epilepsy or progressive myoclonic epilepsy (PME-2) caused by mutations in the *EPM2A* gene or the *NHLRC1* gene and ceroid lipofuscinosis. In patients with less severe or absent epilepsy, mitochondrial disorders and neurodegenerative disorders affecting the cerebellum (i.e., SCAs) should be considered. Essential myoclonus is a relatively benign familial condition characterized by multifocal, very brief, lightning-like movements that are frequently alcohol-sensitive. Mutations in the *epsilon-sarcoglycan* gene have been associated with myoclonus seen in association with dystonia (myoclonic-dystonia).

TREATMENT

Myoclonus

Treatment primarily consists of managing the underlying condition or removing an offending agent. Pharmacologic therapy involves one or a combination of GABAergic agents such as valproic acid (800–3000 mg/d), piracetam (8–20 g/d), clonazepam (2–15 mg/d), levetiracetam (1000–3000 mg/d), or primidone (500–1000 mg/d). Treatment may be associated with striking clinical improvement in chronic cases (e.g., postanoxic myoclonus, progressive myoclonic epilepsy) in which a cortical origin for the myoclonic discharges has been identified. The serotonin precursor 5-hydroxytryptophan (plus carbidopa) may be useful in some cases of postanoxic myoclonus. DBS can be highly effective in myoclonus dystonia.

DRUG-INDUCED MOVEMENT DISORDERS

This important group of movement disorders is primarily associated with drugs that block dopamine receptors (neuroleptics) or central dopaminergic transmission. These drugs are widely used in psychiatry, but it is important to appreciate that drugs used in the treatment of nausea or vomiting (e.g., prochlorperazine [Compazine]) or gastroesophageal disorders (e.g., metoclopramide) are neuroleptic agents and can also cause these disorders. Hyperkinetic movement disorders secondary to neuroleptic drugs