important factors. The presence or absence of bladder and bowel dysfunction, sexual dysfunction, paresthesias or altered sensation, and muscle pain or spasm should also be elicited. Recent history of infectious illness, trauma, new medications, exposure to toxins, alcohol and drug use should also be considered. Table 11-1 lists a few of the serious causes of acute neuromuscular weakness with their distinguishing characteristics and management.

The patient's age, comorbidities, and overall vigor or frailty should be taken into account. A thorough review of organ systems should be performed. The presence or absence of fever, infectious symptoms, fatigue, chest discomfort, dyspnea, malaise, abdominal discomfort, alteration of bowel habits, especially melena or hematochezia, and headache should be elicited. Careful medication review, especially of diuretics, beta-blockers, and psychotropic medications, may suggest

Table 11-1 Neuromuscular Diseases: A Brief Description

DISEASE	MECHANISM	HISTORICAL FEATURES/EXAM FINDINGS	ED MANAGEMENT
Botulism Toxin 12–72 hr postingestion	Neurotransmission Toxin prevents release of acetylcholine at the neuromuscular junction	Ingestion of contaminated canned goods 50% have GI symptoms Postural hypotension Diplopia, blurred vision, ptosis, facial weakness, dysphagia, respiratory compromise, then limb weakness	Supportive care, ICU admission Notify Health Dept/CDC Trivalent antitoxin (May try guanidine hydrochloride, facilitates release of acetylcholine from nerve endings; anticholinesterase drugs not helpful)
Myasthenia Gravis Idiopathic	Neurotransmission Decreased number of functioning acetylcholine receptors	Mild infection may exacerbate symptoms Fluctuating weakness; easy fatigability of voluntary muscles; cranial nerves involved with ptosis and diplopia in >25%; normal pupillary responses; normal sensation; normal reflexes Improves with rest May have a coexisting thymoma (CXR, chest CT)	Supportive care, ICU admission Neurology consult Edrophonium/neostigmine test Bedside spirometry Measure serum acetylcholine receptor antibody levels Tx: Anticholinesterase drugs—neostigmine; pyridostigmine
Organophosphate/ Carbamante Poisoning Immediate–3 wk	Neurotransmission Cholinergic crisis from inhibition of acetylcholine Neuropathy (weeks after exposure)	History of insecticide exposure Gastrointestinal symptoms, agitation, miosis, paralysis, diaphoresis, muscle weakness, bradycardia Cramping muscle pain, distal numbness and paresthesias, progressive muscle weakness; decreased reflexes; can develop flaccid/ wasted leg muscles	Decontamination Supportive care, ICU admission Atropine Pralidoxime (2-Pam)
Tetanus Toxin 3 wk 10–60% fatality	Neurotransmission Toxin interferes with release of inhibitory transmitters including GABA; results in motor nerve hyperactivity	Immunization status History of cutaneous infection Trismus, laryngospasm, painful muscle spasms and rigidity (opisthotonos), autonomic instability	Supportive care, ICU admission Débridement of wounds Tetanus immunoglobulin Penicillin for the infection High-dose benzodiazapines Neuromuscular blockade
Tick Paralysis Toxin 2–7 days Rocky Mountain wood tick and the American dog tick are most common in the USA	Neurotransmission Toxin reduces motor neuron action potential and the action of acetylcholine	History of outdoor activities/tick bite Progressive, ascending, flaccid weakness over several hours may lead to respiratory failure; may present as acute ataxia without muscle weakness; decreased or absent reflexes; ophthalmoplegia and bulbar palsy can occur	Removal of the embedded tick (look at the hairline/in the scalp) Supportive care Full recovery if tick removed; 10% fatality if not recognized
Ciguatoxin Toxin 12–24 hr; neurologic symptoms can last months	Neuropathy Toxin causes cell membrane excitability and instability	History of ingestion of large, tropical fish Diarrhea, abdominal pain, nausea, and vomiting are followed by painful paresthesias, ataxia, altered hot/cold perception, myalgias, bradycardia, and hypotension Rarely, death occurs through respiratory failure	Supportive care, ICU admission Atropine for bradycardia Hydration IV mannitol can be helpful
Diphtheria Toxin 2 wk–3 mo after infection	Neuropathy Lower motor neuron	Immunization status History of throat infection with pseudomembrane; cutaneous infection Palatal weakness, impaired pupillary responses, generalized sensorimotor polyneuropathy; respiratory failure; motor weakness of the proximal muscle groups and extending distally	Supportive care, ICU admission Equine diphtheria antitoxin Erythromycin or penicillin G for 14 days to halt toxin production, treat localized infection and prevent transmission of organisms Immunization

Table 11-1 Neuromuscular Diseases: A Brief Description—cont'd

DISEASE	MECHANISM	HISTORICAL FEATURES/EXAM FINDINGS	ED MANAGEMENT
Gullian-Barré Syndrome Idiopathic 1–4 wk 75% recover 5% fatality	Neuropathy Lower motor neuron Immune-mediated polyneuropathy Multiple variants	May have a history of infection; viral infection; *Campylobacter jejuni* in 15–40% Symmetrical ascending motor neuropathy; decreased/absence reflexes; mild sensory involvement; autonomic dysfunction; can progress to respiratory compromise	Lumbar puncture: CSF with elevated protein but normal WBC Bedside spirometry Plasmapheresis and IVIG Consider ICU admission Neurology consult
Transverse Myelitis Idiopathic, postinfectious, autoimmune Rapid onset (hours–days)	Neuropathy Upper motor neuron Axonal demyelination	Loss of spinal cord functions with symptoms depending on the level of the lesion; thoracic is most common Acute, focal back pain; distal muscle weakness; abnormal sensation; urinary retention/loss of bowel control; muscles may be flaccid; decreased or absent reflexes initially Differentiate from spinal cord compression, trauma or infarct; may be the first sign of multiple sclerosis	Supportive care, ICU admission if C-spine level for respiratory support Spine radiograph to evaluate for boney lesion Stat MRI/CT myelogram Decompress bladder
Electrolyte Imbalance	Myopathy	History of nausea/vomiting/diarrhea History of renal failure, alcohol dependence, new medication Ascending symmetric muscle weakness with normal to diminished reflexes	ECG Electrolyte panel: Na, K, Cl, PO_4, Ca, and Mg Renal function Correct the abnormality; close hemodynamic monitoring
Polymyositis Autoimmune	Myopathy	History of connective tissues disorders or cancer Progressive at a variable rate; muscle weakness and wasting; ascending pattern with proximal limb and girdle muscle involvement; muscle pain; dysphagia; respiratory difficulty; can have an erythematous periorbital and eyelid rash (dermatomyositis)	Elevated CPK, rhabdomyolysis rare Normal ESR Supportive care Corticosteroids

Adapted and expanded from LoVecchio, et al: Approach to generalized weakness and peripheral neuromuscular disease. Emerg Med Clin North Am 15:605, 1997.
CDC, Centers for Disease Control and Prevention; CPK, creatine phosphokinase; CSF, cerebrospinal fluid; CT, computed tomography; CXR, chest radiograph; ESR, erythrocyte sedimentation rate; GABA, γ-aminobutyric acid; GI, gastrointestinal; ICU, intensive care unit; IVIG, intravenous immunoglobulin; MRI, magnetic resonance imaging; WBC, white blood cell count.

electrolyte disturbances or generalized medication side effects.

In adults older than age 50, particularly women, the complaint of generalized weakness should prompt consideration of cardiac ischemia.

In adults older than age 65, a complaint of weakness may be the only symptom of a serious infection, electrolyte disturbance, or cardiovascular compromise. When this complaint is combined with a recent fall, altered mental status, or urinary incontinence, urosepsis should be considered. When this complaint is accompanied by the report of poor sleep, dyspnea, or decreased exercise tolerance, acute coronary syndrome or heart failure should be considered. Consideration should be given to situational orthostasis resulting in a sensation of weakness accompanied by a presyncopal feeling; examples include postparandial hypotension and post-tussive or micturition near-syncope.

Examination

Fever, hypotension, tachycardia, or tachypnea may provide clues regarding the source of the patient's complaint (Table 11-2). If severe weakness is present, an assessment of the patient's ability to maintain the airway and the adequacy of respiration is indicated (Fig. 11-1).

The neurologic exam should focus on clarifying if the patient is experiencing true loss of strength along with the distribution of the deficits (Table 11-3). A complete examination, including cranial nerves, and gait, where possible, is helpful. The motor exam should be systematic and thorough. Muscle bulk, strength, tone, and the presence or absence of abnormal movements should be noted. Sarcopenia (age-associated loss of muscle mass and function) is normal in the older adult. In this situation, the loss of power is uniform in all limbs. Walking on heels, toes, and in tandem is a good test of strength as well as coordination and proprioception. Gait apraxia has a wide differential and should prompt investigation for cerebellar abnormality; normal pressure hydrocephalus should be considered in the patient who has simultaneous incontinence and decreased cognitive function. Fine muscle fasciculations typically point to an LMN disorder, whereas spasticity, greater in the flexors than extensors, is seen in UMN lesions.

Ancillary Testing

Patients presenting with weakness can have myriad underlying abnormalities. Although testing will be guided by the history and exam, virtually all patients require a complete blood count to evaluate for anemia or blood loss and serum electrolytes, glucose, and creatinine. An electrocardiogram