

TREMOR

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Tremor Differential Diagnosis

Introduction: A tremor is often described as trembling or shaking. It is due to involuntary contraction of opposing muscle groups causing the affected body part(s) to move in a rhythmic, oscillatory way. Tremor is the most common movement disorder and can be found in almost 5% of the population over 40 years old. There are many types of tremors with varying causes, clinical features, and prognoses. In a chiropractic setting, a tremor is often an incidental finding noted when performing a physical exam on a patient. This document will provide an overview of tremors and how to approach a patient with this presentation.

Etiology: A vast number of diseases, disorders, medications, toxins, and substances can cause tremors. Etiologies can be genetic, acquired, and/or idiopathic. Neural dysfunction or lesions that cause tremor may result from injury, ischemia, metabolic abnormalities, or a neurodegenerative disorder. Sometimes tremor is a familial condition (e.g., essential tremor). A thorough history and in-office physical exam are necessary to identify and diagnose tremor types and the associated etiologies. In this document we will make note of many of the potential causes of tremors, but the focus will be on four main types:

- Tremor of Parkinson Disease
- Essential Tremor
- Physiologic (Enhanced)
- Cerebellar

Classification and characteristics: Clinical classification of tremor is based on history, tremor characteristics revealed on physical exam, associated neurologic and systemic signs, and, in some cases, additional testing. Tremors are commonly described by their shared clinical features or by the underlying etiology and are most often categorized as either rest tremors, or action tremors, with some tremor types sharing features of both. Tremors can also be classified by the etiology or underlying condition that may be causing the tremor.

Table 1: Tremor Types and Definitions

The chart below provides common definitions of each tremor type and differentiates the clinical features of rest and action tremor subtypes. Examples of etiologies that would cause each tremor subtype are listed, and the most common conditions as discussed in this document are in **bold** below, under the Examples column.

Tremor Type and General Features	Clinical Features	Examples
Rest Tremor		
Occurs in a body part that is supported in such a way that skeletal muscle activation is neither necessary nor intended (body part is at rest).	<ul style="list-style-type: none">● Tremor most evident when the patient is recumbent on a bed or seated with affected body part supported.● Tremor is often enhanced by the performance of cognitive tasks or motor tasks with other body parts.● Tremor is often suppressed, at least temporarily, by voluntary muscle contraction, as with voluntary activity.	<ul style="list-style-type: none">● Parkinson Disease● Severe or Long-Standing Essential Tremor● Parkinsonian syndrome (medications)● Midbrain (rubral) tremor● Wilson Disease

Action Tremors		
Postural: Occurs in an attempt to maintain a specific posture or position against the force of gravity.	<ul style="list-style-type: none"> Tremor most evident when a person maintains a position against gravity, including extending the upper limbs horizontally, sitting erect without support for the upper body, standing, protruding the tongue. 	<ul style="list-style-type: none"> Enhanced Physiologic tremor (drug induced or toxic or anxiety/fatigue) Essential Tremor Cerebellar disease Task-specific Tremor Other Extrapyrmidal disorders: PD, Wilson disease, Dystonia Neuropathic tremor
Kinetic: Occurs during any voluntary movement. Intention tremor (subtype of kinetic tremor): Occurs with purposeful movement toward a target.	<ul style="list-style-type: none"> Tremor exacerbated and most evident when performing a specific task, including finger-to-nose testing, heel-to-shin testing, reaching, writing, drawing, pouring water into a cup, eating with utensils, speaking. Typically, the tremor will become worse as an individual gets closer to their target. 	<ul style="list-style-type: none"> Cerebellar disease Essential Tremor Multiple Sclerosis Midbrain stroke Midbrain Trauma
Isometric: Occurs when voluntary muscle contraction is opposed by a rigid, stationary object.	<ul style="list-style-type: none"> Tremor exacerbated and most evident during a voluntary muscle contraction that is not accompanied by any movement including: pushing against a wall, flexing the wrist against a table, making a fist. 	<ul style="list-style-type: none"> Aqueduct stenosis Relapsing polyradiculoneuropathy Pontine lesions Head trauma

Chart Adapted From: Up to Date, Merck Professional Site, Habib-ur-Rehman. Abdo, Crawford

History: The following should be asked during a history to help with differential diagnosis of a tremor as part of a complete chief complaint history:

- Age of onset
 - <40 consider Wilson disease (see appendix A for description of Wilson disease)
 - >60 consider Parkinson disease
 - Essential tremor can begin at any age, but is more common >40
- Onset-gradual or sudden
 - Sudden may suggest toxic or substance withdrawal or ingestion. Withdraw from substances such as benzodiazepines, alcohol and opioids can cause physiologic tremor.
- Progression
 - Slowly progressive is typical of essential tremor and Parkinson Disease
- Affected body parts, unilateral or bilateral (see appendix E)
 - Unilateral consider Parkinson disease
 - Bilateral consider essential or physiologic tremor
 - Cerebellar tremor may be unilateral or bilateral
- Aggravating or relieving factors/movements/positions (See Table 1)
 - Rest: Parkinson disease, severe or long-standing essential tremor
 - Action:
 - Postural: enhanced physiologic tremor, essential tremor, cerebellar disease
 - Kinetic: cerebellar disease, essential tremor

- Associated symptoms: other neurological signs such as gait change, stiffness, abnormal eye movements, etc.
 - May suggest specific condition (see Table 2)
 - Rule out red flags (see appendix I and “red flags” below)

Past/family/social history:

- Medications (See appendix B for medications and drugs that may cause tremors)
 - Seizure medications can cause a tremor that mimics essential tremor
- Medical history
 - Hyperthyroidism, consider physiological tremor
 - Liver disease, consider Wilson disease
 - Substance abuse (see above-onset)
- Family history of tremor
 - Suspect essential tremor if there is a family history

Physical Exam: The physical exam is an immensely important part of differentiating between types of tremors and their various causes. When a patient presents with a tremor it is helpful to perform a few key exam procedures to differentiate between resting, postural, and kinetic tremors. In addition, we must consider whether there are any other abnormalities on physical exam, especially neurologic changes, that are suggestive of a specific disease.

Key exam procedures

1. Resting tremor: Observe the patient at rest
2. Postural tremor: Ask patient to hold out hands/arms
3. Kinetic tremor:
 - a. Instruct patient to draw a spiral
 - b. Have patient perform finger to nose test

Key findings

1. Tremor present at rest > resting tremor > most commonly Parkinson disease
2. Tremor provoked by holding hands/arms outstretched in front of the body > postural tremor > think essential tremor or enhanced physiologic tremor
3. Tremor activated by having the patient perform activities
 - a. abnormal spiral drawing, otherwise normal neurologic exam > think essential tremor
 - b. increased tremor amplitude and/or past pointing with finger to nose testing > think cerebellar tremor and perform additional neuro tests for cerebellar dysfunction

Step 1 - Observe the patient at rest.



The easiest way to do this is to look for a tremor to emerge while the patient is seated with hands in lap. If the tremor is present in the part of the body that is supported and relaxed, in this case the patient's hand(s), it is considered a resting tremor and the most likely cause is Parkinson disease.

The characteristic resting tremor seen in Parkinson disease is a unilateral pill-rolling tremor. It often includes involuntary movement of the thumb and index finger, rotating flexion/extension of the wrist and/or pronation and supination of the forearm. In addition to a resting tremor of the hand, some patients with Parkinson may also have resting tremors of the jaw, chin or leg.

When a patient presents with a resting tremor, look for other possible signs of Parkinson disease that would support the diagnosis. See Appendix C for a list of additional physical exam findings associated with Parkinson disease.

NOTE: Although a resting tremor is most often indicative of Parkinson disease, in some cases a *severe* or *long-standing* essential tremor may also occur at rest. Wilson disease (in patients under 40-years-old) and medication induced Parkinsonian syndrome should also be ruled out.

Step 2 - Ask the patient to stretch their hands/arms out in front of them and observe for onset of a tremor. If this sustained position activates the patient's tremor, we classify it as a postural tremor of the upper extremities and should consider essential tremor, enhanced physiologic tremor and drug induced tremor as potential causes.

NOTE: If the patient has difficulty maintaining balance while simply sitting or standing upright, they may have a cerebellar lesion and additional testing should be performed (see Step 4 below).



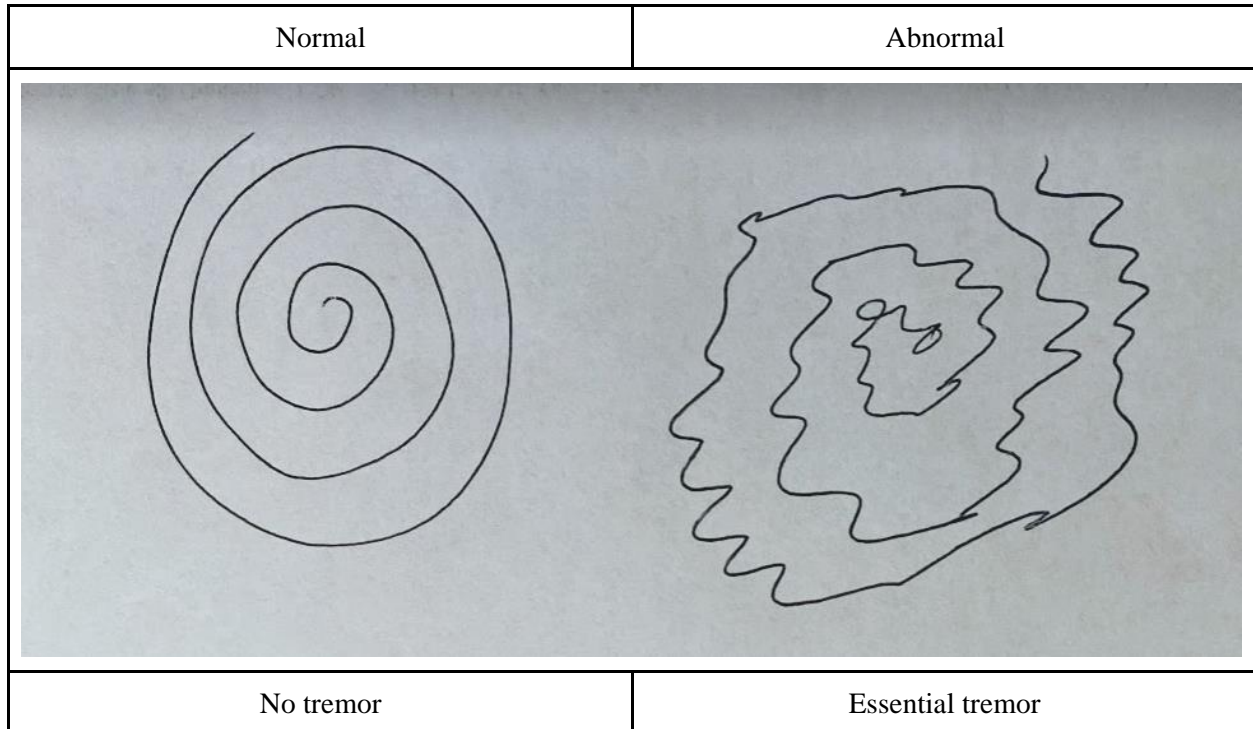
Occasionally, an enhanced physiologic tremor may be due to hyperthyroidism. Look for any classic signs of hyperthyroidism to support this as a potential diagnosis:

- Vital signs should be reviewed for tachycardia, hypertension, or fever.
- General examination should note any cachexia or psychomotor agitation.
- Eyes should be checked for any signs of exophthalmos or eyelid lag.

- Thyroid should be palpated for nodules and enlargement.

Step 3 - Instruct the patient to draw a spiral.

Asking the patient to draw a spiral on a piece of paper is an example of a task that can activate a kinetic tremor.



A kinetic tremor of the arms and hands is the most frequently encountered presentation of essential tremor, seen in over 90% of patients with essential tremor. It may also be provoked by having the patient perform other routine tasks that require active use of the hands/arms such as writing a sentence or drinking from a cup. When a kinetic tremor is identified during the exam, it is crucial to assess for any other abnormal neurologic findings on exam before concluding that it is an essential tremor.

Step 4 - Have the patient perform the finger to nose test. This will also provoke a kinetic tremor.

Instructions: Have the patient quickly alternate between touching their own nose and the examiner's finger. The examiner moves their finger to various locations throughout testing. Ideally, the examiner's finger should be far away enough from the patient that they extend the arm almost fully to reach the target.



If the patient's tremor gets worse as they approach the target (examiner's finger or patient's nose) it is called an intention tremor, a subtype of kinetic tremor.

If the patient overshoots the target (examiner's finger or patient's nose) during finger to nose testing this is called dysmetria or past pointing.



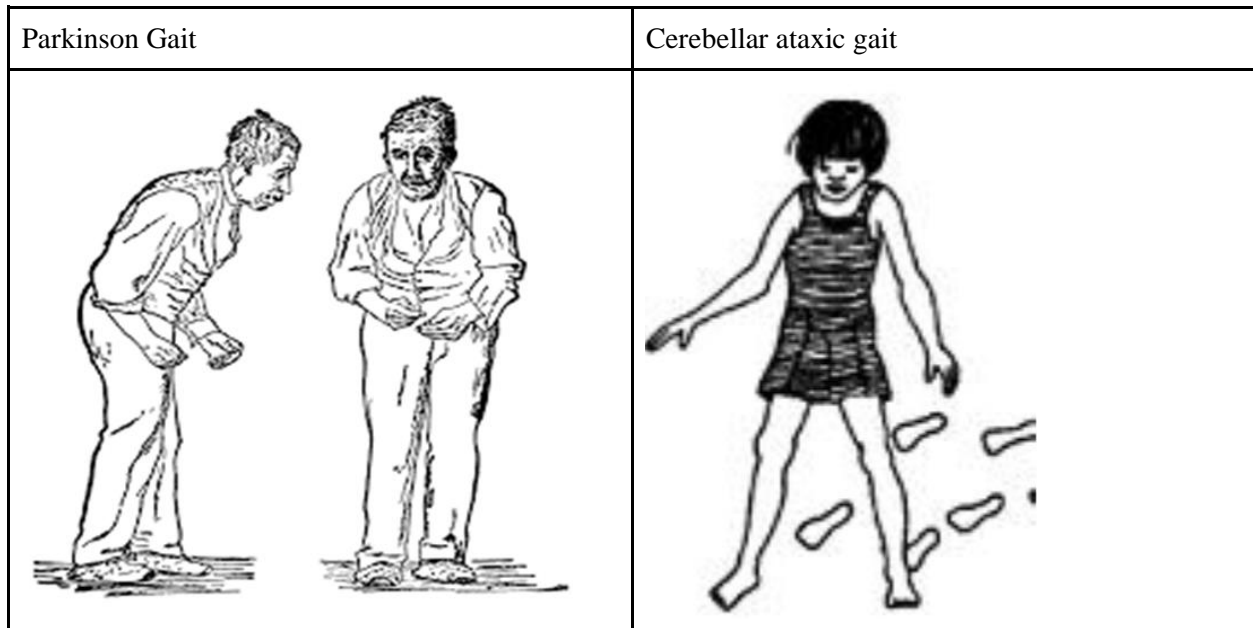
Intention tremors and past pointing are worrisome for pathology involving the cerebellum, such as having a stroke or multiple sclerosis. When either is present on finger to nose testing, the practitioner should perform additional tests to evaluate cerebellar function, such as heel to shin and rapid alternating hands tests. See **Appendix D for explanations of cerebellar tests.**

Additional Exam Considerations

- **Ask the patient to walk**

Observing the patient's gait as they walk across the exam room or down the hallway can provide supportive information about your suspected diagnosis. We expect a patient with essential tremor to have a normal gait and in fact tremors displayed during the exam with other tremor activating tests tend to disappear when the patient walks. On the other hand, a patient with Parkinson disease will often shuffle

and keep the feet close together when walking and a patient with a cerebellar disorder classically develops a broad-based gait and may veer back and forth while walking (called cerebellar ataxia or ataxic gait).



For a discussion of tremor frequency and summary of anatomical location see appendix E. Although frequency is an important part of the tremor evaluation, it is not easily quantified on exam and often requires additional specialty testing.

Differential Diagnosis:

Table 2: Differentiating Characteristics of Tremor Types: The chart below identifies clinical characteristics from the history and physical exam components that differentiate the most common tremor type

Characteristics Differentiating the Most Common Tremor Types				
Characteristic	Tremor of Parkinson Disease	Essential Tremor	Physiologic (Enhanced)	Cerebellar
Tremor Overview				
General Characteristics	Mixture of rest and action tremors; occasionally action tremor alone. Leg or foot tremor more common than with	Bilateral postural or kinetic tremor of the hands and forearms or isolated head tremor without evidence of dystonia; absence of other neurologic signs or recent	Physiologic tremor is usually not visible under ordinary circumstances, although some people have a natural proclivity to demonstrate mild, non-	Caused by known cerebellar pathology. The frequent association with ataxia, dysmetria, titubation, and other cerebellar signs serves to

	essential tremor, usually does not produce head tremor.	trauma preceding the onset of tremor.	disabling physiologic tremulousness, presence of known cause (e.g., medications, hyperthyroidism, hypoglycemia).	identify the cerebellar origin of intention tremor. The most common causes are multiple sclerosis, midbrain trauma, and stroke.
Tremor type	Resting tremor (less commonly postural)	Kinetic or Postural (less commonly intention and resting tremors)	Postural	Intention
Key History Elements				
Age	Older age (> 60)	All age groups	N/A	N/A
Family history	Usually negative	Positive in > 60% of patients	N/A	N/A
Tremor onset	Unilateral	Bilateral	Bilateral	UL or BL (lesion location dependent)
Key Physical Exam and Clinical Elements				
Muscle tone	Cogwheel rigidity	Normal (no rigidity)	Normal to mild hypertonicity	Hypotonia often noted
Facial expression	Decreased	Normal	Normal	Normal
Gait	Decreased arm swing, narrow-based and shuffling gait.	Normal or mild imbalance	Normal	Ataxia, wide-based and other gait abnormalities may be present
Tremor speed/frequency	Slow (4-6Hz)	Fast (6-12 Hz)	High/Fast frequency (10 to 12 Hz),	Relatively low frequency (3 to 4 Hz).
Other Neuro or Clinical Features	Cardinal symptoms include bradykinesia, postural instability, and rigidity. Many non-motor symptoms are also included in PD diagnosis.	Alcohol suppresses tremor	Thyroid and anxiety disorders may need clinical investigation.	Ipsilateral involvement to the lesion; abnormal finger to nose, imbalance, and heel to shin test.
Decrease of Tremor Symptoms	With movement and sleep	At rest	Removal of tremor enhancers (caffeine, anxiety, medication, hypoglycemia, etc.)	N/A

Management	Dopamine enhancement; physical therapy; speech therapy	Pharmacologic treatment may be utilized either intermittently or daily (propranolol, primidone, other CNS interactive meds)	Reduction or removal of the responsible offending medication or toxin; usually, nonpharmacologic treatment options are sufficient	Treatment of underlying cause; Cerebellar tremor lacks any useful pharmacotherapy
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Other Non-Tremor Differentials: There are several otherwise classified movement disorders that may be mistaken for tremor. Below is a list of these conditions. When observing a patient with abnormal movement(s) if it is unclear whether the patient is presenting with a tremor or a different movement disorder, they should be referred for further evaluation. A neurologist can help to rule out each of these other conditions to assure that the patient is indeed presenting with a tremor-based condition. Please see Appendix F for clinical definitions of these disorders and conditions.

- asterixis
- athetosis
- myokymia
- neuromyotonia
- motor tics
- myoclonus
- dystonia
- chorea
- hemiballismus
- clonus
- epilepsy partialis continua
- tardive dyskinesia

Ancillary Testing

Labs

If you suspect essential or enhanced physiologic tremors, ordering a comprehensive metabolic panel to rule out hypoglycemia, electrolyte imbalance, kidney or liver dysfunction is a reasonable starting point. If other etiologies are more likely, referring to PCP or neurologist for further lab work up is indicated. See the chart below for common workup.

Lab Evaluation by Tremor Type				
Rule out Wilson disease (age <40 y.o.)	Tremor of Parkinson Disease	Essential Tremor	Physiologic (Enhanced)	Cerebellar
Look for: Low serum ceruloplasmin Elevated Urine copper levels Elevated liver function tests	No specific lab tests to make diagnosis	No specific tests to make diagnosis but may be helpful to rule out other causes depending on clinical presentation with: Standard electrolyte panel Blood urea nitrogen Liver function tests	Glucose (rule out hypoglycemia) TSH, free T4 to assess for hyperthyroidism Maybe urine drug screen if clinically suspected - maybe pheochromocytoma if clinical features	No specific lab tests to make diagnosis

		maybe screening for heavy metal poisoning (lead, arsenic)		
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Imaging

Imaging is not part of the routine workup for tremors, except in the case of a suspected cerebellar tremor. When the history and exam suggest a cerebellar etiology, the patient should be referred to a neurologist for imaging of the brain. The patient will likely undergo a CT and/or MRI to evaluate for a possible stroke, brain mass, multiple sclerosis, or other pathology affecting the cerebellum.

Imaging may also be needed if the etiology is unclear based on the history and exam findings. Referral to the PCP or a neurologist to determine the most appropriate next steps is warranted in cases where there are significantly overlapping clinical features.

Red Flags

The following findings are of particular concern and indicate a higher likelihood that a tremor is from a pathological source and may need urgent work up (including laboratory analysis, imaging, and neurological referral).

- Abrupt onset
- Onset in people < 50 and with no family history of essential tremor
- Other neurologic deficits (e.g., change in mental status, motor weakness, cranial nerve palsy, ataxic gait, dysarthria)
- Tachycardia and agitation

See appendix I to identify conditions that should be considered when a red flag is identified--each condition will need a thorough work up and potential referral to PCP or neuro specialist.

Management: As a rule, tremor and any other abnormal neurologic signs on physical exam, especially if they are new, should prompt referral to either the primary care provider (PCP) or directly to a neurologist. The goal of this referral is to identify appropriate workup, including lab tests and/or imaging, that may be out of the scope of your evaluation. If during a thorough history and physical exam, it is determined that the etiology of the tremor is either medication induced or that the tremor has red flag components, it is imperative that the patient be referred to their PCP or neurologist for a full work up and possibly a discussion of medication discontinuation. If you are confident that your patient has an essential tremor, they should be referred to their primary care physician, but if you think your patient has an enhanced physiologic tremor because of something modifiable, such as too much caffeine, no referral is warranted.

Treatment for tremor depends on the cause and type of tremor and may include avoidance of triggers (such as eliminating caffeine in physiologic tremor), beta blockers and GABA interactive medications (essential tremor), dopamine-based therapies (parkinsonians). Treatment options for tremors usually target the nervous system, most notably the sympathetic nervous system peripheral innervations and GABA mediated CNS inhibition. There is a chart below that indicates the most common pharmaceutical treatment options for each common tremor diagnosis. Treatment for tremors is largely based on etiology, identifying underlying cause and addressing pathophysiology. As tremor diagnosis is most made in-office

based on patient presentation and symptoms, with no direct lab or imaging that can be used to monitor treatment, symptoms must be closely monitored to gauge effect of treatments.

Dopaminergic therapies are the mainstay for Parkinson's based tremor. Medications that provide precursors to dopamine (levodopa) and medications that prevent the breakdown of dopamine to increase neuronal exposure to dopamine (MOAI and COMPI) are the main therapeutic inroads. Tremors that are exacerbated by sympathetic neurotransmission (evident by an increase in tremor symptoms when the patient is anxious, fearful, or nervous) are often addressed with medications that block sympathetic stimulation. There is evidence that these tremor subtypes also respond positively to non-pharmaceutical therapeutics that can address this sympathetic overdrive as well. (MBSR, nervine botanical therapies, etc.). See appendix H for table.

A patient's medication list should be scrutinized before initiating any dietary, supplemental, or herbal interventions. Most of these treatment options available do not address the potential underlying cause of a tremor, but rather decrease the symptom displayed of a tremor. Patients are often asked to reflect on their quality of life and how much the tremor is impacting their quality of life before initiating a pharmaceutical intervention. A similar conversation should be initiated when discussing natural treatment options.

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Appendix A: Wilson Disease

Wilson disease is a genetic disorder that affects copper metabolism and often leads to liver problems. It is a rare condition, but it must be considered as a possible cause of tremor in patients ≤ 40 years old. A person with Wilson disease may present with a variety of tremor types as well as other abnormalities of movement (rigidity and dystonia are common).

Tremor characteristics:

- Bilateral upper limbs
- Asymmetric
- Rest or intention or
- sometimes Wing-beating (describe or add pic)

Most patients that have neurologic abnormalities due to Wilson disease also have pigmented deposits in the corneas, called Kayser-Fleischer (KF) rings.

Appendix B: Drugs and medications that may cause tremor

- | | |
|---|---|
| • caffeine | • neuroleptics (commonly) |
| • nicotine | • metoclopramide (commonly) |
| • cocaine | • antivertigo medications (prochlorperazine) |
| • amphetamines | • valproic acid |
| • beta-agonists | • calcium channel blockers cinnarizine, flunarizine |
| • anticonvulsants (especially sodium valproate) | • selective serotonin reuptake inhibitors |
| • thyroxine | • lithium |
| • lithium | • tetrabenazine |
| • tricyclic antidepressants | • amiodaron |
| • corticosteroids | |

Appendix C: Key Physical Exam Features seen with Parkinson Disease

Parkinson physical exam findings:

- resting tremor
- bradykinesia
- postural instability
- rigidity
- freezing
- slow, shuffling, narrow based gait - See gait section above.
- festination

Bradykinesia means there is slowed movement and may be noted on general exam of the Parkinson patient by absence of facial expressions (aka masked facies).

Postural instability is a loss of balance when standing upright that occurs in the later stages of Parkinson disease. This may be provoked during the exam by having the practitioner perform the pull test and noting if there is a loss of reflexes that normally prevent a patient from falling backward.

Rigidity is the presence of increased muscle tone with passive movement. It may also be referred to as cogwheel rigidity due to muscle stiffness having a ratchet-like quality.


Freezing describes the hesitation exhibited by a patient with Parkinson disease when starting a new movement, such as when getting up from a chair or turning around while walking.

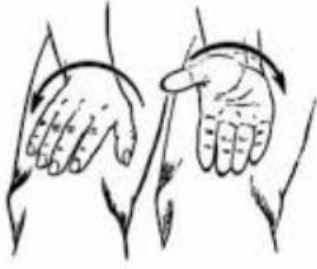
Festination is rapid acceleration of steps, a type of abnormal movement called tachykinesia.

Parkinson Disease is also identified as having many non-motor symptoms and an assessment of these symptoms is necessary for a diagnosis.

Note: Parkinsonian tremor may be exacerbated by stress (mental distraction) or initiation of movement of other body parts including walking.

Appendix D: Pictures of cerebellar physical exam procedures

Test	How to perform	Normal result	Abnormal result
Heel to Shin			
	Ask patient to slide the heel of one extremity down the shin of the other extremity and observe; repeat on other side	Heel stays on the shin and moves smoothly down to the foot	Patient not able to control smooth movement and the heel wobbles from side to side during testing
Rapid Alternating Hands			

	<p>Ask patient to rapidly pronate and supinate one hand on the other and observe; repeat on other side</p>	<p>Able to perform smoothly and quickly</p>	<p>Unable to perform rapidly with good coordination = dysdiadochokinesia</p>
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Appendix E: Tremor Frequency and Anatomical Distribution

In addition to describing tremors by type of tremor (resting vs action as outlined in Table 1) it is also common for tremors to be described by the tremor frequency (speed) or by the part of the body that is affected.

Although tremor frequency (oscillations per second) can be used to describe tremors, it is not particularly helpful in clinical diagnosis. Determining the exact tremor frequency during a physical exam is quite challenging and many pathologic tremors have a range of frequency of 4 to 8 Hertz. See Table 2: Differentiating Characteristics of Tremor Types for Tremor speed/frequency for the four main tremors discussed in this document. If it is necessary to determine the exact tremor frequency, a neurologist may consider utilizing EMG (electromyography). More often, it is reasonable to simply note if a tremor frequency is slow (in the range of ~3-6Hz) or fast (in the range of ~ 6-12 Hz).

Tremors can occur in many locations in the body. The anatomic distribution of tremor can be classified as:

- Focal (only one body region is affected, such as voice, head, jaw, or one limb)
- Segmental (two or more contiguous body parts in the upper or lower body are affected, such as head and arm)
- Hemi-tremor (one side of the body is affected)
- Generalized (upper and lower body are affected)

Appendix F: Definitions of tremor DDX (otherwise classified movement disorders)

- **Asterixis:** flapping movement with extended wrist occurring with hepatic encephalopathy or other encephalopathy
- **Athetosis:** is nonrhythmic, slow, writhing, sinuous movements predominantly in distal muscles, often alternating with postures of the proximal limbs.
- **Myokymia:** involuntary localized quivering of a few muscles, usually face or eyelid; may indicate brainstem disease such as multiple sclerosis

- **Neuromyotonia:** quivering and twitching of muscles in setting of other symptoms such as muscle cramping, stiffness, walking difficulty; often seen as a paraneoplastic syndrome or autoimmune disease
- **Motor tics:** ‘stereotyped’ character of the recurrent movements, often preceded by rising discomfort or urge (‘sensory tic’) that is relieved by the actual movement (‘itch and scratch’ analogy)
- **Myoclonus:** myoclonic movements are sudden, brief, shock-like involuntary movements, which are usually positive (caused by muscle contraction) but can sometimes be negative (due to brief loss or inhibition of muscular tonus).
- **Chorea:** Sudden, jerky, involuntary movements that are abrupt, unpredictable and nonrhythmic, resulting from a continuous random flow of muscle contractions; pattern of movements randomly changes from one body part to another,
- **Dystonia:** an involuntary abnormal co-contraction of antagonistic muscles, which may cause sustained abnormal postures or twisting and repetitive movements.
- **Hemiballismus** is unilateral rapid, nonrhythmic, non-suppressible, wildly flinging movement of the proximal arm and/or leg; rarely, such movement occurs bilaterally (ballismus); may be considered a severe form of chorea.
- **Clonus:** involuntary and rhythmic muscle contractions caused by a permanent lesion in descending motor neurons.
- **Epilepsia partialis continua:** considered the status epilepticus (SE) equivalent of focal onset seizure with retention of awareness, it can manifest as focal motor or sensorimotor clonic seizures without jacksonian march.

Appendix G: Other (Less Common) Tremor Disorders and Descriptions

<i>Tremor</i>	<i>Description</i>
Psychogenic Tremor	Features consistent with psychogenic tremor are abrupt onset, spontaneous remission, change in tremor characteristics, and extinction with distraction.
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.
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 20 Hz) and synchronous among ipsilateral and contralateral muscles.
Dystonic Tremor	The tremor is usually irregular and jerky, and certain hand or arm positions will extinguish the tremor. Other signs of dystonia (e.g., abnormal flexion of the wrists) are usually present

Wilson Disease	Wilson disease is a rare, autosomal-recessive disorder that manifests in persons five to 40 years of age, sometimes with a wing-beating tremor.
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Appendix H: Therapeutics for Common Tremor Types

Tremor Type/Condition	Therapeutic MOA	Pharm Example	Herbal Example	Nutraceutical/Diet Example	Lifestyle	Manual medicine/Physical Therapy
Essential Tremor:	GABA Analogue. Anticonvulsants (GABA analogue, sodium channel inhibition)	Topiramate Gabapentin Barbiturate (Primidone)	Nervine Herb (Valerian, Kava, Lemon Balm, etc.)	GABA Mediterranean Diet Showed Decrease in symptoms	Stress management, removal of sympathetic triggers (stress, panic, caffeine). Alcohol can lessen tremor, but abuse risk is high.	Physical therapy: increase strength in muscles with tremor
	Block sympathetic stimulation	Non-Selective Beta Blocker (Propranolol)	N/A			
	Neurotransmitter Augmentation	2nd Generation Antipsychotics (Clozapine)	N/A			
		Antidepressants (Mirtazapine)	N/A			
Parkinson's Associated Tremor: Parkinson disease is treated.	Dopamine Precursor and Agonists	Levodopa (with Carbidopa) Amantadine	Mucuna pruriens	Tyrosine (precursor to Dopa) and NT support Vitamins (B6) Protein Redistribution Diet	Stress Management through MBSR Regular movement/exercise	Massage therapy: Neuromuscular therapy Whole body vibration (duration of effects unknown)
	Anticholinergic	N/A				
Physiologic Tremor: No treatment is necessary unless symptoms are bothersome; treat	Benzodiazepine (GABA) Used when tremor with chronic anxiety	Diazepam Lorazepam Oxazepam	Nervine Herb (Valerian, Kava, Lemon Balm, etc.)	GABA	Avoiding triggers* can help prevent or reduce symptoms.	

underlying and exacerbating conditions.						
	Block sympathetic stimulation	Non-Selective Beta Blockers (Propranolol)	N/A			

*Common Physiologic Tremor Triggers: caffeine, nicotine, fatigue/exhaustion, sleep deprivation, pharmaceutical drugs (see appendix A), stress and anxiety.

Appendix I: Red Flag Condition Considerations

More thorough decision-making points with red flag identification: a [thorough](#) review of systems and focused neurological history should target the identification of symptoms of causative disorders as listed in the chart below. Each of these conditions can have tremor as an associated symptom, if the symptoms below are positive, need an urgent referral and work up.

ROS Indications	Condition
Multiple episodic neurologic problems (UL loss of sensation, change in vision, urinary or bowel incontinence)	Multiple sclerosis
Recent sudden onset of motor weakness, language difficulties, or dysarthria	Stroke
Confusion and fever	Meningitis, encephalitis, brain abscess, or brain tumor
Muscle rigidity, gait and postural problems, and slowness of movement	Parkinson disease or other forms of parkinsonism
Weight loss, increased appetite, palpitations, diarrhea, and heat intolerance	Hyperthyroidism
BL sensory deficits (glove and stocking presentation)	Peripheral neuropathy
Alcohol withdrawal or drug toxicity	Agitation and hallucinations

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