fibers. These myopathies may not affect the size of individual motor unit action potentials and are detected by a discrepancy between the electrical activity and force of a muscle.

Psychogenic Weakness Weakness may occur without a recognizable organic basis. It tends to be variable, inconsistent, and with a pattern of distribution that cannot be explained on a neuroanatomic basis. On formal testing, antagonists may contract when the patient is supposedly activating the agonist muscle. The severity of weakness is out of keeping with the patient's daily activities.

Hemiparesis Hemiparesis results from an upper motor neuron lesion above the midcervical spinal cord; most such lesions are above the foramen magnum. The presence of other neurologic deficits helps localize the lesion. Thus, language disorders, for example, point to a cortical lesion. Homonymous visual field defects reflect either a cortical or a subcortical hemispheric lesion. A "pure motor" hemiparesis of the face, arm, and leg often is due to a small, discrete lesion in the posterior limb of the internal capsule, cerebral peduncle, or upper pons. Some brainstem lesions produce "crossed paralyses," consisting of ipsilateral cranial nerve signs and contralateral hemiparesis (Chap. 446). The absence of cranial nerve signs or facial weakness suggests that a hemiparesis is due to a lesion in the high cervical spinal cord, especially if associated with the Brown-Séquard syndrome (Chap. 456).

Acute or episodic hemiparesis usually results from focal structural lesions, particularly rapidly expanding lesions, or an inflammatory process. *Subacute hemiparesis* that evolves over days or weeks may relate to subdural hematoma, infectious or inflammatory disorders (e.g., cerebral abscess, fungal granuloma or meningitis, parasitic infection, multiple sclerosis, sarcoidosis), or primary and metastatic neoplasms. AIDS may present with subacute hemiparesis due to toxoplasmosis or primary central nervous system (CNS) lymphoma. *Chronic hemiparesis* that evolves over months usually is due to a neoplasm or vascular malformation, a chronic subdural hematoma, or a degenerative disease.

Investigation of hemiparesis (Fig. 30-3) of acute origin starts with a computed tomography (CT) scan of the brain and laboratory studies. If the CT is normal, or in subacute or chronic cases of hemiparesis, magnetic resonance imaging (MRI) of the brain and/or cervical spine (including the foramen magnum) is performed, depending on the clinical accompaniments.

Paraparesis *Acute paraparesis* is caused most commonly by an intraspinal lesion, but its spinal origin may not be recognized initially if the legs are flaccid and areflexic. Usually, however, there is sensory loss in the legs with an upper level on the trunk, a dissociated sensory loss suggestive of a central cord syndrome (Chap. 456), or hyperreflexia in the legs with normal reflexes in the arms. Imaging the spinal cord (Fig. 30-3) may reveal compressive lesions, infarction (proprioception usually is spared), arteriovenous fistulas or other vascular anomalies, or transverse myelitis (Chap. 456).

Diseases of the cerebral hemispheres that produce acute paraparesis include anterior cerebral artery ischemia (shoulder shrug also is affected), superior sagittal sinus or cortical venous thrombosis, and acute hydrocephalus.

Paraparesis may result from a cauda equina syndrome, for example, after trauma to the low back, a midline disk herniation, or an intraspinal tumor; although the sphincters are often affected, hip flexion often is spared, as is sensation over the anterolateral thighs. Rarely, paraparesis is caused by a rapidly evolving anterior horn cell disease (such as poliovirus or West Nile virus infection), peripheral neuropathy (such as Guillain-Barré syndrome; Chap. 460), or myopathy (Chap. 462e).

Subacute or chronic spastic paraparesis is caused by upper motor neuron disease. When associated with lower-limb sensory loss and sphincter involvement, a chronic spinal cord disorder should be considered (Chap. 456). If hemispheric signs are present, a parasagittal meningioma or chronic hydrocephalus is likely. The absence of spasticity in a long-standing paraparesis suggests a lower motor neuron or myopathic etiology.

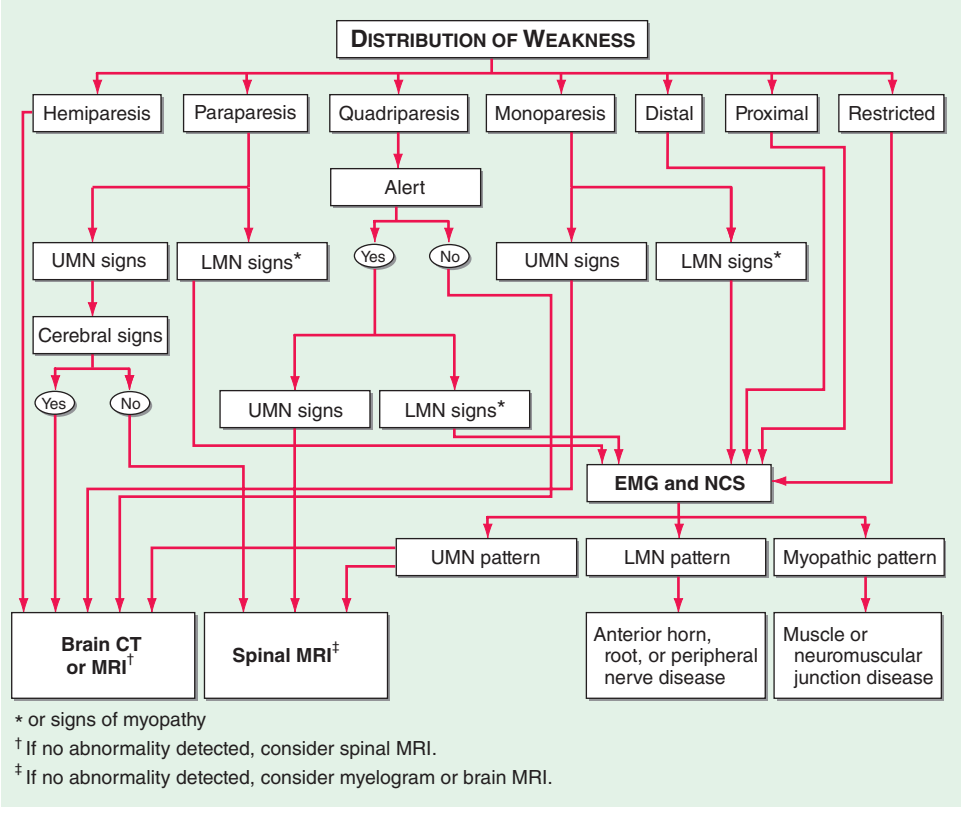

FIGURE 30-3 An algorithm for the initial workup of a patient with weakness. CT, computed tomography; EMG, electromyography; LMN, lower motor neuron; MRI, magnetic resonance imaging; NCS, nerve conduction studies; UMN, upper motor neuron.