Tremors

By Laura M. Struble, PhD, GNP-BC

A tremor is a common, abnormal, involuntary movement of muscles that may be noted during a routine physical exam. It is defined as an unintentional rhythmical oscillatory movement of a body part.[1] Tremors may be a sign of an underlying disease or simply an exaggerated physiologic tremor observed in healthy people. Identifying the type of tremor is necessary to determine underlying neurologic disorders. The gold standard for diagnosing tremors is still history and physical exam.[1]

What is a tremor?

Tremors are commonly associated with movement disorders that have multiple etiologies and an extensive list of differential diagnoses. Most tremors are associated with neurologic conditions related to certain parts of the brain (such as basal motor nuclei, inferior olivary nucleus, and cerebellum) that control muscles throughout the body or a specific body part such as the hands.[2] Pathologic tremors include brain injury essential tremor, multiple sclerosis, Parkinson disease (PD), stroke, and neurodegenerative diseases that damage the brainstem or cerebellum. Metabolic disturbances that affect the brain and may produce tremors include drugs, alcohol, mercury poisoning, hyperthyroidism, hypoglycemia, or vitamin deficiencies (magnesium, thiamin). Insect bites from a black widow spider or red back spider may produce tremors. Finally, genetics may play a role in the development of tremors.[3]

Characteristics of tremor

Tremors can happen at any age, but their likelihood increases with age. They can occur in the hands, head, jaw, tongue, palate, vocal cords, trunk, or legs. Tremor may occur in one part of the body and progress to others, or may be seen in multiple extremities all at once. Diagnosis of the underlying cause becomes difficult when there is more than one type of tremor present. The majority of involuntary tremors increase during emotional stress, strong emotions, and/or physical fatigue. However, if a person is calm and relaxed, most tremors are dampened and usually disappear during sleep.[4] Tremors can be transient and vanish over time or chronic and progressive. Patients with a tremor may have minor functional consequences or extreme functional disabilities. Tremors can be embarrassing, and some patients are afraid to be seen in public. It is important for the NP to assess tremor symptoms quickly and accurately to maximize therapy and avoid unnecessary referrals.

Classification of tremor

Tremors are broadly categorized as hyperkinetic. They have different activation conditions, frequencies, and amplitudes. To be defined as a tremor, a movement must be rhythmic and have a pattern. The widely accepted classification divides tremors into two types: rest and action.[1]

Rest tremor

Rest tremor occurs in a body part that is relaxed and fully supported against gravity (for example, hands resting in the lap). The rate of this type of tremor is usually between 3 and 6 Hz/cycle/second.[5] If the rest tremor is mild or intermittent, it can be brought out or intensified by having the patient concentrate on a different task, such as opening and closing the contralateral hand or performing arithmetic.[4] Rest tremors are commonly observed in PD and account for about 75% of all cases of rest tremors.[5] Rest tremors are seldom seen in other conditions.[6]

Action tremor

An action tremor is produced by voluntary muscle contraction and is further classified as: postural, kinetic, isometric, or task-specific.

1. Postural tremor is produced when the affected body part maintains its position against gravity (such as extending arms in front of the body).
2. Kinetic tremor is produced during a voluntary movement and can be subdivided into simple kinetic tremor or intention tremor.
   a. Simple kinetic tremor occurs with movement of extremities (such as flexion-extension of the wrist).

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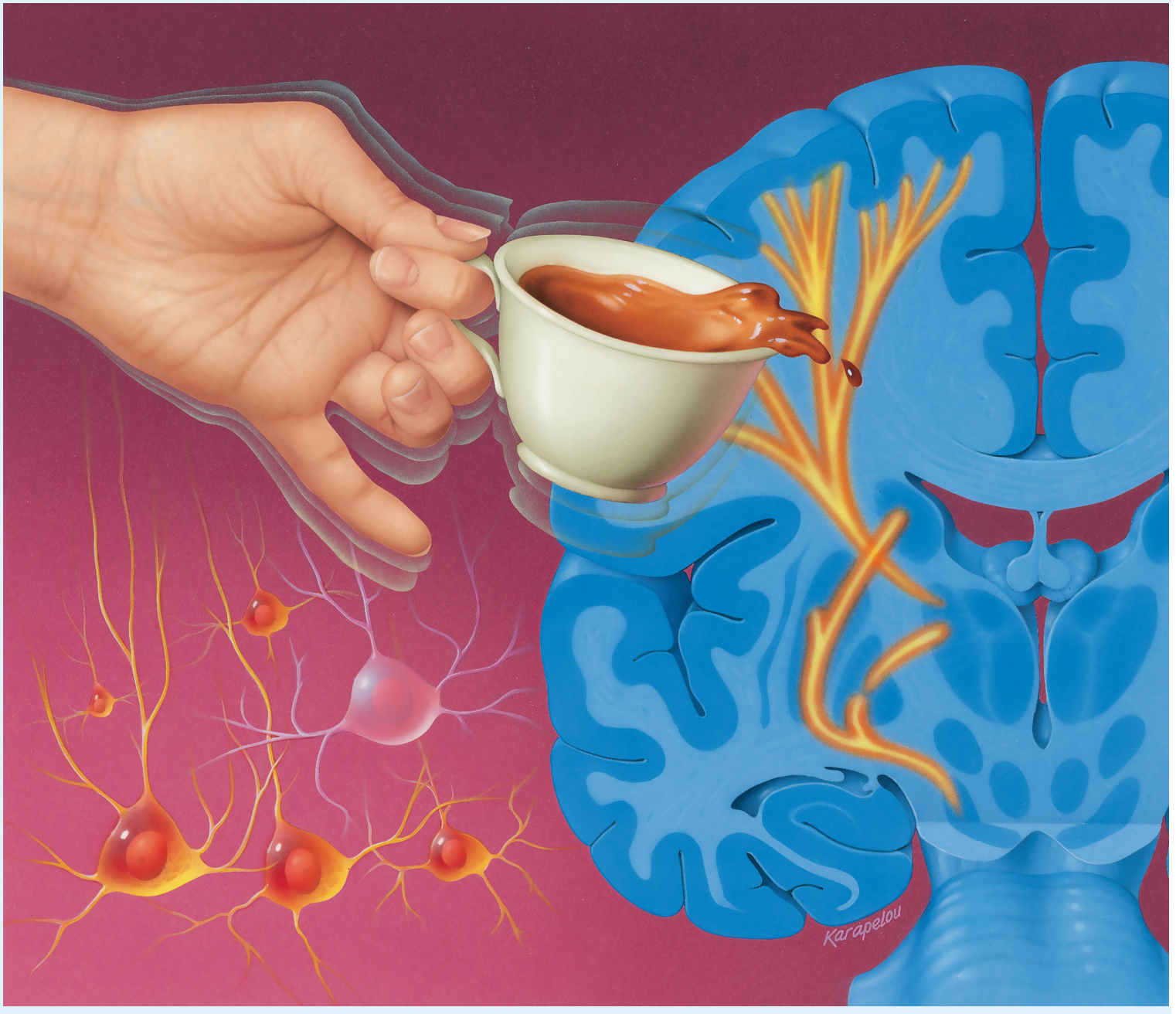

b. Intention tremor is produced with visually guided movements directed toward a specific target with significant amplitude fluctuation when approaching the target (when performing point-to-point movements, such as finger-nose-finger).[7] Intention tremors are commonly seen with cerebellar lesions.[4]

3. Isometric tremor occurs when there are voluntary muscle contractions without movement (such as pushing against a wall).
4. Task-specific tremor is rare and only occurs when performing a specific task such as writing, playing music, or speaking.[4,7,8]

Common tremor syndromes

Tremors are classified according to clinical features (see *Classification and description of tremor syndromes*).[6,8,9]

Physiologic tremor

Physiologic tremor is an action tremor present in everyone. This benign tremor is usually postural and does not interfere with activities of daily living. It is difficult to see with the naked eye and described as a high-frequency, low-amplitude tremor. Increased or enhanced physiologic tremor is described as an easily visible, high-frequency tremor with no evidence of an underlying neurologic disease.[1,11] Emotional stress, fear, or anxiety can exacerbate this tremor. Other precipitating factors include muscle fatigue, lack of sleep, and consuming large amounts of caffeinated drinks. Medical conditions that, if corrected, would reverse exacerbated physiologic tremor include thyrotoxicosis, hypoglycemia, certain drugs (such as some anticonvulsants, antidepressants, and neuroleptics),[14] and withdrawal from benzodiazepines and alcohol.[6,8,12]

Essential tremor

ET, formally known as benign ET, is the most common form of action tremor. It is the most common movement disorder in the world and is diagnosed primarily by clinical presentation.[7,8,12,15] It is almost 20 times more prevalent than PD.[16] Although ET is not life-threatening, the condition does have devastating consequences. Typically, ET is a postural or kinetic action tremor mainly affecting the hands. Patients often complain of difficulty writing, drawing, drinking from a cup, or using a computer. Usually, no other neurologic symptoms are present.[1] ET has an insidious onset and progresses over time. The condition affects males and females equally and occurs most often in adolescents and adults in their 50s or 60s. Almost half of ET cases are considered familial from an autosomal dominant gene.[17] ET usually starts in the arms with the flexion-extension of the wrists or abduction-adduction movement of the fingers.[4] The symptoms of ET are largely symmetrical, occurring primarily in the upper limbs (95%), and specifically in the hands. ET symptoms may also be noted in the head (35%), lower limbs (20%), voice (12%), and trunk (5%).[18] The frequency of the tremor ranges from 4 to 12 Hz and is usually symmetrical. The amplitude of ET tremors increases with fatigue, anxiety, stress, certain medications, and/or cold temperatures.

Pharmacologic treatment options do not cure the tremor, but may improve function and reduce embarrassment. The first-line therapies are propranolol and primidone. If the tremor is not reduced or the adverse reaction profile is too high, second-line therapies include gabapentin, alprazolam, nimodipine, and theophylline.[12,15] Propranolol is the only drug with labeled-use for symptomatic treatment of essential tremor; other drugs listed are off-label use**.** Moderate alcohol consumption can cause a transient decrease or alleviation of an ET; however, there may be a rebound effect once the alcohol has worn off. The use of alcohol to manage ET is not recommended but if the patient indicates that it helps this information supports the diagnosis of ET.[9,18,19]

Tremor in children

Tremor syndromes appear throughout childhood from neonate through adolescence. Movement disorders are sometimes missed in the very young if they are less apparent or slow progressing. It may not be until children show signs of impairment in school or recreational activities that parents consult the primary care provider. As with adults, ET is the most common form of tremor in children and usually seen in the teenage years. Infantile tremors resulting from vitamin B_{12} deficiency and Wilson disease between the ages of 6 and 20 years should always be ruled out. Other childhood tremors are most likely due to underlying metabolic and endocrine disorders, heterodegenerative disorders, or are drug-induced.[10,11]

Drug-induced tremors

Tremor is a frequent adverse reaction of medications related to drug toxicity and withdrawal from certain drugs.[8] Rest tremor, action/postural tremor, and intention tremor can be drug-induced**.** (see *Drugs that can cause rest, action/postural and intention tremors*). Most drug-induced tremors are symmetrical except for parkinsonism tremor. To determine whether a tremor is drug-induced, NPs should rule out other medical etiologies. A temporal relationship to the start of a drug should be investigated to determine whether there is a dose-response relationship. Finally, the tremor course should not be progressive in drug-induced tremors.[13]

Parkinsonism tremor

Parkinsonism tremor is the most common form of rest tremor. Parkinsonism is a group of movement disorders that have

Classification and description of tremor syndromes[6,8,9]

Type of tremor	Tremor frequency	Amplitude	Clinical features	Syndromes
Rest tremor Muscles are at rest	Low to medium	High	Occurs when limb is supported against gravity, muscles are not activated	• PD • Neurodegenerative parkinsonism diseases (multiple systems atrophy, progressive supranuclear palsy) • Drug-induced parkinsonism (neuroleptics, metoclopramide, phenothiazines) • Wilson disease
Action tremor produced by voluntary muscle contractions				
Postural	Medium to high	Low but increases with voluntary movement	Occurs when the limb maintains position against gravity	• Drug-induced (lithium, amiodarone, beta-adrenergic agonists) • ET • Metabolic disorders (thyrotoxicosis, pheochromocytoma, hypoglycemia) • Neuropathic tremor (neuropathy) • Physiologic tremor • Toxins (alcohol withdrawal, heavy metals)
Kinetic	Varies	Does not change with movement	Occurs during any voluntary movement	• Cerebellar lesions (stroke, multiple sclerosis, tumor) • Drug-induced ET • Physiologic • Posttraumatic stress disorder • Psychogenic
Simple kinetic tremor	Varies	Does not change with movement	Occurs during any voluntary movement	
Intention	Low	Increases with guided movement toward a target	Target directed	
Task-specific tremor	Varies	Variable	Occurs during specific activities (for example, writing)	
Isometric tremor	Medium	Variable	Occurs during muscle contraction against a stationary object	

similar features and symptoms. Idiopathic PD is the most common form of parkinsonism and the cause is unknown.[20] It is a progressive disease and primarily a consequence of degeneration of nuclei in a number of dopamine-producing nerve cells in the basal ganglia and related structures. The other forms of parkinsonism tremors have suspected causes such as encephalitis, stroke, Lewy body disease, or are drug-induced.

There are approximately 1.5 million Americans who currently have PD, and it affects both men and women equally.[20] The age of onset is usually 65; only 15% of people are diagnosed with PD under the age of 50.[20] A rest tremor is commonly the first neurologic sign in PD.

Classically, the rest tremor has a low frequency of 2 to 5 Hz and large amplitude. The tremor manifests initially in one hand and/or foot and progresses to the ipsilateral limbs before spreading bilaterally. Active movement strongly inhibits the tremor. In the later stages of PD it is not unusual to see an action or a postural tremor.[4] A PD tremor rarely involves the voice or head tremor, starts in one hand or foot, and is unaffected by alcohol.[16]

Unlike people with ET who do not have any other neurologic abnormalities, the diagnosis of PD is made using other diagnostic criteria. The patient must have a positive sustained response to a dopaminergic agent and two to three PD cardinal signs:

- Rest tremor that may involve the arms, legs, lips, jaw, chin, and tongue. The classic tremor is described as pill-rolling tremor that looks like someone is rolling a coin or pill between the thumb and index finger
- Rigidity or cogwheel rigidity (ratchet-like resistance) during passive range of motion
- Bradykinesia such as decreased arm swing with walking and reduced facial animation
- Postural instability.[7]

PD is idiopathic and neurodegenerative, so pharmacologic agents are used to alleviate the symptoms and slow the progression of the disease. A variety of agents may be used for tremor, such as levodopa, dopamine agonists, anticholinergic agents (such as benztropine mesylate, biperiden hydrochloride, and procyclidine hydrochloride), budipine (off-label), and second-line treatments such as clozapine, propranolol, and clonazepam.[12,21,22] The most important principle in management is to target the most disabling symptoms and individualize the therapy. The mainstay treatment is carbidopa/levodopa in older adults. Patients younger than 70 years with multiple features of PD should start with a dopamine agonist or monoamine oxidase inhibitor. Younger patients will require dopaminergic

Drugs that can cause rest, action/postural, and intention tremors[1,4,8,13,14]

Drug classification	Rest tremor	Action/postural tremor	Intention tremor
Antiarrhythmics		Amiodarone (Cordarone), mexiletine (Mexitil), procainamide (Pronestyl)	
Antibiotics and antifungals	Co-trimoxazole (Septra, Bactrim), amphotericin B (Fungizone, Amphocin)		
Antiepileptics		Valproic acid (Depakene)	
Beta-adrenergic agonists		Albuterol (Proventil), salmeterol (Serevent)	
Immunologic and chemotherapeutics	Thalidomide (Thalomid)	Tamoxifen (Nolvadex), cytarabine (Cytosar-U-), ifosfamide (Ifex)	
Gastrointestinal drugs	Metoclopramide (Reglan)	Metoclopramide (Reglan), cimetidine (Tagamet)	
Hormones		Thyroid hormones, calcitonin (Miacalcin), medroxyprogesterone (Provera)	
Methylxanthines		Theophylline (Theo-24, Theochron), caffeine	
Mood stabilizers	Lithium (Eskalith, Lithobid)	Lithium (Eskalith, Lithobid)	Lithium toxicity (Eskalith, Lithobid)
Neuroleptics	Haloperidol (Haldol)		
Newer antidepressants	SSRIs: Fluoxetine (Prozac), Paroxetine (Paxil) SNRIs: Duloxetine (Cymbalta), Venlafaxine (Effexor) Tetracyclic: Mirtazapine (Remeron)	SSRIs: Fluoxetine (Prozac), Paroxetine (Paxil) SNRIs: Duloxetine (Cymbalta), Venlafaxine (Effexor) Tetracyclic: Mirtazapine (Remeron)	
Tricyclic antidepressants		Amitriptyline, clomipramine (Anafranil), nortriptyline (Pamelor)	
Substance/drug abuse		Cocaine, alcohol withdrawal, 3,4-Methylenedioxymethamphetamine (Ecstasy), nicotine (tobacco)	Alcohol (chronic use)

SSRI = selective serotonin reuptake inhibitor; SNRI = serotonin-norepinephrine reuptake inhibitor.

therapy for a longer period and have an increased risk of levodopa complications such as tremor fluctuations and dyskinesias.[4,23,24] Surgery may be an option in the later stages of the disease. Thalamotomy, pallidotomy, and deep brain stimulation have shown some success in reducing tremors.[25]

Cerebellar tremor

Cerebellar tremor is the most common form of kinetic intention tremor. It is a slow, broad, low-frequency tremor of the extremities that occurs at the end of a purposeful movement such as pressing an elevator button. This type of tremor usually presents perpendicular to the direction of movement and varies in amplitude. Classically, the tremor amplitude increases as the limb is visually guided to the target. The dominant feature is a unilateral or bilateral low-frequency tremor without the presence of a rest tremor.

Cerebellar tremor is caused by lesions or damage to the cerebellum resulting from multiple sclerosis (most common), inherited degenerative disorders, stroke, or tumor. Rarely do these disorders present with just a tremor. Besides an intention or sometimes postural tremor, other characteristics of cerebellar disorders include incoordination, imbalance, dysarthria, and abnormal eye movements. Toxic-related causes of intention tremor include chronic alcohol abuse, lithium, heavy metals, antiepileptic drugs, antidepressants, and neuroleptic medications.[4,15,26] To date, there are no established pharmacologic treatments available for cerebellar tremors except treating the underlying causes. Surgical intervention such as thalamotomy or deep brain stimulation may benefit some patients. Occupational therapists may be able to help by fitting weight to the affected limb or teaching patients to brace the proximal limb during activity.

Psychogenic tremor

Psychogenic tremor is complex and variable with unknown documented incidence or prevalence rates. The etiology may be different in each patient. The patient usually describes an abrupt onset associated with a stressful life event. There is a variable course of tremors and they spontaneously go into remission. Patients are still able to carry out some activities of daily living despite severe tremors.[4] There are neurologic signs in the majority of patients, so the diagnosis is not just based on exclusion. The tremor can be resting, postural, intention, or a combination and can involve any body part. The tremor usually affects the extremities and does not involve the fingers.[27] It increases under direct observation and decreases with distraction. When the patient is asked to tap a beat with the limb contralateral to the tremulous limb, the tremor will decrease or shift to the frequency of the tapping. Referral to a neurologist and/or obtaining electrophysiologic studies may be useful. Successful treatment involves psychotherapy but may also require mild anxiolytics and antidepressants.[12]

Key history questions

NPs should ascertain the following information: age when the tremor started, type of onset (sudden or gradual), duration, progression, and any aggravating (such as tremor increases when tired or when taking a certain medication) or alleviating factors (such as tremor lessens with alcohol). Determine where the tremor started and the rate of progression to other body parts. Ask the patient if the tremor occurs with action, rest or when holding a body part in a particular position. In addition, ask if the tremor interferes with the patient's ability to use the hands or other body parts. Ask if there are any specific job-related disabilities and if the patient is socially embarrassed by the tremor. Have the patient describe any noticeable behavioral changes or memory losses (significant others may provide more accurate information about this). In addition, explore the patient's family history of tremors and medical history for neurologic (seizures, stroke, and head injury), endocrine, and metabolic disorders. Obtain a complete drug history and alcohol use/abuse history.[4,8,28] Be sure to assess for depression and anxiety because these psychologic sequelae are strongly associated neurologic conditions.[29,30]

Physical exam

The single most determining variable in diagnosing tremors is rhythmicity and can be described as regular or irregular oscillations. Other key tremor characteristics are body part or parts, location (distal, proximal), activation condition (when tremor is present), amplitude that is the degree of the limb displacement caused by the involuntary muscle contractions (fine/low, medium, coarse/large), and frequency that is the number of oscillations per second (slower—2 to 3 Hz, slow—less than 6 Hz, rapid—greater than 6 Hz).[18]

First, observe the patient sitting with hands resting in the lap and document tremor characteristics. Be sure to rule out other symptomatic movements such as chorea or dystonia (see *Hyperkinetic-involuntary abnormal movements*). Assess the face for oromotor dystonia (tardive dyskinesia) or masklike face. The patient should also be examined while assuming different positions (such as arms stretched out) and when moving (such as finger-nose-finger). If an ET is suspected, instruct the patient to stretch arms out in front, palms down (full pronation), then turn hands sideways (semipronation), then up into full supination. The semipronation may enhance ET oscillation, whereas tremor may be diminished in the supinate position.[5] Proximal tremors (such as shoulder rotator tremor) are seen in multiple sclerosis and other cerebellar dysfunctions. To elicit a proximal

Hyperkinetic-involuntary abnormal movements[18, 23, 31, 32]

Type	Description	Comments
Akathisia	Patient feels restless and has a desire to move to relieve uncomfortable sensations of crawling, itching, stretching, or creeping feelings. This usually occurs in the legs	Seen with use of antipsychotic medications.
Athetosis	Slow, writhing, continuous, uncontrollable movement of the arms and legs	Seen with perinatal and adult hypoxic ischemic injury. Movements are described as snakelike.
Ballism	Large, abrupt contractions of the extremities that are explosive flail-like movements	Seen with lesions of the subthalamus- (hemorrhage, tumor, or infarct). May only involve one side of the body.
Chorea	Rapid, irregular, jerky, uncoordinated movements, and abnormal posture	Seen with Huntington disease, Wilson disease, and systematic lupus erythematosus.
Dystonia	Sustained muscle contractions, often causing twisting or repetitive movements and abnormal postures	Seen with: (1) primary dystonia (spasmodic torticollis, blepharospasm, or writer's cramp), (2) secondary dystonia (Huntington disease, PD), (3) syndromes or combinations of hereditary and degenerative causes. May be painful as well as incapacitating.
Myoclonus	Random, sudden, shocklike, brief jerking contraction of a muscle	Seen with epilepsy and Creutzfeldt-Jakob disease. May occur once or repetitively and may persist in sleep.
Tics	Involuntary, rapid, nonrhythmic movement or sound, usually involving the face	Seen with transient tics, chronic tics, and Tourette syndrome. Preceded by an urge to move and can be controlled briefly.

tremor, have the patient form wings with their arms (shoulders abducted, elbows flexed, and palms facing down with index fingers pointing to each other in front of the chest).[18]

Important maneuvers to document functional capabilities and help determine the nature of the tremor include drinking from a cup (observe for swallowing difficulties as well observe for intention tremor) and writing or drawing a rhythmic pattern such as a spiral (small, cramped writing indicates PD and tremulous writing indicates ET).[5] Assess for other neurologic signs to help identify etiology. Perform cranial nerve testing, muscle tone, strength, reflexes, coordination, reflexes, and sensation. Test for rigidity (passively flex and extend the wrists and elbows) and cerebellar function (such as balance, gait, finger-nose-finger testing, heel-to-shin testing, rapidly alternate movements, and nystagmus). Exam findings that give clues to etiology of tremor include bradykinesia or rigidity (PD), nystagmus or ataxia (cerebellar lesion), and a wide difference in tremor symptoms and frequency (psychogenic).[4]

Lab workup

To rule out some disorders related to physiologic causes NPs may want to obtain a complete blood cell count with differential, thyroid-stimulating hormone, plasma glucose, and B_{12} level. It is important to exclude Wilson disease in patients younger than 50. It is an autosomal recessive disorder that results in abnormal deposition of copper in the brain and other organs and is life-threatening. Obtain a serum ceruloplasmin level, and a 24-hour urinary copper excretion to rule out this disease.[4,8]

Differential diagnosis

Tremors should be distinguished from other kinds of hyperkinetic movements disorders or dyskinesias. Choric movements, tics, and other involuntary abnormal hyperkinetic movements can quickly be distinguished from tremor by observation and physical exam.

Referral to a neurologist

Any patient with a sudden onset of tremor, asymmetric exam, cerebellar dysfunction, or complicated presentation should be sent for further evaluation. Failure to respond to appropriate medication is another indication for referral. Electromyography, accelerometry, and/or imaging studies may be necessary.[4,33] NP

REFERENCES

1. Deuschl G, Bain P, Brin M. Consensus statement of the Movement Disorder Society on tremor. Ad hoc scientific committee. *Mov Disord.* 1998; 13(suppl 3):2-23.
2. DeMyer W. *Technique of Neurological Examination.* 5th ed. New York: NY: McGraw-Hill; 2004:343-344.
3. Vaughan, JR, Davis MB, Wood, NW. Genetics of parkinsonism: a review. *Ann Human Genet.* 2001; 65(pt 2):111-126.
4. Jankovic J. Treatment of hyperkinetic movement disorders. *Lancet Neurol.* 2009; 8(9):844-856.
5. Velickovic M, Gracies JM. Movement disorders. Keys to identifying and treating tremor. *Geriatrics.* 2002; 57(7):32-36.
6. Chou KL. Diagnosis and management of the patient with tremor. *Med Health RI.* 2004; 87(5):135-138.

7. Colcher A, Simuni T. Clinical manifestations of Parkinson's disease. *Med Clin North Am.* 1999; 83(2):327-347.
8. Rehman H. Diagnosis and management of tremor. *Arch Intern Med.* 2000; 160(16):2438-2444.
9. Zesiewicz TA, Hauser RA. Phenomenology and treatment of tremor disorders. *Neurol Clin.* 2001; 19(3):651-680.
10. Smaga S. Tremor. *Am Acad Fam Physicians.* 2003; 68(8):1545-1552.
11. Grimes DA. Tremor-easily seen but difficult to describe and treat. *Can J Neurol Sci.* 2003; 30(suppl 1):S59-S63.
12. Uddin MK, Rodnitzky RL. Tremor in children. *Semin Pediatr Neurol.* 2003; 10(1):26-34.
13. Garewal G, Narang A, Das KC. Infantile tremor syndrome: a vitamin B12 deficiency syndrome in infants. *J Trop Pediatr.* 1998; 34(4):174-178.
14. Watanabe N, Omori, IM, Nakagawa A, et al. Safety reporting and adverse-event profile of mirtazapine described in randomized controlled trials in comparison with other classes of antidepressants in the acute-phase treatment of adults with depression. *CNS Drugs.* 2010; 24(1):35-53.
15. Bhidayasiri R. Differential diagnosis of common tremor syndromes. *Postgrad Med J.* 2005; 81(962):756-762.
16. Morgan JC, Sethi KD. Drug-induced tremors. *Lancet Neurol.* 2005; 4(12): 866-876.
17. Louis ED. Clinical practice: essential tremor. *N Engl J Med.* 2001; 345(12): 887-891.
18. Uitti RJ. Tremor: how to determine if the patient has Parkinson's disease. *Geriatrics.* 1998; 53(5):30-36.
19. Elble RJ. Diagnostic criteria for essential tremor and differential diagnosis. *Neurology.* 2000; 54(11 suppl 4): S2-S6.
20. Goetz CG, Horn SS. Treatment of tremor and dystonia. *Neurol Clin.* 2001; 19(1):129-144.
21. National Parkinson's Foundation. *2008 about Parkinson's disease.* http://www.parkinson.org/NETCOMMUNITY/Page.aspx?pid=225&srcid=201.
22. Wasielewski PG, Burns JM, Koller WC. Pharmacologic treatment of tremor. *Mov Disord.* 1998; 13(suppl 3):S90-S100.
23. Sethi KD. Tremor. *Curr Opin Neurol.* 2003; 16(4):481-485.
24. Jankovic J, Aguilar LG. Current approaches to the treatment of Parkinson's disease. *Neuropsychiatr Dis Treat.* 2008; 4(4):743-757.
25. Anderson VC, Burchiel KJ, Hogarth P, Favre J, Hammerstad JP. Pallidal vs subthalamic nucleus deep brain stimulation in Parkinson's disease. *Arch Neurol.* 2005; 62(4):554-560.
26. Seeberger LC. Cerebellar tremor- definition and treatment. *2005 CNI Rev.* http:www.thecni.org/review/17-fall05-all.
27. Deuschl G, Köster B, Lücking CH, Scheidt C. Diagnostic and pathophysiological aspects of psychogenic tremors. *Mov Disord.* 1998; 13(2):294-302.
28. Wyne KT. A comprehensive review of tremor. *JAAPA.* 2005; 18(12):43-50.
29. Slaughter JR, Slaughter KA, Nichols D, Holmes SE, Martens MP. Prevalence, clinical manifestations, etiology, and treatment of depression in Parkinson's disease. *J Neuropsychiatry Clin Neurosci.* 2001; 13(2):187-196.
30. Miller KM, Okun MS, Fernandez HF, Jacobson CE, Rodriguez RL, Bowers D. Depression symptoms in movement disorders: comparing Parkinson's disease, dystonia, and essential tremor. *Mov Disord.* 2007; 22(5):666-672.
31. Leckman JF, Bloch MH, King RA, Scahill L. Phenomenology of tics and natural history of tic disorders. *Adv Neurol.* 2006; 99:1-16.
32. Stewart F, Winer W. Hyperkinetic movement disorders. In: Weiner WJ, Goetz C, eds. *Neurology for the Non-Neurologist.* Philadelphia: PA, Lippincott Williams & Wilkins; 2004:155-204.
33. Popp AJ, ed. *A Guide to the Primary Care of Neurological Disorders.* New York, NY: Thieme; 1998:180-183.

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