or elicited by sensory stimulation. Flexion of the elbows and wrists and supination of the arm (decorticate posturing) suggests bilateral damage rostral to the midbrain, whereas extension of the elbows and wrists with pronation (decerebrate posturing) indicates damage to motor tracts in the midbrain or caudal diencephalon. The less frequent combination of arm extension with leg flexion or flaccid legs is associated with lesions in the pons. These concepts have been adapted from animal work and cannot be applied with precision to coma in humans. In fact, acute and widespread disorders of any type, regardless of location, frequently cause limb extension, and almost all extensor posturing becomes predominantly flexor as time passes.

LEVEL OF AROUSAL

A sequence of increasingly intense stimuli is used to determine the threshold for arousal and the motor response of each side of the body. The results of testing may vary from minute to minute, and serial examinations are useful. Tickling the nostrils with a cotton wisp is a moderate stimulus to arousal—all but deeply stuporous and comatose patients will move the head away and arouse to some degree. An even greater degree of responsiveness is present if the patient uses his hand to remove an offending stimulus. Pressure on the knuckles or bony prominences and pinprick stimulation are humane forms of noxious stimuli; pinching the skin causes unsightly ecchymoses and is generally not necessary but may be useful in eliciting abduction withdrawal movements of the limbs. Posturing in response to noxious stimuli indicates severe damage to the corticospinal system, whereas abduction-avoidance movement of a limb is usually purposeful and denotes an intact corticospinal system. Posturing may also be unilateral and coexist with purposeful limb movements, reflecting incomplete damage to the motor system.

BRAINSTEM REFLEXES

Assessment of brainstem function is essential to localization of the lesion in coma (Fig. 328-3). The brainstem reflexes that are examined are pupillary size and reaction to light, spontaneous and elicited eye movements, corneal responses, and the respiratory pattern. As a rule, coma due to bilateral hemispheral disease preserves these brainstem activities, particularly the pupillary reactions and eye movements. However, the presence of abnormal brainstem signs does not always indicate that the primary lesion is in the brainstem because hemispheral masses can cause secondary brainstem damage by the earlier described transtentorial herniations.

Pupillary Signs Pupillary reactions are examined with a bright, diffuse light (preferably not an ophthalmoscope, which illuminates only a limited part of the retina). Reactive and round pupils of midsize (2.5–5 mm) essentially exclude midbrain damage, either primary or secondary to compression. A response to light may be difficult to appreciate in pupils <2 mm in diameter, and bright room lighting mutes pupillary reactivity. One enlarged and poorly reactive pupil (>6 mm) signifies compression or stretching of the third nerve from the effects of a cerebral mass above. Enlargement of the pupil contralateral to a hemispheral mass may occur but is infrequent. An oval and slightly eccentric pupil is a transitional sign that accompanies early midbrain–third nerve compression. The most extreme pupillary sign, bilaterally dilated and unreactive pupils, indicates severe midbrain damage, usually from compression by a supratentorial mass. Ingestion of drugs with anticholinergic activity, the use of mydriatic eye drops, and direct ocular trauma are among the causes of misleading pupillary enlargement.

Unilateral miosis in coma has been attributed to dysfunction of sympathetic efferents originating in the posterior hypothalamus and descending in the tegmentum of the brainstem to the cervical cord. It is therefore of limited localizing value but is an occasional finding in patients with a large cerebral hemorrhage that affects the thalamus. Reactive and bilaterally small (1–2.5 mm) but not pinpoint pupils are seen in metabolic encephalopathies or in deep bilateral hemispheral lesions such as hydrocephalus or thalamic hemorrhage. Even smaller reactive pupils (<1 mm) characterize narcotic or barbiturate overdoses but also occur with extensive pontine hemorrhage. The response to naloxone and the presence of reflex eye movements (see below) assist in distinguishing between these.

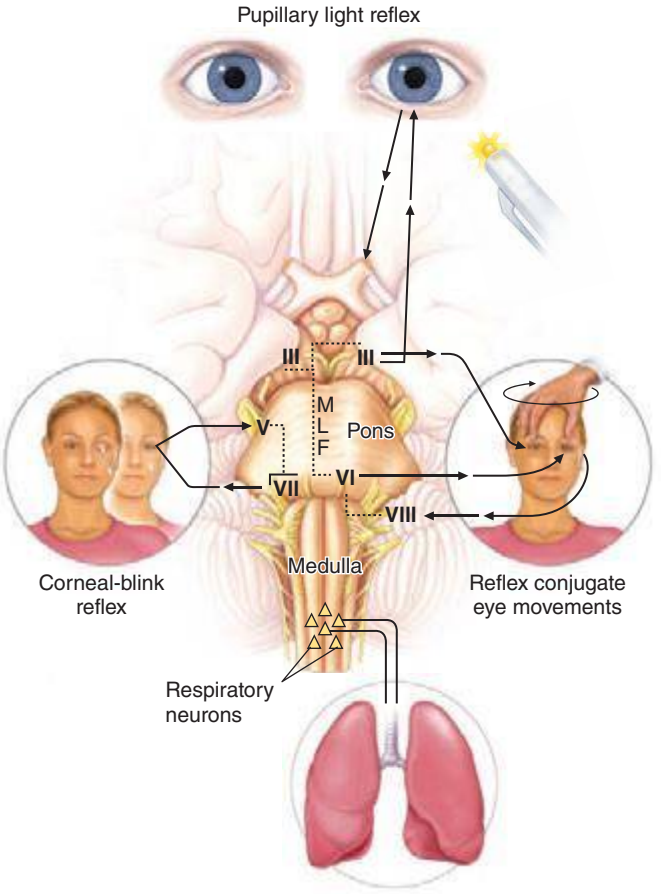

FIGURE 328-3 Examination of brainstem reflexes in coma. Midbrain and third nerve function are tested by pupillary reaction to light, pontine function by spontaneous and reflex eye movements and corneal responses, and medullary function by respiratory and pharyngeal responses. Reflex conjugate, horizontal eye movements are dependent on the medial longitudinal fasciculus (MLF) interconnecting the sixth and contralateral third nerve nuclei. Head rotation (oculocephalic reflex) or caloric stimulation of the labyrinths (oculovestibular reflex) elicits contraversive eye movements (for details see text).

Ocular Movements The eyes are first observed by elevating the lids and observing the resting position and spontaneous movements of the globes. Lid tone, tested by lifting the eyelids and noting their resistance to opening and the speed of closure, is progressively reduced as unresponsiveness progresses. Horizontal divergence of the eyes at rest is normal in drowsiness. As coma deepens, the ocular axes may become parallel again.

Spontaneous eye movements in coma often take the form of conjugate horizontal roving. This finding alone exonerates damage in the midbrain and pons and has the same significance as normal reflex eye movements (see below). Conjugate horizontal ocular deviation to one side indicates damage to the pons on the opposite side or, alternatively, to the frontal lobe on the same side. This phenomenon is summarized by the following maxim: *The eyes look toward a hemispheral lesion and away from a brainstem lesion.* Seizures also drive the eyes to one side but usually with superimposed clonic movements of the globes. The eyes may occasionally turn paradoxically away from the side of a deep hemispheral lesion ("wrong-way eyes"). The eyes turn down and inward with thalamic and upper midbrain lesions, typically thalamic hemorrhage.