaller than the anatomic distribution of the nerve.

Paralysis of the femoral nerve must be distinguished from hysterical paralysis and reflex muscular atrophies that follow fractures of the femur or lesions of the knee joint. Hysteric paralysis can be diagnosed by the presence of the knee jerk and by special tests. In hysteric paralysis, when the patient is in the recumbent position and attempts to elevate the “paralyzed” limb, there is an absence of the normal fixing movements (downward pressure of the heel) of the opposite leg (*Hoover sign*).

Orthopedic appliances that fix the knee joint in extension are of value in the treatment of femoral nerve paralysis. Transposition of tendons should be considered if paralysis persists.

Sciatic Nerve

The sciatic nerve is the largest nerve in the body. Its terminal branches consist of two distinct nerves, which are antagonists of each other: the common peroneal (external popliteal) and the tibial (internal popliteal) nerves. The common peroneal arises from the posterior and the tibial nerve from the anterior portion of the sacral plexus (i.e., L-4 to S-3). The main trunk of the sciatic nerve innervates th