

FAST FACTS AND CONCEPTS #114
MYOCLONUS**Nicholas DeMonaco and Robert Arnold MD**

Background Myoclonus is an abnormal movement described as a sudden, brief, shock-like, involuntary movement caused by active muscle contraction (positive myoclonus) or inhibition of ongoing muscle contraction (negative myoclonus). Myoclonus can have a distribution that is focal, multifocal, or generalized. This *Fast Fact* discusses its causes, evaluation, and therapy.

Characteristics and Differential Diagnosis Hiccups are an example of normal, physiological positive myoclonus, while asterixis is an example of negative myoclonus seen with metabolic encephalopathy. In nocturnal myoclonus or periodic leg movement disorder, there is activity in the flexor muscles of the legs and feet during light sleep. It can be seen in the setting of chronic nervous system diseases or in elderly patients with no other abnormalities. The brief, shock-like movements of myoclonus may be difficult to distinguish from other involuntary movements such as cramps, spasms, fasciculations, and dystonia. Fasciculations are brief involuntary muscle twitches that, unlike myoclonus, often do not result in movement across a joint. Dystonia is characterized as slow, repetitive, patterned, sustained movements (an example is writers cramp). An acute dystonic reaction is often caused by dopamine blocking medications including certain antipsychotics (haloperidol), antiemetics (metoclopramide), and calcium-channel blockers.

Causes The etiologies of myoclonus are numerous. Near the end of life, metabolic abnormalities and medication-induced myoclonus predominate. Metabolic causes include liver failure, renal failure, hyponatremia, and hypoglycemia. The medications and toxins associated with myoclonus include opioids, anticonvulsants (gabapentin, phenytoin, valproate, lamotrigine, and phenobarbital), tricyclic antidepressants and selective serotonin reuptake inhibitors, contrast dye, anesthetics, antibiotics (penicillins, cephalosporins, imipenem, and quinolones), cannabinoids and the chemotherapeutic agent ifosfamide. Opioid-induced myoclonus occurs commonly and is often misdiagnosed (See *Fast Facts #57, 58*). When myoclonus occurs due to toxins or medications, the jerks are usually multifocal or generalized, may be provoked by a stimulus or voluntary movement, and are often accompanied by encephalopathy. Other causes of myoclonus include focal CNS damage from tumors, stroke, and encephalitis, generalized CNS dysfunction such as encephalopathies (viral, metabolic, genetic, or neurodegenerative), seizure disorders, anoxic injury, and disorders affecting the spinal cord and peripheral nerves.

Treatment Myoclonus can disrupt sleep, make coordinated movements difficult, and be bothersome to patients or families. Treatment consists of correction of the underlying cause and symptomatic treatment of the myoclonus. If the offending agent is a non-essential medication, it should be discontinued. In the case of opioid-induced myoclonus, rotation to a different opioid may help. Benzodiazepines are the primary symptomatic treatment at end-of-life. While any benzodiazepine will work, clonazepam and lorazepam are commonly used. A continuous infusion of midazolam has also been suggested given the drug's compatibility with morphine and short half-life, allowing rapid dose titration. Sedation is likely when using benzodiazepines. If sedation is to be avoided, anticonvulsants such as levetiracetam (1,000-3,000 mg/day) and valproic acid (1200-2000 mg/day) may be helpful. The muscle relaxant dantrolene in doses of 50-100 mg/day has been reported as effective.

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