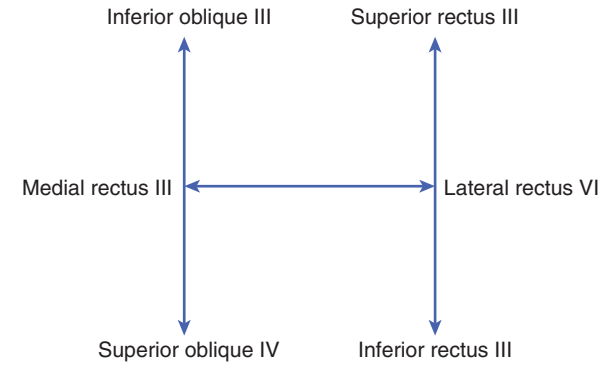

FIGURE 112-5 Movements of eye muscles and their innervation.

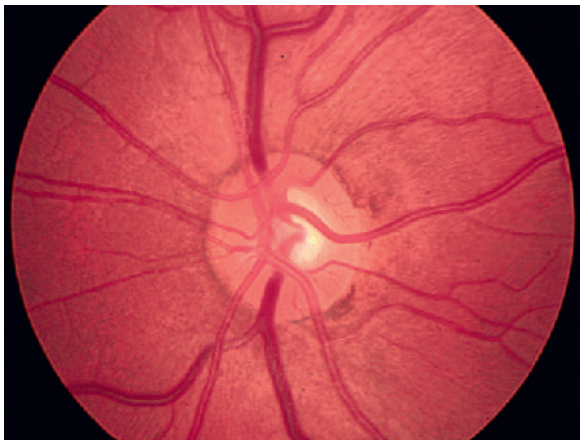

FIGURE 112-6 A normal optic disk on fundoscopic examination.

TABLE 112-1 MAJOR CAUSES OF ACUTE OPHTHALMOPLEGIA

CONDITION	DIAGNOSTIC FEATURES
BILATERAL	
Botulism	Contaminated food; high-altitude cooking; pupils involved
Myasthenia gravis	Fluctuating degree of paralysis; responds to edrophonium chloride (Tensilon) IV
Wernicke encephalopathy	Nutritional deficiency; responds to thiamine IV
Acute cranial polyneuropathy	Antecedent respiratory infection; elevated CSF protein level
Brainstem stroke	Other brainstem signs
UNILATERAL	
P Comm aneurysm	Third cranial nerve, pupil involved
Diabetic-idiopathic	Third or sixth cranial nerve, pupil spared
Myasthenia gravis	As above
Brainstem stroke	As above

CSF, Cerebrospinal fluid; IV, intravenous; P Comm, posterior communicating artery.

hemiplegia. Lesions of the brainstem cause conjugate paralysis to the ipsilateral side (eyes looking toward the side of the hemiplegia). Lesions of the medial longitudinal fasciculus, which connects the nuclei of the oculomotor and abducens nerves, lead to *internuclear ophthalmoplegia.* In this case, horizontal gaze results in failure of adduction in one eye and nystagmus in the abducting eye. The lesion is on the side of failed adduction; bilateral lesions are frequently seen in multiple sclerosis. Table 112-1 lists the major causes of acute opthalmoplegia.

Funduscopy

The retina should be carefully examined in each patient by direct ophthalmoscopy, which provides a magnified view of the fundus without the necessity for dilation of the pupil (Fig. 112-6).

Monocular Visual Loss

Loss of vision in one eye may be caused by lesions of the cornea, lens, vitreous, retina, or optic nerve. Careful funduscopic examination will usually reveal ocular and retinal lesions, but acute lesions of the optic nerve (optic neuritis) may not be associated with abnormalities of the optic nerve head. *Optic neuritis* is characterized by inflammation of the optic nerve accompanied by non-homonymous visual defects. The term *papillitis* refers to ophthalmoscopically observable changes in the optic nerve; *retrobulbar neuritis* refers to this condition without observable changes in the funduscopic examination findings ("the doctor sees nothing and the patient sees nothing").

The patient with optic neuritis complains of difficulty with vision in the affected eye. Loss of vision may be insidious and recognized only when the unaffected eye is accidentally occluded. Patients often complain of periorbital pain on eye movement on presentation. The evolution of visual loss is highly variable, progressing over a period ranging from less than a day to several weeks, although most patients will have reached their maximal visual deficit in 3 to 7 days. Patients may describe their vision as blurred or dim, and colors may appear less bright than usual or "gray." Red desaturation may occur with optic neuritis and may be detected using Ishihara color plates. At the time the patient is first examined, visual acuity may range from almost 20/20 to the extreme of total blindness. Examination of the visual field shows defects within the central 25 degrees, with central and paracentral scotomas being the most common types. An afferent pupillary defect is frequently present. The funduscopic examination is abnormal in only about one half of the cases. The disc may appear hyperemic with blurred margins, and hemorrhages, when present, are few and found only on the disc or in the area immediately surrounding the disc. Optic neuritis should be treated acutely with high-dose intravenous corticosteroids because this is proven to shorten time to recovery. The most common cause of optic neuritis is multiple sclerosis. Bilateral optic neuritis is much less common and may coincide with longitudinally extensive transverse myelitis, known as *neuromyelitis optica* (NMO) or *Devic disease.* The recent discovery of antibodies directed toward aquaporin 4 (a water channel present on astrocytes and vascular endothelial cells), associated with NMO, has identified this as a separate disease entity, with a different treatment regimen emerging for it. The NMO antibody is the first sensitive and specific biomarker associated with a central demyelinating disorder.

The optic nerve may be compressed by tumors that originate in the nerve itself or in the region of the optic chiasm. Pathologic processes that appear acutely as optic disc edema frequently result in a secondary optic atrophy, including papilledema, optic neuritis, and ischemic optic neuropathy. Glaucoma is responsible