

Tremors in Elderly Persons: Clinical Features and Management

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Tremor, the most common movement disorder in the general population, is an involuntary, rhythmic, oscillatory movement of a body part. It is produced by activation of reciprocally innervated, antagonistic muscle groups, which leads to their repeated contractions.¹ Tremors can lead to social and physical deterioration and may be a harbinger of a complex medical syndrome. Although many forms of tremor may begin earlier in life, they progress with time and often do not come to the attention of a physician until a patient is elderly. In one study, 98% of a multiethnic cohort of normal older adults had a mild detectable tremor on testing; in one third of the cases, the tremor worsened with activity.²

Many patients feel that tremors are a part of aging and fail to report them to their physicians, which denies them adequate treatment. Under the current health care system, most patients with tremors are likely to initially present to their primary care physician. A study by Marttila et al³ showed that 25% of the patients diagnosed by nonneurologists to have Parkinson's disease actually had essential tremor. This illustrates the importance of clinicians recognizing tremors and their underlying causes in order to manage them successfully.

The 3 most common forms of tremor in the elderly population are essential tremor, parkinsonian tremor, and cerebellar tremor. This article reviews the clinical characteristics, etiology and pathogenesis, and treatment of these types of tremor. The clinical features and management of several other types of tremor are addressed, as well.

DIFFERENTIAL DIAGNOSIS FOR MOVEMENT DISORDERS

It is important to differentiate tremor from other involuntary movements, including choreiform movements, athetosis, dystonia, myoclonic movements, and tics. Choreiform movements are involuntary and rapid but jerky in nature, rather than rhythmic. They are prominent in the face and distal extremities and give the patient a fidgety appearance. Chorea presenting in elderly patients is generally either senile chorea

(which is not an inherited disorder) or, rarely, chorea following a cerebrovascular accident. Although Sydenham's chorea and Huntington's chorea are rare in older persons, Huntington's chorea always must be ruled out.⁴

Athetosis constitutes slow writhing movements of the arms and legs that commonly occur on movement. It may be associated with hemiplegia and often occurs in children secondary to birth trauma.

Dystonic movements are slow, prolonged movements of either the trunk muscles or a distal muscle group. They may be associated with tremors of the affected part (dystonic tremor). Examples of dystonia include spasmodic torticollis involving the neck muscles, writer's cramp due to spasm of the shoulder muscles, and blepharospasm, a form of dystonia that consists of involuntary, repeated eye closure and is seen in elderly women. Blepharospasm is worsened by stress and can be confused with early Parkinson's disease. Patients with blepharospasm may also have twitching of oral or facial muscles, which is known as Meige's syndrome.⁴

Myoclonic jerks are sudden, brief, and shock-like and lack the rhythmicity of tremors. In adults and the elderly, myoclonus is rare and occurs in association with Creutzfeldt-Jakob disease, uremia, hypomagnesemia and liver failure.⁴ Tics are brief contractions of a muscle, which may be confused with tremors due to their repetitive nature. Tics commonly occur in children and can produce features such as sniffing, grunting, pouting, and grimacing.

CLINICAL CLASSIFICATION OF TREMOR

Tremors may be classified based on their clinical features (**Figure 1**). Most tremors can be characterized as either a resting tremor or an action tremor. Resting tremor occurs in a limb or body part that is completely supported against gravity with absolutely no contraction

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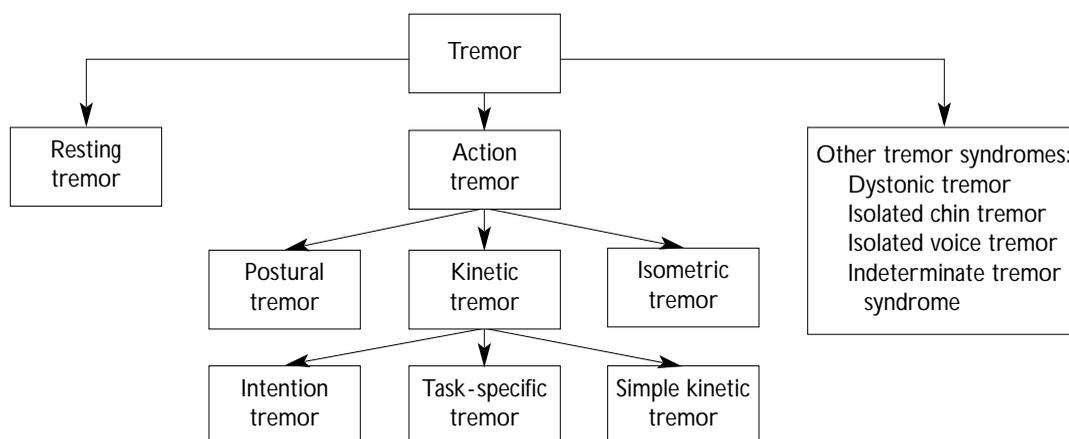


Figure 1. Clinical classification of tremor. (Data from Habib-ur-Rehman. Diagnosis and management of tremor. Arch Intern Med 2000;160:2438–44.)

Table 1. Causes of Tremors in the Elderly

Action Tremor	Resting Tremor
Parkinsonism	Parkinsonism
Essential tremor	Alcohol withdrawal
Cerebellar disease	Essential tremor*
Holmes' tremor	Neurosyphilis
Physiologic tremor	
Drugs (except those causing parkinsonism)	

*Although essential tremor is predominantly an action tremor, rarely, in advanced conditions, it may have a resting component. (Elble RJ. Diagnostic criteria for essential tremor and differential diagnosis. Neurology 2000;54[11 Suppl 4]:S2–6.)

Data from Patten JP, editor. Neurological differential diagnosis. 2nd ed. London: Springer Publication;1998.

of the skeletal muscle.⁵ If the limb is not supported, its muscles will be expending energy to maintain position; the tremor present in such a condition is an action tremor. For example, essential tremor is a type of action tremor that may be mistaken for a resting tremor if the affected limb is not completely at rest during examination. Resting tremor is best observed when the patient is distracted. In Parkinson's disease, the physician may observe mild tremor in the arm contralateral to the one he or she has asked the patient to swing, even if the patient is unaware of the tremor.⁴

Action tremor includes postural, kinetic, and isometric tremors.⁶ Postural tremor occurs when a body part is voluntarily held motionless against gravity—for example, pointing at an object or protruding the

tongue.⁷ Kinetic tremor occurs during voluntary movement of a body part. An intention tremor is a kinetic tremor that worsens during the completion of a visually guided, goal-directed movement (eg, pouring tea into a cup).⁶ Task-specific tremor occurs during complex tasks such as writing or playing a musical instrument. Simple kinetic tremor occurs with acts such as a simple turning of the wrist or foot. Isometric tremor occurs when a voluntary muscle contraction is opposed by a stationary force. It can be elicited by having the subject make a fist or push against a wall or other stationary object.

EVALUATION OF THE PATIENT WITH TREMOR

A wide range of disorders cause resting and action tremor (**Table 1**), and some can manifest both types of tremor activity. As laboratory tests are not available for the diagnosis of many common causes of tremor, a detailed physical examination is a physician's best diagnostic tool. Information regarding the patient's current and past medical history, as well as any family history of tremors, should be obtained. All medications used currently and in the past must be listed. A detailed neurologic examination to evaluate motor and sensory nervous systems, the extrapyramidal system, and cerebellar function is required.

Careful observation during the performance of each neurologic test can yield invaluable information. For example, during a finger-to-nose test, a cerebellar action tremor manifests during the terminal part of the test, just before the finger touches the nose. In contrast, a parkinsonian tremor may either disappear with the onset of finger-to-nose testing or may remain constant throughout the range of motion.

Table 2. Comparison of Essential Tremor and Parkinsonian Tremor

Characteristic	Essential Tremor	Parkinsonian Tremor
Type of tremor	Action, usually symmetrical	Rest and action, initially unilateral
Agents or states that improve the tremor	Alcohol, relaxation; voluntary suppression is possible	Sleep; voluntary suppression is not possible
Agents or states that worsen the tremor	Excitement, anger, fatigue, fear, caffeine, nicotine, β -agonists	Emotional stress, excitement, fatigue
Other findings	Occasional cogwheel rigidity	Bradykinesia, cogwheel rigidity, gait disturbance, depression, cognitive decline
Head involvement	Titubation commonly present	Titubation is usually absent; chin, jaw, and lip tremor may be present
Voice involvement	Tremulous voice	Low-volume voice

CLINICAL FEATURES OF DISORDERS ASSOCIATED WITH TREMOR

Essential Tremor

Essential tremor is the most common cause of tremor in elderly patients. It has a bimodal peak of onset in the second and sixth decades of life.⁷ Also known as senile tremor, it constitutes a mixture of postural and kinetic tremors. It is generally bilateral and is gradually progressive. It affects upper limbs in 95% of cases, the head in 34%, lower limbs in 20%, the voice in 12%, the face in 5%, and the trunk in 5%.⁷ A considerable number of patients with mild essential tremor (up to 50%)⁸ may be unaware of their tremor, and it does not come to the attention of their physician. Some patients may present with only isolated head tremor.

Although benign, essential tremor in its severe form can cause functional and social impairment, titubation, and a tremulous voice. Severe essential tremor may be accompanied by cogwheel rigidity. If the postural form of essential tremor is mistakenly characterized as resting tremor, the patient's condition may be wrongly diagnosed as Parkinson's disease, especially if cogwheel rigidity is present. However, in essential tremor, muscle tone and strength are normal. The clinical features of essential tremor and parkinsonian tremor are compared in **Table 2**.

Essential tremor has been observed to be precipitated and alleviated by several factors (Table 2). It improves with alcohol consumption but is generally not suspected to cause alcoholism in patients.⁵ Typically, there is an absence of signs of other neurologic disorders, although essential tremor may coexist with Parkinson's disease.

Etiology and pathogenesis. The etiology and pathophysiology of essential tremor have not been elucidated. At least 50% of patients with essential tremor have a positive family history. The familial form is an autosomal dominant disease with variable penetrance.¹ Autopsies of persons who had essential tremor revealed no histologic abnormalities.⁹ Routine computed tomography and magnetic resonance imaging scans are typically normal.¹ It has been proposed that enhanced oscillations in the olivocerebellar to rubrothalamic pathways are the cause of essential tremor.⁹ Increased glucose metabolism in the olivary nuclei and increased blood flow in the red nucleus, cerebellum, and thalamus have been observed bilaterally on positron emission tomography of patients with essential tremor.¹⁰ In animal models, a tremor similar to essential tremor was produced on stimulation of olivary nuclei with the alkaloid harmaline and serotonergic drugs,¹¹ lending further support to the involvement of this site in the genesis of tremor.

Cerebellar Tremor

Cerebellar tremor is a pure action tremor that is absent at rest. On examination, a combination of terminal intention tremor and some postural tremor can be seen. Dysmetria (ie, difficulty alighting on the target during voluntary movement) also characterizes cerebellar lesions. This manifests in the finger-to-nose test as "past-pointing." Other features include ataxia (uncoordinated or inaccurate movement), nystagmus, muscular hypotonia, decreased reflexes, and dysidiadochokinesia (ie, an inability to perform rapid alternating movements). Titubation (head nodding) may be present when the patient is upright but not on supination.¹²

Involvement of the laryngeal muscles leads to an uncoordinated voice, but unlike in parkinsonism, the voice is of normal volume.

Etiology and pathogenesis. The cerebellum receives input from the motor cortex regarding the next intended movement of the limbs and the trunk.⁴ The cerebellum also receives sensory information from the peripheral nerves regarding limb position and movement. Under normal circumstances, the cerebellum uses the information from these sources to anticipate and prevent the occurrence of movement errors, such as tremors.¹³ Vascular accidents, malignancy, and chronic alcohol or drug abuse⁴ may cause a lesion in the deep cerebellar nuclei (ie, the dentate, globose, or emboliform nuclei) or in their connections to the contralateral thalamus via the brachium conjunctivum. Such a lesion disrupts the sensorimotor feedback loop between the limb and the motor cortex, which is oscillatory in nature, and tremor results.

Parkinsonism

Parkinsonism is a movement disorder that can lead to severe physical and emotional disability. Although tremor is a hallmark of parkinsonism, it is not the cause of physical limitation in patients with the disease. Rather, functionality is severely limited by the bradykinesia, rigidity, and postural instability that are also characteristic of the disease.

Both resting and action tremor (kinetic type) can be present in this disease, and it is commonly unilateral in the early stages (Table 2). In Parkinson's disease of elderly onset (idiopathic Parkinson's disease), resting tremor may be an uncommon initial symptom.¹⁴ The same may hold true for drug-induced parkinsonism (DIP).⁴ Common causes of DIP include neuroleptic agents, calcium channel blockers, valproic acid, tacrine, bethanechol chloride, reserpine, and methyl dopa.¹⁵ Lower-potency neuroleptics or novel neuroleptics (eg, clozapine) that lack prominent striatal receptor blockade are less likely to cause DIP.¹⁶ Clinically, DIP cannot be distinguished from idiopathic Parkinson's disease; however, DIP develops subacutely within the first weeks of drug introduction or an increase in drug dosage.

Tremor may be a simple oscillatory movement or it may be compound, such as a pill-rolling movement. The tremor affects the distal extremities (especially the hands) and the facial, jaw, and tongue muscles. Anxiety, emotional upset, and fatigue exacerbate the tremor, and it disappears during sleep. Cogwheel rigidity may be present and the voice volume is low.

Monosymptomatic resting tremor is a predominant resting tremor (occasionally with a postural compo-

nent) that occurs in the absence of other parkinsonian features such as bradykinesia and rigidity.^{5,6} It must be of 2-year duration to fit the diagnostic criteria and may be a form of Parkinson's disease.

Etiology and pathogenesis. The anatomy of the structures involved in production of parkinsonian tremor is briefly described.¹⁷ Subcortical grey matter is grouped into nuclei known as *basal ganglia*, which include the striatum, globus pallidus, subthalamic nucleus, and substantia nigra. Nerve cells in the substantia nigra pars compacta of the midbrain produce dopamine. Dopamine is transported along the axons to the caudate nucleus and the putamen, which collectively form the striatum. Dopamine helps modulate the inhibitory output of the striatum to the globus pallidus interna and substantia nigra pars reticulata. These 2 nuclei, in turn, inhibit certain thalamic nuclei that have a stimulatory influence on the cerebral cortex.

In patients with Parkinson's disease, dopamine is depleted as a result of the death of dopaminergic cells in the substantia nigra. In patients with neuroleptic drug-induced parkinsonism, dopaminergic D₂ receptor blockade results in decreased dopamine activity. Both conditions can lead to disinhibition of the globus pallidum, causing suppression of the thalamic nuclei, which results in reduced excitation of the motor cortex. Because the motor cortex depends on the basal ganglia to facilitate movements generated by the cortex, the outcome of this depletion or underactivity of dopamine is a movement disorder.

This pathway does not explain the origin of the rest tremor seen in parkinsonism. In Parkinson's disease, it is known that the death of nigrostriatal neurons leads to abnormal intermittent oscillations in the neurons of the motor cortex, basal ganglia, and thalamus and that these abnormal oscillations can produce tremor.⁹ The exact cause of the oscillations is unclear, however. The cerebellum is involved in control of the parkinsonian rest tremor.¹⁸

Drug-Induced Tremors

Drugs are associated with other types of tremor besides DIP. Long-term use of antipsychotic agents can also cause a postural tremor called tardive tremor. It is a low frequency murmur that is usually postural but may occur at rest or with movement.⁶ Even short-term use of intravenous high-dose haloperidol (> 240 mg) has been associated with tremors, rigidity and dyskinesia.¹⁹ Sympathomimetic agents, cyclosporine, lithium, and tricyclic antidepressants are known to enhance physiologic tremor.¹⁵ Lithium overdose produces ataxia, tremor,

confusion, cardiac arrhythmias, gastrointestinal distress, muscle twitching, and fasciculations. It can be diagnosed by measuring serum levels of lithium.²⁰

Tremors are one of the common clinical features of serotonin syndrome, which is most commonly caused by interaction between serotonergic agents and monoamine oxidase inhibitors. Symptoms resolve after the discontinuation of the serotonergic drugs.²¹

Barbiturate withdrawal can produce anxiety, restlessness, delirium, seizures, and tremor.²² Abstinence from ethanol can produce similar symptoms within the first few days; when severe, this can lead to delirium tremens, which consists of gross tremor, altered sensorium, fever, and tachycardia. Benzodiazepine withdrawal leads to anxiety, nausea, vomiting, tremor, hallucinations, and seizures. Tremors may also be caused by hallucinogen use and may accompany phencyclidine (PCP) withdrawal.²²

Chronic exposure to metallic mercury produces an intention tremor that can be accompanied by erethism (memory loss, excitability, insomnia, and delirium). This may be observed in workers in the felt-hat industry and sometimes in dentists.²³ Acute exposure to mercury or its vapors can also lead to tremors.

Physiologic Tremor

Normal individuals can manifest an action tremor, especially while fatigued or experiencing emotions such as fear, anxiety, or stress.⁶ Physiologic tremor can also result from medical conditions such as hypothermia, hypoglycemia, pheochromocytoma, thyrotoxicosis, and alcohol withdrawal.⁵

A characteristic feature of physiologic tremor is the disappearance of the tremor on removal of the precipitating agent or resolution of the underlying state. In a patient with alcoholism, physiologic tremor should be differentiated from asterixis (repetitive partial flexion of the wrists during sustained wrist extension). Asterixis is a sign of metabolic encephalopathy and accompanies other signs of liver failure in these patients.

Dystonic Tremor

Patients with dystonia may have a localized tremor that can occur in a part affected by dystonia (eg, tremulous writer's cramp) or in an unaffected part (eg, postural arm tremor in a patient with cervical dystonia). This tremor is irregular in nature and is accompanied by abnormal posturing. When it involves the head, dystonic tremor may be confused with titubation (senile tremor). It may be distinguished from titubation by having the patient turn the head opposite to the direction of the dystonic pull; worsening of the tremor indicates

dystonic tremor rather than titubation.¹ If the voice is affected, it is either crackling or breath-like in nature.

Psychogenic Tremor

Also called hysterical or functional tremor, psychogenic tremor is characterized by sudden onset and remission⁵ and changes in severity over time. A history of mental illness and unrelated neurologic signs may be present. Tremor amplitude typically decreases with distraction.

Other Tremors

Holmes' tremor (red nuclear tremor) presents as a terminal intention tremor or resting tremor that is violent in nature. Even a slight attempt to move a limb can lead to severe jerking movements that may lead to injury. Voice involvement can lead to inarticulate speech.⁴

Task-specific tremor is a type of action tremor associated with certain tasks, such as writer's tremor.⁵ Palatal tremor includes symptomatic palatal tremor, which manifests as rhythmic movements of the soft palate and is usually associated with a brainstem or cerebellar lesion, and essential palatal tremor, which manifests as rhythmic ear clicks and is not associated with the presence of a brain lesion.²⁴ Certain neuropathies, including chronic inflammatory demyelinating neuropathy, Guillain-Barré syndrome, and dysgammaglobulinemic neuropathy, are also associated with tremor.⁶ The term "indeterminate tremor syndrome" is assigned to a condition in which the patient has essential tremor along with certain neurologic signs that are insufficient to make a diagnosis of any neurologic syndrome.⁶

PHARMACOLOGIC MANAGEMENT OF TREMOR

Control of tremor is not always essential. Many patients with mild essential tremor do not feel the need to bring the tremor to the attention of their physicians, let alone get treatment for it.

Essential Tremor

β -Adrenergic blockers (eg, propranolol) and the anticonvulsive agent primidone are the mainstays of treatment for essential tremor (**Table 3**). β -Blockers can cause confusion and dizziness in elderly patients. Furthermore, they must be used cautiously in patients with asthma, heart failure, or diabetes. When essential tremor is induced by a drug that is medically necessary, β -blockers may be used to suppress the tremor.¹⁵ Primidone has a response rate of up to 75%²⁵ in patients with tremor and can be used instead of β -adrenergic blockers.

Table 3. Pharmacologic Agents for Treatment of Essential Tremor

β-Adrenergic blockers
Metoprolol
Propranolol
Benzodiazepines
Clonazepam
Diazepam
Botulinum toxin injection
Calcium channel blockers
Gabapentin
Primidone

Data from Habib-ur-Rehman. Diagnosis and management of tremor. Arch Intern Med 2000;160:2438–44.

Gabapentin is well tolerated by older patients.¹ Benzodiazepines may be used to control the anxiety that enhances tremor. Severe drowsiness and confusion may occur in the elderly with their use. Calcium channel blockers are not very effective for the treatment of tremor, but they may be tried if other agents fail.¹

The alternative agents acetazolamide²⁶ and metazolamide²⁷ have not been shown to significantly improve essential tremor. A double-blind placebo-controlled study showed no benefits with acetazolamide but found alprazolam effective in essential tremor treatment.²⁶

Patients may use alcohol to self-treat essential tremor; however, this practice should be discouraged, and the patient should be monitored because of the potential risk of alcoholism. Long-term use of alcohol is not beneficial because of the development of tolerance. In older patients, alcohol use can lead to falls, drowsiness, and drug interactions.

Botulinum toxin is a new treatment mode that is currently under investigation. A randomized, double-blind trial evaluated the use of botulinum toxin type A in 133 patients.²⁸ Although postural hand tremor was significantly reduced, kinetic hand tremor showed no long-term improvement. Improved functional ability was not observed, probably owing to the dose-dependent hand weakness caused by the toxin itself. Botulinum toxin type A has produced a modest response in patients with head and voice tremor but carries the risk of producing dysphasia.¹

Strength training programs have been shown to decrease the magnitude of essential tremor but did not improve functional ability.²⁹

Parkinsonian Tremor

In patients with early Parkinson's disease in whom

Table 4. Pharmacologic Agents for Treatment of Parkinsonian Tremor

Amantadine
Anticholinergic agents
Benztropine mesylate
Biperiden
Trihexyphenidyl
Bromocriptine mesylate
Levodopa-carbidopa

Data from Evidente VG. Understanding essential tremor. Differential diagnosis and options for treatment. Postgrad Med 2000;108:138–40, 143–6, 149.

tremor is the only manifestation, there is no functional limitation; the use of medication is thus essential only when other symptoms are present.¹² All anti-parkinsonian drugs must be started at a small dose and gradually increased in strength and frequency. Anticholinergic agents constitute a reasonable first line of treatment in Parkinson's disease (**Table 4**).¹² Potential adverse effects include urinary retention, confusion, and hallucinations; they also can worsen glaucoma in older patients. When anticholinergic agents are inadequate, amantadine can be added. If maximum tolerable dosages of both drugs are unsuccessful in treating the patient, they are discontinued and levodopa-carbidopa is begun. One of the problems of using levodopa-carbidopa is the fluctuation between high and low blood levels of the drugs, leading to on-off phenomenon and dyskinesia. To avoid this, smaller doses can be given at regular intervals. Bromocriptine can help in the management of on-off phenomenon and decrease the dose of levodopa. Recent studies have found apomorphine and clozapine useful in the treatment of parkinsonian tremor.^{30,31}

Drug-Related Tremor

While most drug-induced tremors disappear after discontinuation of the drug, drug-induced parkinsonian tremor may persist for years after discontinuation.¹⁵ Treatment of drug-induced parkinsonian tremor is similar to that of Parkinson's disease.

Withdrawal symptoms produced by sedatives such as barbiturates and benzodiazepines are treated by substituting related shorter-acting drugs given in tapering doses. Alcohol withdrawal symptoms can be treated with benzodiazepines. Delirium tremens is best managed by adequate sedation of affected patients and supportive treatment.²² Antipsychotic agents should be avoided in these patients because these agents lower the seizure

threshold. Hallucinogen and PCP users can be managed with supportive care and benzodiazepines. Frank psychosis can occur with PCP use and requires treatment with antipsychotic agents.

Acute inorganic mercury exposure is treated with dimercaprol.²³ Chronic mercury poisoning is treated with *N*-acetyl penicillamine.²³ Lithium intoxication is treated with gastrointestinal decontamination, intravenous hydration, diuresis, urine alkalinization, and supportive treatment. Hemodialysis may be required for severe cases.²⁰

Treatment of Other Tremors

Cerebellar tremors are extremely difficult to treat. Some progress has been reported with clonazepam and ondansetron.^{32,33} Holmes' tremor has shown some response to levodopa.³⁴ Physiologic tremor is best treated by removal of the causative agent or resolution of the precipitating state. Small doses of β -blockers may also help.⁶ Dystonic head tremors respond to botulinum toxin injections.³⁵ Propranolol, primidone, alcohol, and botulinum toxin may be beneficial in the treatment of task-specific tremor.²⁵ Neuropathic tremor does not always improve with resolution of the underlying neuropathy and may respond to propranolol.⁵ Palatal tremors occasionally respond to local botulinum toxin injection.⁵

SURGICAL MANAGEMENT OF TREMORS

Surgical treatment is considered only for severe disabling tremors after medical treatment has failed. Two procedures used in the treatment of Parkinson's disease and essential tremor are thalamotomy and thalamic stimulation targeting the ventralis intermedialis nucleus of the thalamus. Thalamotomy is performed via thermocoagulation. Thalamic stimulation is performed by local implantation of a pulse generator.⁶ These procedures, when performed on one side of the brain, improve symptoms on the contralateral side. Thalamic stimulation has the advantage of being feasible bilaterally; however, a bilateral thalamotomy may lead to bulbar neurologic deficits (ie, dysphagia and dysarthria) and cognitive neurologic deficits, as well as visual field defects and hemiparesis.⁶

These procedures improve tremor in 80% to 90% of patients with Parkinson's disease. Although thalamotomy improves essential tremor in 69% to 93% of patients,³⁶ thalamic stimulation is preferred because of the bilateral nature of the disease. One-year follow-up of patients with disabling essential tremor treated with bilateral thalamic stimulation showed reduced tremor and better functional ability.³⁷

Thalamic surgery may be beneficial in Holmes'

tremor but is not usually performed because of the risks involved and the possibility of spontaneous remission.⁵

CONCLUSION

Because of their frailty and coexistent medical problems, geriatric patients are at high risk of morbidity and mortality when afflicted by a movement disorder such as tremor. Elderly patients may also be at risk of developing multiple forms of tremor (eg, resting and action). Because the presence of tremors is often considered a part of aging, many patients may not benefit from the treatment opportunities available.

In the evaluation of tremor, particular attention must be paid when the tremor is sudden in onset, occurs in uncommon sites (eg, the lower extremities, tongue, or chin), is preceded by a neurologic event, or is associated with other signs of neurologic dysfunction. Careful assessment and the judicious use of medication, when needed, will help these patients retain their physical independence longer. **HP**

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(continued on page 49)

(from page 37)

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