

Palpitations

By [Andrea D. Thompson](#), MD, PhD, University of Michigan;
[Michael J. Shea](#), MD, Michigan Medicine at the University of Michigan
Reviewed/Revised Aug 2024

Pathophysiology | Etiology | Evaluation | Treatment | Geriatrics Essentials | Key Points

Palpitations are the perception of cardiac activity. They are often described as a fluttering, racing, or skipping sensation. They are common; some patients find them unpleasant and alarming. Palpitations can occur in the absence of heart disease or can result from life-threatening heart disorders. The key to diagnosis and treatment is to “capture” the rhythm on ECG and make careful observations during the palpitations.

Pathophysiology of Palpitations

The mechanisms responsible for the sensation of palpitations are unknown. Ordinarily, sinus rhythm at a normal rate is not perceived, and palpitations thus usually reflect changes in cardiac rate or rhythm. In all cases, it is the abnormal movement of the heart within the chest that is felt. In cases of isolated extrasystoles, the patient may actually perceive the augmented postextrasystolic beat as the “skipped” beat rather than the premature beat itself, probably because the extrasystole blocks the next sinus beat and allows longer ventricular filling and thus a higher stroke volume.

The clinical perception of cardiac phenomena is highly variable. Some patients are aware of virtually every premature ventricular beat, but others are unaware of even complex atrial or ventricular tachyarrhythmias. Awareness is heightened in patients who are sedentary, anxious, or depressed and reduced in those who are active and happy. In some cases, palpitations are perceived in the absence of any abnormal cardiac activity.

Etiology of Palpitations

Some patients simply have heightened awareness of normal cardiac activity, particularly when exercise, febrile illness, or anxiety increases heart rate. However, diligent evaluation for arrhythmia as the cause of palpitations is warranted. Arrhythmias range from benign to life threatening.

The **most common arrhythmias** include

- [Premature atrial contractions](#) (PACs)
- [Premature ventricular contractions](#) (PVCs)

Both of these arrhythmias usually are harmless.

Other common arrhythmias include

- [Paroxysmal supraventricular tachycardia](#) (PSVT)
- [Atrioventricular nodal reentrant tachycardia](#)
- [Atrial fibrillation](#) or [atrial flutter](#)
- [Ventricular tachycardia](#)

Bradyarrhythmias rarely cause patients to sense palpitations, although some patients are aware of the slow rate.

Causes of arrhythmias

Some arrhythmias (eg, PACs, PVCs, PSVT) often occur spontaneously in patients without serious underlying disorders, but others are often caused by a serious cardiac disorder.

Serious cardiac causes include [myocardial ischemia](#) or other myocardial disorders, congenital heart disease (eg, [Brugada syndrome](#), [arrhythmogenic right ventricular cardiomyopathy](#), [congenital long QT syndrome](#), [Wolf-Parkinson-White syndrome](#)), [valvular heart disease](#), and conduction system disturbances (eg, disturbances that cause bradycardia or [heart block](#)). Patients with [orthostatic hypotension](#) commonly sense palpitations caused by sinus tachycardia upon standing.

Noncardiac disorders that increase myocardial contractility (eg, thyrotoxicosis, [pheochromocytoma](#), [anxiety](#)) may cause palpitations.

Some medications and drugs, including digitalis glycosides, caffeine, alcohol, nicotine, and sympathomimetics (eg, albuterol, amphetamines, cocaine, dobutamine, epinephrine, ephedrine, isoproterenol, norepinephrine, and theophylline), frequently cause or exacerbate palpitations. Most antiarrhythmic medications themselves can cause arrhythmias.

Metabolic disturbances, including [anemia](#), hypoxia, hypovolemia, and electrolyte abnormalities (eg, diuretic-induced [hypokalemia](#)), can trigger or exacerbate palpitations.

Consequences

Many arrhythmias that cause palpitations have no adverse physiologic consequences of their own (ie, independent of the underlying disorder). However, bradyarrhythmias, tachyarrhythmias, and heart blocks can be unpredictable and may adversely affect cardiac output and cause hypotension or death. [Ventricular tachycardia](#) sometimes degenerates to [ventricular fibrillation](#).

Evaluation of Palpitations

A complete history and physical examination are essential. Observations by other medical personnel or reliable observers should be sought.

History

History of present illness should cover the frequency and duration of palpitations and provoking or exacerbating factors (eg, emotional distress, activity, change in position, intake of caffeine or other medications or drugs). Important associated symptoms include syncope, light-headedness, tunnel vision, dyspnea, and chest pain. Asking the patient to tap out the rate and cadence of palpitations is better than a verbal description and often suggests a diagnosis, as in the “missed beat” of atrial or ventricular extrasystoles or the rapid total irregularity of [atrial fibrillation](#).

Review of systems should cover symptoms of causative disorders, including heat intolerance, weight loss, and tremor ([hyperthyroidism](#)); chest pain and dyspnea on exertion ([myocardial ischemia](#)); and fatigue, weakness, heavy vaginal bleeding, and/or dark tar-like stools ([anemia](#)).

Past medical history should identify known potential causes, including documented arrhythmias and heart or thyroid disorders. Family history should note occurrences of syncope (sometimes mistakenly described as seizures) or sudden death at an early age.

The drug profile should be reviewed for offending prescription medications (eg, antiarrhythmics, digoxin, beta-agonists, theophylline, and rate-limiting drugs); over-the-counter medications (eg, cold and sinus medications, dietary supplements containing stimulants), including alternative medicines; and illicit drugs (eg, cocaine, methamphetamines). Caffeine (eg, coffee, tea, numerous soft drinks and energy drinks), alcohol, and tobacco use should be determined.

Physical examination

The **general examination** should note whether an anxious demeanor or psychomotor agitation is present. Vital signs are reviewed for fever, hypertension, hypotension, tachycardia, bradycardia, tachypnea, and low oxygen saturation. Orthostatic changes in blood pressure (BP) and heart rate should be measured.

Examination of the head and neck should note any abnormality or dyssynchrony of the jugular pulse waves compared with the carotid pulse or auscultated heart rhythm and findings of hyperthyroidism, such as thyroid enlargement or tenderness and exophthalmos. The conjunctivae, palmar creases, and buccal mucosa should be inspected for pallor.

Cardiac auscultation should note the rate and regularity of the rhythm as well as any murmurs or extra heart sounds that might indicate underlying valvular or structural heart disease.

Neurologic examination should note whether resting tremors or brisk reflexes are present (suggesting excess sympathetic stimulation). An abnormal neurologic finding suggests that seizures rather than a cardiac disorder may be the cause if syncope is one of the symptoms.

Red flags

Certain findings suggest a more serious etiology:

- Light-headedness or [syncope](#) (particularly if injury occurs as a result of syncope)

- [Chest pain](#)
- Dyspnea
- New onset of irregularly irregular heart rhythm
- Heart rate >120 beats/minute or < 45 beats/minute while at rest
- Significant underlying heart disease
- Family history of recurrent syncope or sudden death
- Exercise-induced palpitations or, particularly, exercise-induced syncope

Interpretation of findings

History (see table [Suggestive Historical Findings in Patients With Palpitations](#)) and, to a lesser extent, physical examination provide clues to the diagnosis.

Palpation of the arterial pulse and cardiac auscultation may reveal a rhythm disturbance. However, the examination is not always diagnostic of a specific rhythm, except when it identifies the unique irregular irregularity of some cases of rapid [atrial fibrillation](#), the regular irregularity of coupled [atrial](#) or [ventricular extrasystoles](#), the regular tachycardia at 150 beats/minute of [PSVT](#), and the regular bradycardia of < 35 beats/minute of complete [atrioventricular block](#).

Careful examination of the jugular venous pulse waves simultaneously with cardiac auscultation and palpation of the carotid artery allows evaluation of atrial rhythm through jugular waves while the auscultated sounds or the pulse in the carotids are the product of ventricular contraction.

Thyroid enlargement or tenderness with exophthalmos suggests thyrotoxicosis. Marked hypertension and regular tachycardia are consistent with [pheochromocytoma](#).

TABLE

Suggestive Historical Findings in Patients With Palpitations

Finding	Possible Cause
Occasional skipped beats	PACs , PVCs
Rapid, regular palpitations with sudden onset and termination	PSVT , atrial flutter with 2:1 atrioventricular block, VT
Often history of recurrence	
Syncope following palpitations	Sinus node dysfunction , atrioventricular bypass tract (such as in Wolff-Parkinson-White syndrome), congenital long QT syndrome , VT, atrial fibrillation , atrial flutter
Palpitations during exercise or an emotional episode	Sinus tachycardia (particularly in healthy people) Ventricular arrhythmia from exercise-induced ischemia (particularly in people with congenital arrhythmic disorders or CAD)
Palpitations following episodic* drug use	Drug-induced cause
Sense of doom, anxiety, or panic	Suggests (but does not confirm) a psychological factor
Recent surgery (postoperative period)	Sinus tachycardia (eg, due to infection, bleeding, pulmonary embolism, pain), atrial fibrillation, atrial flutter
Recurrent episodes since childhood	Supraventricular arrhythmia (eg, atrioventricular nodal reentrant bypass tract , Wolff-Parkinson-White syndrome) Congenital long QT syndrome (usually manifests during adolescence)
Family history of syncope or sudden death	Brugada syndrome , congenital long QT syndrome , inherited dilated cardiomyopathy , arrhythmogenic right ventricular cardiomyopathy , or hypertrophic cardiomyopathy

* The role of regular use of drugs (particularly medications) or substances (eg, daily caffeine) can be hard to determine; sometimes a trial of withdrawal is diagnostic. All drugs with

cardiovascular effects, most psychoactive drugs, and drugs capable of causing hypokalemia or hypomagnesemia must be suspected.

CAD = coronary artery disease; PACs = premature atrial contractions; PSVT = paroxysmal supraventricular tachycardia; PVCs = premature ventricular contractions; VT = ventricular tachycardia.

Testing

Testing typically is done:

- ECG, sometimes with ambulatory monitoring
- Laboratory testing
- Sometimes imaging studies, stress testing, or both

ECG is done, but unless the recording is done while symptoms are occurring, it may not provide a diagnosis. Many cardiac arrhythmias are intermittent and show no fixed ECG abnormalities; exceptions include

- [Wolff-Parkinson-White syndrome](#)
- [Long QT syndrome](#)
- [Arrhythmogenic right ventricular cardiomyopathy](#).
- [Brugada syndrome](#) and its variants

If no diagnosis is apparent and symptoms are frequent, [Holter monitoring](#) for 24 to 48 hours is useful; for intermittent symptoms, an event recorder worn for longer periods and activated by the patient when symptoms are felt is better. These tests are used mainly when a sustained arrhythmia is suspected, rather than when symptoms suggest only occasional skipped beats. Patients with very infrequent symptoms that clinicians suspect represent a serious arrhythmia may have a device implanted beneath the skin of the upper chest. This device, often called a loop recorder, continuously records the rhythm and can be interrogated by an external machine that allows the cardiac rhythm to be printed. Finally, a variety of commercially available products that patients may be using may provide additional useful information. These products include fitness trackers, which monitor heart rate, and mobile ECG monitors that are available for phones and watches.

Laboratory testing is needed in all patients. All patients should have a complete blood count and measurement of serum electrolytes, including magnesium and calcium. Further testing should be aimed at suspected causes. The cardiac biomarker troponin should be measured in patients with ongoing arrhythmias, chest discomfort, or other symptoms suggesting active or recent coronary ischemia, myocarditis, or pericarditis.

Thyroid function tests are indicated when atrial fibrillation is newly diagnosed or there are symptoms of [hyperthyroidism](#). Patients with paroxysms of high BP should be evaluated for pheochromocytoma.

Sometimes [tilt-table testing](#) is done in patients with postural syncope.

Imaging is sometimes needed. Patients with newly diagnosed arrhythmia, findings suggesting cardiac dysfunction or findings suggesting structural heart disease require [echocardiography](#), and sometimes [cardiac MRI](#). Patients with symptoms on exertion require stress testing sometimes with [stress echocardiography](#), [nuclear scanning](#), or [PET](#).

Treatment of Palpitations

Precipitating [medications and substances that can cause syncope](#) are stopped. If dangerous or debilitating arrhythmias are caused by a necessary medication, a different medication should be tried.

For isolated PACs and PVCs in patients without structural heart disease, simple reassurance is appropriate. For otherwise healthy patients in whom these phenomena are disabling, a beta-blocker can be given provided efforts are made to avoid reinforcing the perception by anxious patients that they have a serious disorder.

Identified rhythm disturbances and underlying disorders are investigated and treated (see table [Some Treatments for Arrhythmias](#)). However, for rapid tachyarrhythmias in a patient in hemodynamic distress, cardioversion should precede further diagnostic assessment.

TABLE

Some Treatments for Arrhythmias

Disorder	Treatment*
Narrow complex tachycardias	
Multifocal atrial extrasystoles	Reassurance, a non-dihydropyridine calcium channel blockers, or a beta-blocker
Atrial fibrillation	<p>Anticoagulation</p> <p>For rate control:</p> <ul style="list-style-type: none"> • Beta-blockers • Verapamil • Diltiazem • Digoxin <p>For rhythm control:</p> <ul style="list-style-type: none"> • Antiarrhythmic medications (eg, ibutilide, amiodarone, propafenone, dronedarone, sotalol, dofetilide) • Cardioversion • Radioablation <p>Sometimes a Maze procedure</p>
Atrial flutter	<p>Anticoagulation</p> <p>Radioablation (often the best treatment)</p> <p>Sometimes DC cardioversion, digoxin, beta blocker, and/or verapamil</p>
Ectopic supraventricular tachycardia (eg, atrial tachycardia)	Sometimes, DC cardioversion, rate control medications (excluding digoxin) , antiarrhythmics, overdrive pacing, and/or ablation
Reentrant supraventricular tachycardias (eg atrioventricular nodal reentrant tachycardia)	<p>Vagotonic maneuvers</p> <p>AV nodal blocking medications (eg, beta blockers, verapamil)</p> <p>Ablation (often the best treatment)</p>
Broad complex tachycardias	
	Immediate pharmacotherapy or DC cardioversion

<u>Ventricular tachycardia</u>	Amiodarone, sotalol, propafenone, lidocaine, mexiletine, flecainide, radioablation Sometimes an implanted defibrillator
<u>Torsade de pointes</u>	If unstable, immediate DC cardioversion, magnesium, and/or potassium; sometimes an implanted defibrillator Ongoing treatment as needed with magnesium, potassium, a beta-blocker, isoproterenol, or overdrive cardiac pacing Sometimes an implanted defibrillator
<u>Ventricular fibrillation</u>	Defibrillation Sometimes medications (eg, amiodarone) Sometimes an implanted defibrillator
<u>Brugada syndrome</u>	Usually DC cardioversion or an implanted defibrillator
* Always identify and correct causes and exacerbating factors (eg, electrolyte abnormalities, hypoxemia, drugs).	
AV = atrioventricular; DC = direct current.	

Geriatrics Essentials: Palpitations

Older adults are at particular risk of adverse effects of antiarrhythmics; reasons include lower glomerular filtration rate and concomitant use of other medications. When pharmacotherapy is needed, lower doses should be used to start. Subclinical conduction abnormalities may be present (recognized on ECG or other studies), which might worsen with use of antiarrhythmics; such patients may require a pacemaker to allow the use of antiarrhythmics.

Key Points

- Palpitations are a frequent but relatively nonspecific symptom.
- Palpitations are not a reliable indicator of a significant arrhythmia, but palpitations in a patient with structural heart disease or an abnormal ECG may be a sign of a serious problem and warrant investigation.
- An ECG or other recording done during symptoms is invaluable; a normal ECG in a symptom-free interval does not rule out significant disease.

- Most antiarrhythmics themselves can cause arrhythmias.
- If in doubt about a rapid tachyarrhythmia in a patient in hemodynamic distress, cardiovert first and ask questions later.



Copyright © 2025 Merck & Co., Inc., Rahway, NJ, USA and its affiliates. All rights reserved.