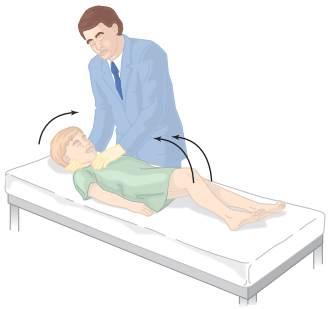

FIGURE 105-2 Elicitation of Brudzinski's sign of meningeal irritation, as seen in infectious meningitis or subarachnoid hemorrhage. (From Aminoff MJ, Greenberg DA, Simon RP: Clinical neurology, Stamford, Conn., 1996, Appleton and Lange.)

a few fingerbreadths of the chest, patients with irritated meninges reflexively flex one or both knees. This sign, called *Brudzinski's reflex*, is usually asymmetrical and not dramatic, but any evidence of knee flexion during passive neck flexion mandates that the cerebrospinal fluid be examined.

Is CT required before lumbar puncture in this setting? In the absence of lateralized signs (e.g., hemiparesis) supporting a superimposed mass lesion, a spinal puncture should be performed immediately. Although rare cases of herniation after lumbar puncture have been reported in children with bacterial meningitis, the urgency of diagnosis and treatment at the point of coma is paramount. The time required for CT may result in a fatal therapeutic delay. An alternative approach involves obtaining blood cultures and immediately initiating antibiotic therapy with subsequent lumbar puncture. With this approach, the cerebrospinal fluid cell count, glucose determination, and protein content are unchanged, and Gram stain and culture often remain positive despite a short period of antibiotic treatment. Bacterial antigens in the cerebrospinal fluid or blood can also be detected.

Separation of Structural from Metabolic Causes of Coma

The goal of this differential diagnosis is achieved by neurologic examination. Because the evaluation and potential treatments for structural and metabolic coma are widely divergent and the disease processes in both categories are often rapidly progressive, initiating prompt medical and surgical evaluation may be lifesaving. Identification of a structural versus a metabolic cause is accomplished by focusing on three features of the neurologic examination: the *motor response* to a painful stimulus, *pupillary function,* and *reflex eye movements.*

Motor Response

Asymmetrical or reflex function of the motor system provides the clearest indication of a mass lesion. Elicitation of a *motor response* requires that a painful stimulus be applied, to which the patient will react. The patient's arms should be placed in a semiflexed posture, and a painful stimulus should be applied to the head or trunk. Strong pressure on the supraorbital ridge or pinching of the skin on the anterior chest or inner arm is the most useful method; finger nail bed pressure is also used, but it makes the interpretation of upper limb movement difficult.

The neurologic examination of a patient with an expanding hemispheric mass lesion is shown in Figure 105-1. Hemispheric masses in their *early diencephalic* stage (i.e., compromising the brain above the thalamus), produce appropriate movement of one upper extremity—that is, movement toward the painful stimulus. The attenuated contralateral arm movement reflects a hemiparesis. This lateralized motor response in a comatose patient establishes the working diagnosis of a hemispheric mass. As the mass expands to involve the thalamus (*late diencephalic* stage), the response to pain becomes reflex arm flexion associated with extension and internal rotation of the legs *(decorticate posturing)*; asymmetry of the response in the upper extremities is seen. With further brain compromise at the midbrain level, the reflex posturing in the arms changes such that both arms and legs respond by extension *(decerebrate posturing)*; at that level, the asymmetry tends to be lost. At this point, the pupils become midposition in size, and the light reflex is lost, first unilaterally and then bilaterally. With further progression to the level of the pons, the most frequent finding is no response to painful stimulation, although spinal-mediated movements of leg flexion may occur.

The classic postures illustrated in Figure 105-1, and particularly their asymmetry, strongly support the presence of a mass lesion. However, these motor movements, especially early in coma, are most frequently seen as fragments of the fully developed, asymmetrical flexion or extension of the arms (illustrated as decorticate and decerebrate postures in Figure 105-1). A small amount of asymmetrical flexion or extension of the arms in response to a painful stimulus carries the same implications as the full-blown postures of decortication or decerebration.

Metabolic lesions do not compromise the brain in a progressive, level-by-level manner as do hemispheric masses, and they rarely produce the asymmetrical motor signs typical of masses. Reflex posturing may be seen, but it lacks the asymmetry of decortication seen with a hemispheric mass, and it is not associated with the loss of pupillary reactivity at the stage of decerebration.

Pupillary Reactivity

In metabolic coma, one feature is central to the examination: Pupillary reactivity is present. This reactivity is seen both early in metabolic coma, when an appropriate motor response to pain may be retained, and late in coma, when no motor responses can be elicited. The pupillary reaction in metabolic coma is lost only when coma is so deep that the patient requires ventilatory and blood pressure support.

Reflex Eye Movements

The presence of inducible lateral eye movements reflects the integrity of the pons and midbrain. These reflex eye movements (see Fig. 105-1) are brought about with the use of passive head rotation to stimulate the semicircular canal input to the vestibular system (so-called *doll's eyes maneuver*) or by inhibiting the function of one semicircular canal by infusing ice water against the tympanic membrane (caloric testing).

In metabolic coma, reflex eye movements may be lost or retained. Lack of inducible eye movements with the doll's eyes maneuver, in the setting of preserved pupillary reactivity, is