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**Overview of tremor**

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**INTRODUCTION** — Tremor is defined as a rhythmic and oscillatory movement of a body part with a relatively constant frequency and variable amplitude. It is caused by either alternating or synchronous contractions of antagonistic muscles. Tremor is the most common of all movement disorders, occurring from time to time in most normal individuals in the form of exaggerated physiologic tremor [[1](http://www.uptodate.com/contents/overview-of-tremor/abstract/1)].

This topic will cover the classification, clinical features, diagnostic evaluation, and treatment of tremor. The treatment of essential tremor (ET) is discussed separately. (See ["Pharmacologic treatment of essential tremor"](http://www.uptodate.com/contents/pharmacologic-treatment-of-essential-tremor?source=see_link) and ["Surgical treatment of essential tremor"](http://www.uptodate.com/contents/surgical-treatment-of-essential-tremor?source=see_link).)

**CLASSIFICATION** — Tremors may be broadly classified into static and action tremors ([table 1](http://www.uptodate.com/contents/image?imageKey=PC%2F68810&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link)). Static tremors may be further divided into those occurring at rest (resting tremor) and those occurring with the head and limbs held in a fixed posture (postural tremor) ([table 2](http://www.uptodate.com/contents/image?imageKey=NEURO%2F72091&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link) and [table 3](http://www.uptodate.com/contents/image?imageKey=PC%2F77497&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link)). Action tremors remain unchanged during the course of a voluntary movement, while intention tremors increase during the course of goal-directed movement. The term kinetic tremor has been used to designate action and intention tremor.

**RESTING TREMOR** — Parkinson disease (PD) and other parkinsonian syndromes are the most common causes of resting tremor. This tremor is evident with the affected body part supported and completely at rest and temporarily dampens or disappears during voluntary activity. Resting tremors usually fluctuate in amplitude and may appear and disappear depending upon the degree of patient repose, whether the patient feels he or she is under observation, and other unknown factors.

Resting tremor is typically less disabling than postural, action, or intention tremors because of its absence during voluntary activity. However, the tremor may quickly reappear as soon as the body part assumes a new resting posture and may therefore interfere with the use of eating utensils, handwriting, typing, and other purposeful postures or movements. In such cases, resting tremor is more of a handicap or disability than pure resting tremor, which is of greater cosmetic concern.

In the examination, resting tremor is usually activated while carrying out repetitive movements of the opposite limb or during walking.

**Idiopathic Parkinson disease** — The resting tremor in PD typically appears first in one hand. It later may or may not spread to involve the ipsilateral leg and/or the contralateral upper limb. Leg or foot tremor is more commonly due to PD than to essential tremor ET. The face, lips, and jaw may be involved but, unlike ET or cerebellar disease, PD only occasionally produces tremor of the entire head. (See ["Clinical manifestations of Parkinson disease", section on 'Tremor'](http://www.uptodate.com/contents/clinical-manifestations-of-parkinson-disease?source=see_link&anchor=H3#H3).)

When the tremor is limited to distal muscles of the hand it may produce a characteristic "pill-rolling" tremor, with a frequency of 4 to 6 Hz. With increasing severity, the tremor may become more continuous, larger in amplitude, and more proximal in distribution, but the frequency remains constant.

**Tremor-dominant Parkinson disease** — A low amplitude resting tremor of the hand or jaw, unaccompanied by other manifestations of parkinsonism, sometimes occurs as an isolated finding and may not progress to more generalized PD. However, tremor is such a common first sign of PD that it just as often leads the way to progressive and more disabling symptoms such as generalized bradykinesia, gait disturbance, and postural instability.

Patients can go years into their disease with tremor as the only or the most prominent sign of their illness. When this happens, this is referred to as tremor-dominant PD, although it is most reliably applied in retrospect when the relatively benign course has already been charted.

Other disorders associated with resting tremor include Wilson's disease, non-Wilsonian hepatocerebral degeneration, and midbrain injury due to stroke, trauma, or demyelinating disease. Resting tremor may also occur as a "spillover" phenomenon in a variety of disorders in which very severe postural-action tremors predominate, such as Wilson's disease, severe forms of ET [[2](http://www.uptodate.com/contents/overview-of-tremor/abstract/2)], and other cerebellar or extrapyramidal syndromes.

**SWEDDs** — The term SWEDDs (Scans Without Evidence of Dopaminergic Deficit) has been used designate patients with relatively isolated upper extremity resting and postural tremor resembling early PD who failed to evolve over time into more generalized PD [[3](http://www.uptodate.com/contents/overview-of-tremor/abstract/3)]. Unlike patients with PD, these individuals lacked evidence for nigrostriatal dopamine deficiency on dopamine transporter imaging (DaTSCAN). Patients with SWEDDs sometimes exhibit reduced arm swing and mild focal dystonia on the affected side, and may have jaw or head tremor or facial hypomimia, but no signs of parkinsonian akinesia [[3](http://www.uptodate.com/contents/overview-of-tremor/abstract/3)]. They are therefore to be distinguished from individuals with tremor-dominant PD.

**Rubral tremor** — Rubral tremor, sometimes called Holmes tremor, occurs with midbrain injury and is probably due to combined lesions of the superior cerebellar peduncle, substantia nigra, and red nucleus that interrupt the outflow pathway from the cerebellum to the motor thalamus. This tremor is present at rest but tends to have a slower frequency (3 to 5 Hz) than typical Parkinson tremor (5 to 7 Hz). It also persists unchanged or increases with postural changes or goal-directed activity. It may produce a combination of rest, postural, action, and intention (kinetic) tremors.

**POSTURAL AND ACTION TREMORS** — Postural and action tremors comprise the largest group of tremors. They are elicited during examination under two circumstances: with the arms suspended against gravity in a fixed posture; and during the course of goal-directed activity.

**Physiologic tremor** — Normal individuals have a very low amplitude, high frequency physiologic tremor of approximately 10 to 12 Hz that is not visible under ordinary circumstances. Many factors can enhance the tremor to the point of detection, most often by increasing sympathetic activity [[1](http://www.uptodate.com/contents/overview-of-tremor/abstract/1)].

* Common drugs that increase adrenergic activity include beta-adrenergic agonists such as [terbutaline](http://www.uptodate.com/contents/terbutaline-drug-information?source=see_link), [isoproterenol](http://www.uptodate.com/contents/isoproterenol-drug-information?source=see_link), epinephrine, amphetamines, selective serotonin reuptake inhibitors (SSRIs) and tricyclic antidepressants, levodopa, nicotine, and xanthines such as [theophylline](http://www.uptodate.com/contents/theophylline-drug-information?source=see_link) and caffeine.
* Anxiety, excitement, fright, muscle fatigue, hypoglycemia, alcohol and opioid withdrawal, thyrotoxicosis, fever, and pheochromocytoma also enhance adrenergic activity. (See ["Neurologic manifestations of hyperthyroidism and Graves' disease", section on 'Tremor'](http://www.uptodate.com/contents/neurologic-manifestations-of-hyperthyroidism-and-graves-disease?source=see_link&anchor=H8#H8).)
* Miscellaneous drugs and toxins that increase physiological tremor by other uncertain means include [lithium](http://www.uptodate.com/contents/lithium-drug-information?source=see_link), antidepressants, corticosteroids, sodium [valproate](http://www.uptodate.com/contents/valproate-drug-information?source=see_link), bromides, mercury, lead, and arsenic.

Enhancement of physiologic tremor is the most common cause of postural and action tremors. Thus, a medical rather than primary neurologic cause for postural-action tremor should be considered in most cases.

**Essential tremor** — Essential tremor (ET) is the most common neurologic disorder that causes postural or action tremor, with an estimated prevalence worldwide of up to 5 percent of the population [[4-6](http://www.uptodate.com/contents/overview-of-tremor/abstract/4-6)]. The incidence of ET increases with age, although it often affects young individuals, especially when it is familial.

ET is referred to as familial tremor when there is a family history (approximately 50 percent of cases have an autosomal dominant pattern of inheritance) and benign essential tremor when it is sporadic. The use of "benign" as a modifier for ET has historically been employed to distinguish it from Parkinson disease (PD); it is best omitted, since the tremor can be severe and disabling [[7](http://www.uptodate.com/contents/overview-of-tremor/abstract/7)]. While prospective data are limited, ET may be associated with an increased risk for developing Parkinson disease [[8](http://www.uptodate.com/contents/overview-of-tremor/abstract/8)].

ET appears to be genetically complex [[9](http://www.uptodate.com/contents/overview-of-tremor/abstract/9)]:

* Mutations in the FUS gene have been identified by exome sequencing as the cause of ET in a multigenerational family with hereditary ET [[10](http://www.uptodate.com/contents/overview-of-tremor/abstract/10)]. Different FUS mutations cause a form of familial amyotrophic lateral sclerosis. (See ["Familial amyotrophic lateral sclerosis", section on 'ALS6 (FUS gene)'](http://www.uptodate.com/contents/familial-amyotrophic-lateral-sclerosis?source=see_link&anchor=H6#H6).)
* Dominantly inherited ET has been tentatively linked to genetic loci on chromosomes 2p, 3q13, and 6p23 [[11-14](http://www.uptodate.com/contents/overview-of-tremor/abstract/11-14)]. Other families with ET are not linked to any of these loci [[15](http://www.uptodate.com/contents/overview-of-tremor/abstract/15)].
* First-degree relatives of patients with ET have an increased risk of developing the disorder, particularly when the proband develops ET at an early age [[16](http://www.uptodate.com/contents/overview-of-tremor/abstract/16)]. The lack of 100 percent concordance among monozygotic twins suggests that environmental factors are involved in the pathogenesis of the disease [[17](http://www.uptodate.com/contents/overview-of-tremor/abstract/17)].
* Genome-wide association studies in Icelandic and European subjects with ET have identified sequence variations in the LINGO1 gene [[18-20](http://www.uptodate.com/contents/overview-of-tremor/abstract/18-20)], which appears to play a role in oligodendrocyte differentiation and axonal myelination. Findings from another genome-wide association study of European subjects suggest that ET is associated with polymorphisms in the glial glutamate transporter gene (SLC1A2) [[21](http://www.uptodate.com/contents/overview-of-tremor/abstract/21)].

Accumulating evidence suggests that the neuropathology of ET is localized in the brainstem (locus ceruleus) and cerebellum [[22](http://www.uptodate.com/contents/overview-of-tremor/abstract/22)]:

* One of the largest published studies analyzed postmortem findings from 33 patients with ET compared with 21 controls [[23](http://www.uptodate.com/contents/overview-of-tremor/abstract/23)]. Brainstem Lewy bodies were found in eight (24 percent) ET cases; in the remaining 25 (76 percent) ET cases with no Lewy bodies, degenerative changes in the cerebellum (Purkinje cell loss and axonal swelling) were significantly more frequent than in controls.
* In another case-control study, depletion of pigmented neurons in the locus ceruleus and cerebellar gliosis were significantly more common in 21 subjects with ET than in 21 controls [[24](http://www.uptodate.com/contents/overview-of-tremor/abstract/24)].
* A study using diffusion tensor MRI has shown changes consistent with neurodegeneration in the dentate nucleus and superior cerebellar peduncle of patients with familial ET [[25](http://www.uptodate.com/contents/overview-of-tremor/abstract/25)].
* In contrast, a neuropathology study that compared findings from seven patients with ET, six patients with PD and two normal control brains found a similar age related reduction in Purkinje cell numbers in all three groups and no evidence of Lewy body pathology [[26](http://www.uptodate.com/contents/overview-of-tremor/abstract/26)].

The significance of these findings is currently uncertain, and further age controlled neuropathologic and neuroradiologic information is needed.

**Clinical features of ET** — ET is a heterogenous disorder with considerable variability in the character of the tremor, the circumstances in which it is exacerbated, and its association with other neurological deficits [[27,28](http://www.uptodate.com/contents/overview-of-tremor/abstract/27,28)]. It varies from a low amplitude, high frequency postural tremor of the hands to a much larger amplitude, postural and action tremor that is activated by particular postures and actions. ET most often affects the hands and arms and can be asymmetric. It can also affect the head, voice, chin, trunk, and legs.

The diagnostic criteria for ET are shown in the Table ([table 4](http://www.uptodate.com/contents/image?imageKey=NEURO%2F62078&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link)) [[29](http://www.uptodate.com/contents/overview-of-tremor/abstract/29)].

ET becomes immediately apparent in the arms when they are held outstretched; it typically increases at the very end of goal-directed movements such as drinking from a glass or finger-to-nose testing. Cerebellar outflow tremor should be considered when the tremor oscillations increase steadily before arriving at the target rather than at the termination of goal-directed activity, although a distinction between the two is often difficult.

Tremor in the legs is unusual in ET. Head tremor may be vertical ("yes-yes") or horizontal ("no-no") and is usually associated with hand or voice tremor. Head tremor rarely occurs in isolation in ET [[30](http://www.uptodate.com/contents/overview-of-tremor/abstract/30)]. When it does, the possibility of cervical dystonia with dystonic head tremor should be considered.

By definition, tremor should be the only neurologic manifestation of ET. However, in some severe cases, mild gait disorder and cerebellar signs may be present. In addition, preliminary studies suggest that very mild cognitive deficits with reduced performance on tests of memory and frontal executive function may be more common in patients with ET than age-matched controls [[31-34](http://www.uptodate.com/contents/overview-of-tremor/abstract/31-34)], and that ET may be associated with an increased risk of dementia [[35,36](http://www.uptodate.com/contents/overview-of-tremor/abstract/35,36)].

Some patients with ET develop enhanced physiologic tremor due to anxiety or other adrenergic mechanisms, thereby aggravating the underlying tremor. ET is typically relieved by small amounts of alcohol but, in contrast with physiologic tremor, is not usually aggravated by caffeine.

**Differential diagnosis of ET** — It is a common mistake to refer to any postural or action tremor as “essential tremor” since there is a broad differential diagnosis for tremors of this type. The most common differential diagnosis is with parkinsonian tremor ([table 5](http://www.uptodate.com/contents/image?imageKey=NEURO%2F60694&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link)). Differentiation from classical resting tremor should be straightforward; however, some patients with PD also have a postural-action tremor indistinguishable from ET [[37](http://www.uptodate.com/contents/overview-of-tremor/abstract/37)]. In fact, it is not unusual for patients with PD to present with a postural tremor shortly before they display other signs of PD. Likewise, patients with severe ET may have a rest component to their tremor. The presence of subtle bradykinesia or micrographia in early cases of parkinsonian postural tremor helps make the diagnosis, although these signs may not appear until later. Conversely, elderly patients with ET may have mild bradykinesia and limb rigidity as a nonspecific accompaniment of aging.

Head tremor is more likely to be a manifestation of ET, whereas tremor of the jaw or lips is more typically parkinsonian. Head tremor is also common in cervical dystonia (spasmodic torticollis) where it may be due either to a coincidence of the two disorders or to a manifestation of dystonic muscle spasm.

Ataxia, dysmetria, proximal distribution of the tremor, or gait disorder usually suggests a cerebellar disorder rather than ET. However, mild cerebellar signs are present in some severe familial tremors ([table 6](http://www.uptodate.com/contents/image?imageKey=NEURO%2F54735&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link)) [[38](http://www.uptodate.com/contents/overview-of-tremor/abstract/38)]. Voice tremor rarely occurs in isolation; when it does, it is important to differentiate it from spasmodic dysphonia. Essential voice tremor produces a quavering voice best identified by asking the patient to hold a steady note such as “ahhhh” or “eeeee” unaccompanied by the hoarseness, straining, and voice breaks characteristic of spasmodic dysphonia.

**Primary writing tremor** — Many action tremors are particularly severe during the act of writing. Tremor that occurs exclusively while writing, and not during other voluntary motor activities, is referred to as primary writing tremor [[39](http://www.uptodate.com/contents/overview-of-tremor/abstract/39)]. This tremor is limited to the hand and causes relatively large amplitude supination-pronation movements at a frequency of 5 to 6 Hz. The low frequency and large amplitude of the tremor, its frequent occurrence in writer's cramp or writer's dystonia, its relative resistance to [propranolol](http://www.uptodate.com/contents/propranolol-drug-information?source=see_link), and its occasional response to anticholinergic drugs suggest a closer relationship to dystonia than to ET.

**Orthostatic tremor** — Orthostatic tremor is limited to the legs and trunk, and occurs exclusively while standing [[40-43](http://www.uptodate.com/contents/overview-of-tremor/abstract/40-43)]. Both high and low frequency orthostatic tremors have been described; their relationship to ET is uncertain. In cases of high frequency tremor, movements of the legs may be so low in amplitude that they initially escape clinical detection. Orthostatic tremor is uniquely, but not always, responsive to treatment with [clonazepam](http://www.uptodate.com/contents/clonazepam-drug-information?source=see_link) (see ['Treatment'](http://www.uptodate.com/contents/overview-of-tremor?topicKey=NEURO%2F4895&elapsedTimeMs=8&source=search_result&searchTerm=tremor&selectedTitle=1%7E150&view=print&displayedView=full#H22) below).

**Cerebellar tremors** — Cerebellar tremors can be postural, action, or intention (kinetic). In severe cases, they can spill over to also occur at rest. Tremor frequency is typically low (3 to 4 Hz) and can be associated with ataxia and dysmetria.

Rubral tremor is caused by disturbances of cerebellothalamic projections (see ['Rubral tremor'](http://www.uptodate.com/contents/overview-of-tremor?topicKey=NEURO%2F4895&elapsedTimeMs=8&source=search_result&searchTerm=tremor&selectedTitle=1%7E150&view=print&displayedView=full#H6) above); it is usually present at rest and increases during postural fixation and voluntary activity. Titubation of the head and neck ("to and fro" movements) may be associated with cerebellar tremor; it is distinguished from essential head tremor by the presence of other cerebellar findings.

**NEUROPATHIC TREMOR** — Tremor is sometimes associated with large fiber peripheral neuropathy. This association is most commonly observed in hereditary neuropathies, during the recovery phase of some cases of Guillain-Barré syndrome, and in chronic inflammatory demyelinating polyneuropathy (CIDP). Muscle weakness and loss of proprioceptive or muscle spindle inputs may account for the tremor. The frequency and amplitude of neuropathic tremor may vary greatly when associated with a proprioceptive deficit.

**INTENTION TREMOR** — Intention tremor, also called kinetic tremor, is due to disturbances anywhere along the path of the cerebellar outflow projection system from the dentate nucleus of the cerebellum to the motor division (ventral lateral nucleus) of the thalamus. The most common causes are multiple sclerosis, midbrain trauma, and stroke. Heredodegenerative diseases of the cerebellum, severe forms of essential tremor (ET), Wilson's disease, hepatocerebral degeneration, and mercury poisoning may also produce this tremor.

The tremor typically increases in severity as the hand moves closer to its target, in contrast to postural and action tremors, which either remain constant throughout the range of motion or abruptly increase at terminal fixation. Intention tremors are usually very large in amplitude due to involvement of proximal muscles and are sometimes difficult to distinguish from severe cerebellar ataxia. The frequent association with ataxia, dysmetria, titubation, and other cerebellar signs serves to identify the cerebellar origin of intention tremor.

**PSYCHOGENIC TREMOR** — Useful criteria for the diagnosis of psychogenic tremor have been established [[44,45](http://www.uptodate.com/contents/overview-of-tremor/abstract/44,45)]. These are typically complex resting, postural, and action tremors with abrupt onset, a static course, changeable features, functional disability out of proportion to tremor magnitude, and resistance to treatment. Any body part may be involved, but, remarkably, the fingers are often spared with much of the upper limb tremor occurring at the wrist [[45](http://www.uptodate.com/contents/overview-of-tremor/abstract/45)]. Other features of psychogenic movement disorders are also often present, such as inconsistent display of symptoms and clinical features that are incongruous with known tremors.

Examination usually shows variable tremor frequency or tremor entrainment (ie, shift of tremor frequency to the speed of contralateral finger tapping), especially with distraction maneuvers such as repetitive tapping tasks with an uninvolved opposite hand or foot.

**EVALUATION** — The diagnostic approach to patients with tremor involves the history, physical examination, and selected laboratory studies. Action tremor is most common and, of these, essential tremor (ET) and enhanced physiologic tremor are the most frequent diagnoses [[46](http://www.uptodate.com/contents/overview-of-tremor/abstract/46)]. Patients with tremor due to other disorders such as hyperthyroidism, Parkinson disease (PD), dystonia, Wilson disease, or drug-induced tremors frequently have additional signs or symptoms that help point to the diagnosis, although this is not always the case. The criteria for ET and methods for distinguishing it from other tremors are shown in the tables ([table 1](http://www.uptodate.com/contents/image?imageKey=PC%2F68810&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link) and [table 2](http://www.uptodate.com/contents/image?imageKey=NEURO%2F72091&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link) and [table 3](http://www.uptodate.com/contents/image?imageKey=PC%2F77497&topicKey=NEURO%2F4895&rank=1%7E150&source=see_link)).

**History** — The history concerning the onset of tremor is usually straightforward, since it is a highly visible symptom that is readily evident to the patient and family members. Examination of previous handwriting samples may be useful in determining the precise time of onset. Precipitating, aggravating, or relieving factors such as caffeine, alcohol, medications, exercise, fatigue, or stress should be elicited; a complete list of all medications should be reviewed to exclude the possibility of enhanced physiologic tremor (see ['Physiologic tremor'](http://www.uptodate.com/contents/overview-of-tremor?topicKey=NEURO%2F4895&elapsedTimeMs=8&source=search_result&searchTerm=tremor&selectedTitle=1%7E150&view=print&displayedView=full#H8) above).

Family history in ET reflects an autosomal dominant pattern of inheritance in approximately half of all patients. Parkinsonian tremor is usually sporadic, but a family history of PD is present in approximately 15 percent of cases; the disease affects first-degree relatives (parents, siblings or offspring) in about half of the familial cases. Autosomal PD affecting three or more generations is rare.

**Examination** — Examination begins with observations of the tremor during the interview. Many patients with tremor are more symptomatic during the early part of the examination because of stress than after they become acclimated to the doctor-patient encounter. Patients should be observed sitting, lying down (with the affected body part fully supported), and walking. Horizontal or vertical head tremor is usually associated with tremor elsewhere. Isolated head tremor should raise the possibility of cervical dystonia or midline cerebellar syndromes. Localized face, jaw, and lip tremors are more commonly a manifestation of parkinsonism. Essential voice tremor is readily audible and may be further enhanced by having the patient hold a prolonged note.

Tremor in the arm is observed with the affected limb fully supported at rest, with the limb elevated against gravity, and during goal-directed movements. Most resting tremors cease with changes in limb posture but may reappear following repositioning to another fixed posture, referred to as re-emergent tremor. Parkinsonian tremor, by contrast with ET, is usually activated by repetitive movements of the opposite hand, during walking, and during mental distraction such as reciting the months of the year backwards.

Postural and action tremors are best elicited with the arms held outstretched; with the shoulders abducted, elbows flexed, and index fingers held an inch apart in front of the face; during finger-to-nose maneuvers; and while drinking or pouring from a paper cup. Writing and drawing may demonstrate the large, tremulous, angulated loops of ET or the micrographia of parkinsonism.

Tremor of the leg should be assessed with the limb at rest, during heel-to-shin testing, and while standing and walking. Leg tremor is more commonly due to parkinsonism than ET.

The gait is almost always normal in patients with ET, while it is characteristically narrow-based and shuffling in PD, and is wide-based and ataxic in cerebellar disorders. The gait may have histrionic qualities in patients with psychogenic tremor.

Tremor frequency should be estimated at rest and during postural and action maneuvers. Enhanced physiologic tremor is high in frequency (10 to 12 Hz), ET can be either low or high in frequency, and parkinsonian rest tremor is usually low in frequency (4 to 6 Hz). Orthostatic tremor is 15 to 20 Hz. Psychogenic tremors tend to vary in frequency as well as amplitude from moment to moment and either become more irregular or subside entirely when the patient is asked to carry out a complex, repetitive motor task with the contralateral limb. (See ['Psychogenic tremor'](http://www.uptodate.com/contents/overview-of-tremor?topicKey=NEURO%2F4895&elapsedTimeMs=8&source=search_result&searchTerm=tremor&selectedTitle=1%7E150&view=print&displayedView=full#H17) above.)

**Laboratory studies** — The routine laboratory evaluation of tremor should include tests of thyroid function, diagnostic studies to exclude Wilson's disease, and screening for heavy metal poisoning such as mercury or arsenic, if an environmental cause is suspected. Wilson disease should be suspected in anyone under age 40 who has tremor or other involuntary movement or posture [[1](http://www.uptodate.com/contents/overview-of-tremor/abstract/1)]. (See ["Wilson disease: Diagnostic tests"](http://www.uptodate.com/contents/wilson-disease-diagnostic-tests?source=see_link).) Hypoglycemia and pheochromocytoma may need to be ruled out in patients with enhanced physiologic tremor.

Brain imaging can be useful in patients suspected clinically of having a structural cause for tremor, such as Wilson disease, brain trauma, stroke, or mass lesion, but is otherwise usually not indicated.

Quantitative computerized analysis of tremor is available in some tertiary care facilities, but its ability to reliably distinguish between tremor types has not been established.

**TREATMENT** — The treatment of tremor depends upon the underlying cause.

**Enhanced physiologic tremor** — Enhanced physiologic tremor is best managed by reduction or removal of the responsible offending medication or toxin; diagnosis and treatment of possible associated endocrine disorders; and dealing with stress, anxiety, or fatigue (see ['Physiologic tremor'](http://www.uptodate.com/contents/overview-of-tremor?topicKey=NEURO%2F4895&elapsedTimeMs=8&source=search_result&searchTerm=tremor&selectedTitle=1%7E150&view=print&displayedView=full#H8) above). Single doses of [propranolol](http://www.uptodate.com/contents/propranolol-drug-information?source=see_link) taken in anticipation of social situations that are likely to exacerbate tremor, as with tremor associated with public speaking, are useful in some patients.

**Rest tremor** — Management of rest tremors, such as those associated with Parkinson disease (PD) or other parkinsonian disorders, is accomplished by treatment of the underlying disorder and usually consists of anticholinergic drugs or other antiparkinson agents such as [amantadine](http://www.uptodate.com/contents/amantadine-drug-information?source=see_link), dopamine agonists, levodopa, and [zonisamide](http://www.uptodate.com/contents/zonisamide-drug-information?source=see_link). This topic is discussed separately. (See ["Pharmacologic treatment of Parkinson disease"](http://www.uptodate.com/contents/pharmacologic-treatment-of-parkinson-disease?source=see_link).)

**Cerebellar tremor** — There is no useful pharmacotherapy for cerebellar tremor. The rare patient with severe intention tremor and little or no ataxia, such as sometimes occurs in multiple sclerosis, may be helped by deep brain stimulation of the ventral intermediate nucleus of the thalamus. (See ["Surgical treatment of essential tremor"](http://www.uptodate.com/contents/surgical-treatment-of-essential-tremor?source=see_link).)

**Orthostatic tremor** — Orthostatic tremor is limited to the legs and trunk and occurs exclusively while standing. Clinical experience suggests that orthostatic tremor is uniquely, but not always, responsive to treatment with [clonazepam](http://www.uptodate.com/contents/clonazepam-drug-information?source=see_link) [[41](http://www.uptodate.com/contents/overview-of-tremor/abstract/41)], although little data are available and no controlled trials exist. There is anecdotal evidence suggesting that [gabapentin](http://www.uptodate.com/contents/gabapentin-drug-information?source=see_link) is effective.

**Essential tremor** — [Propranolol](http://www.uptodate.com/contents/propranolol-drug-information?source=see_link) and [primidone](http://www.uptodate.com/contents/primidone-drug-information?source=see_link) are the most effective and well-studied medications for the treatment of essential tremor (ET). The medical treatment of ET is discussed in detail separately. (See["Pharmacologic treatment of essential tremor"](http://www.uptodate.com/contents/pharmacologic-treatment-of-essential-tremor?source=see_link).)

Both deep brain stimulation and unilateral thalamotomy are effective for the treatment of medically refractory ET. This topic is discussed separately. (See ["Surgical treatment of essential tremor"](http://www.uptodate.com/contents/surgical-treatment-of-essential-tremor?source=see_link).)

**INFORMATION FOR PATIENTS** — UpToDate offers two types of patient education materials, “The Basics” and “Beyond the Basics.” The Basics patient education pieces are written in plain language, at the 5th to 6th grade reading level, and they answer the four or five key questions a patient might have about a given condition. These articles are best for patients who want a general overview and who prefer short, easy-to-read materials. Beyond the Basics patient education pieces are longer, more sophisticated, and more detailed. These articles are written at the 10th to 12th grade reading level and are best for patients who want in-depth information and are comfortable with some medical jargon.

Here are the patient education articles that are relevant to this topic. We encourage you to print or e-mail these topics to your patients. (You can also locate patient education articles on a variety of subjects by searching on “patient info” and the keyword(s) of interest.)

* Basics topics (see ["Patient information: Tremor (The Basics)"](http://www.uptodate.com/contents/tremor-the-basics?source=see_link))
* Beyond the Basics topics (see ["Patient information: Tremor (Beyond the Basics)"](http://www.uptodate.com/contents/tremor-beyond-the-basics?source=see_link))

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Topic 4895 Version 9.0

**GRAPHICS**

**Common tremors**

|  |
| --- |
| **Resting tremor** |
| Parkinson disease |
| Parkinsonian syndromes |
| Midbrain (rubral) tremor |
| Wilson's disease |
| Severe essential tremor |
| **Postural-action tremor** |
| Enhanced physiologic tremor |
| Essential tremor |
| Primary writing tremor |
| Other extrapyramidal disorders |
| Parkinson disease |
| Wilson's disease |
| Dystonia |
| Cerebellar disease |
| Peripheral neuropathy |
| **Intention tremor (cerebellar outflow)** |
| Cerebellar disease |
| Multiple sclerosis |
| Midbrain stroke |
| Midbrain trauma |

Graphic 68810 Version 1.0

**Classification of tremor**

|  |  |  |
| --- | --- | --- |
| **Tremor type** | **Definition** | **Examples** |
| Rest tremor | Occurs in a body part that is supported in such a way that skeletal muscle activation is neither necessary nor intended. | The patient is recumbent on a bed or seated on a couch with the body part supported. Tremor is often enhanced by the performance of cognitive tasks or motor tasks with other body parts, and it is often suppressed, at least temporarily, by voluntary muscle contraction. |
| Postural tremor | Occurs in an attempt to hold a body part motionless against the force of gravity. | Extending the upper limbs horizontally, pointing at objects, sitting erect without support for the upper body, protruding the tongue. |
| Action tremor (including kinetic and isometric tremor) | Occurs during any voluntary contraction of skeletal muscle. Isometric tremor occurs during a muscle contraction against a rigid stationary object. | Finger-to-nose testing, heel-to-shin testing, reaching, writing, drawing, pouring water into a cup, eating with utensils, speaking. With isometric tremor: pushing against a wall, flexing the wrist against a table, making a fist. |

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Graphic 72091 Version 13.0

**Differential diagnosis of tremor**

|  |  |
| --- | --- |
| **Tremor** | **Description** |
| Essential tremor | Bilateral postural or kinetic tremor of the hands and forearms (usually 4 to 6 Hz) or isolated head tremor without evidence of dystonia. Absence of other neurologic signs or recent trauma preceding the onset of tremor. |
| Physiologic tremor | Enhanced physiologic tremor. High frequency (10 to 12 Hz), presence of known cause (see text). |
| Parkinson disease | Mixture of rest and action tremors; occasionally action tremor alone. Leg or foot tremor more common than with essential tremor, usually does not produce head tremor. Frequency 4 to 6 Hz. |
| Orthostatic tremor | Postural tremor in the torso and lower limbs while standing; may also occur in the upper limbs. Suppressed by walking. Tremor is high frequency (14 to 18 Hz) and synchronous among ipsilateral and contralateral muscles. |
| Cerebellar tremor | Postural, intention, or action tremor. Relatively low frequency (3 to 4 Hz). Associated with ataxia and dysmetria. |
| Neuropathic tremor | Variable tremor type and frequency, usually postural and kinetic tremor in the involved extremities. Other signs of peripheral neuropathy present. |
| Rubral or midbrain tremor | Mixture of rest, postural, and intention tremor with frequency of 2 to 5 Hz. Always associated with signs of brainstem or cerebellar damage. |

*Elble RJ. Diagnostic criteria for essential tremor and differential diagnosis. Neurology 2000; 54(11 Suppl 4):S2.*

Graphic 77497 Version 3.0

**Criteria for diagnosis of essential tremor**

|  |  |
| --- | --- |
| **Core criteria** | **Secondary criteria** |
| Bilateral action tremor of the hands and forearms (but not rest tremor) | Long duration (>3 years) |
| Absence of other neurologic signs, with the exception of cogwheel phenomenon | Positive family history |
| May have isolated head tremor with no signs of dystonia | Beneficial response to alcohol |

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Graphic 62078 Version 14.0

**Differentiating Parkinson disease and essential tremor**

|  |  |  |
| --- | --- | --- |
| **Clinical features** | **Parkinson disease tremor** | **Essential tremor** |
| Age at onset | >50 | Bimodal 2nd and 6th decade |
| Gender | M≥W | M=W |
| Family history | >25 percent | >50 percent |
| Asymmetry | +++ | + |
| Frequency | 4 to 6 Hz | 4 to 10 Hz |
| Character | At rest | Postural, kinetic |
| Supination-pronation | Flexion-extension |
| Distribution | Hands, legs, chin, tongue | Hands, head, voice |
| Associated features | Bradykinesia, rigidity, gait difficulty, postural instability, micrographia | Deafness, dystonia, parkinsonism |

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Graphic 60694 Version 14.0

**Symptoms or signs suggestive of tremor other than essential tremor**

|  |  |
| --- | --- |
| **Symptom or sign** | **Likely differential diagnosis** |
| Unilateral tremor, leg tremor, rigidity, bradykinesia, rest tremor | Parkinson disease |
| Gait disturbance | Parkinson disease, cerebellar tremor |
| Focal tremor | Dystonic tremor |
| Isolated head tremor with abnormal posture (head tilt or turning) | Dystonic tremor |
| Sudden or rapid onset | Psychogenic tremor, toxic tremor |
| Current drug treatment that may cause or exacerbate tremor | Drug-induced or toxic tremor |

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**Print Options:**



Text



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Graphics



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**Disclosures**

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