SECTION IV

Cardiovascular

SYMPTOMS

MEDICAL DIAGNOSES

CHAPTER 38

Palpitations

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I. Definition (Clementy et al., 2018; Gale & Camm, 2016; Giada & Raviele, 2018)
   A. Subjective sensation described as heart skipping, fluttering, pounding, or racing
   B. Uncomfortable awareness of one's own heartbeat
   C. Disagreeable sensation of throbbing or movement in the chest or adjacent area
   D. Any rhythm that is not normal sinus rhythm with normal AV conduction

II. Physiology/Pathophysiology (Gale & Camm, 2016; Giada & Raviele, 2018; Wells & Tonkin, 2016; Wexler et al., 2017; Wilken, 2016)
   A. Normal: An electrical conduction system coordinates the muscular contractions of the cardiac cycle.
      1. An electrical impulse originating in the sinoatrial node of the right atrium stimulates each muscle contraction.
      2. The impulse travels through the atria to the AV node in the atrial septum down to the bundle of His and its branches, then Purkinje fibers, stimulating ventricular contraction.
      3. During sinus rhythm, the heart rate is in the normal range, the P waves are normal on ECG, and the rate is stable.
   B. Pathophysiology (see Table 38-1)

III. Clinical features: Clinical significance of palpitations is dependent on whether they are related to actual changes in cardiac function or an altered sensation of heartbeats with approximately 1% mortality. Up to 15%–22% of complaints never reach a confirmatory diagnosis because of intermittent nature. Palpitations are one of the most common problems of outpatients who seek medical attention. Usually benign, palpitations can occasionally be a manifestation of potentially life-threatening arrhythmia. Cardiac origin is present in 34%–43% of cases (Clementy et al., 2018; Gale & Camm, 2016; Giada & Raviele, 2018; Harskamp et al., 2017; Inayat et al., 2017; Matura et al., 2016; Miranda et al., 2018; Ruzieh et al., 2018; Tokuda et al., 2017; van der Wardt et al., 2017; Wexler et al., 2017; Wilken, 2016; Zhang et al., 2017).
   A. Etiology (see Table 38-1)
      1. Cardiac dysrhythmias causing palpitations occur as primary clinical conditions (e.g., Wolff-Parkinson-White syndrome) or secondary, induced by stressors on the heart (e.g., hypoxia, acidosis). Older adult patients are at greater risk of developing dysrhythmias and consequently palpitations.
         a) Rapid heart rates exceeding 150 bpm indicate dysrhythmias, such as sinus tachycardia, atrial flutter, atrial fibrillation, supraventricular tachycardia, or ventricular tachycardia. The sensation of palpitations may be related to the high heart rate or irregularity of the rhythm.

TABLE 38-1 Pathophysiology and Etiology of Palpitations

Pathophysiologic Mechanism	Etiology Examples
Autonomic dysregulation with altered blood flow and syncope/near syncope	Hypertension Musculoskeletal deformities (e.g., pectus excavatum, kyphosis) Orthostatic hypotension: neurologic disease, fibromyalgia, calcium channel blockers, withdrawn hypertensive Pheochromocytoma Retroperitoneal or pelvic disorders (e.g., endometriosis, hematoma, polycystic ovary syndrome) Temporal lobe seizures with orthostatic tachycardia Vasomotor syndrome
Direct cardiac stimulation	Cardiac injury/contusion, tumor involvement Fluid and electrolyte imbalances Hyperthyroidism Stimulant medications/drugs, alcohol, anticholinergics, amphetamines, cocaine, nicotine, caffeine Thiamine deficiency
Extra-cardiac stimulation of the vagus nerve	Acidosis Beta-blocker withdrawal Constipation Extreme nausea/vomiting
Hyperdynamic circulation or increased blood volume	Hypercapnia Hyperthermia Hyperthyroidism Paget disease Pregnancy Renal failure Valvular incompetence
Simulation of atrial irritability	Lung cancer Pleural effusions Pneumonia
Sudden changes in heart rate or rhythm or cardiac movement in the thorax	Atrial fibrillation Ectopic heartbeats; premature contractions (e.g., atrial, junctional, ventricular) Exercise Intermittent heart block/bradycardia, ventricular tachycardia
Sympathetic stimulation (adrenergic surge) with increased automaticity and contractility	Anemia Anxiety Heart failure Hypoglycemia Hypoxemia Panic attack Stimulant medications/recreational drugs

Note. Based on information from Gale & Camm, 2016; Giada & Raviele, 2018; Wells & Tonkin, 2016; Wexler et al., 2017; Wilken, 2016.

b) Bradycardic dysrhythmias, such as sinus bradycardia or heart block, cause compensatory hyperdynamic cardiac performance and palpitations. Although not exclusively associated with bradycardia, prolonged QT syndrome (congenital or disease or medication induced) can cause both bradycardia and irregular beats with palpitations.

c) Intermittent irregular heartbeats, such as premature atrial, junctional, or ventricular beats, and blocked beats, produce irregular cardiac output that can be sensed as palpitations. Slow ventricular response atrial fibrillation also has been associated with palpitations, likely due to its irregularity.

2. Structural heart abnormalities and acquired heart pathologies produce turbulent blood flow through the heart, causing the sensation of palpitations.
   a) Inflammatory/infectious heart disorders: Endocarditis, myocarditis
   b) Valvular disorders: Mitral valve (most common)
   c) Displaced devices or sensing wires: Pacemakers, implantable defibrillators
   d) Cardiomyopathy with or without accompanying dysrhythmias; more common with hypertrophic cardiomyopathy
   e) Malignancy involving the heart: Lymphoma, myxoma, sarcomas (e.g., angiosarcoma, Kaposi sarcoma, synovial sarcoma), hepatocellular cancer
   f) Rheumatoid conditions involving the heart: SLE, systemic scleroderma, amyloidosis, relapsing chondritis, advanced Sjögren syndrome
   g) PH
3. Blood volume variations
   a) Hypervolemia produces hyperdynamic cardiac performance that can cause palpitations, such as with heart failure, renal failure, SIADH, and pregnancy.
   b) Hypovolemia causes compensatory increased cardiac contractility that can cause palpitations, such as with adrenal insufficiency, nausea and vomiting, hyperglycemia, and hypercalcemia.
4. Autonomic dysregulation occurring as a result of a neurologic condition (e.g., neurologic orthostasis) or loss of vascular tone (e.g., sepsis) can cause a near-syncopal syndrome associated with palpitations.
5. Sleep deprivation causes heart rate variability that can be felt as palpitations.
6. Etiologies with poorly defined mechanisms
   a) Lactose or other food intolerances
   b) Narcolepsy
   c) Athletics: More common in older individuals practicing endurance sports
   d) Postconcussion orthostatic tachycardia

B. History: Palpitations may be the first or only manifestation of dysrhythmias. A thorough health history can reflect strong risk factors or other previously overlooked signs or symptoms.
   1. History of cancer and cancer treatment
   2. Current medications: Prescribed (e.g., antidepressant, weight loss medications, antiparkinsonism), over the counter (e.g., allergy/cold medications containing antihistamines), herbals/nutraceuticals
   3. History of presenting symptoms: Precipitating factors, onset, location, duration, prodromal or associated symptoms (e.g., dizziness, diaphoresis, nausea), alleviating factors (ask patient if coughing or Valsalva maneuver stops symptoms); symptoms lasting more than five minutes likely have a cardiac origin.
   4. Changes in ADLs: Interfering with self-care, influencing perception of QOL
   5. Past medical history: Heart defects, mitral valve prolapse, dysrhythmias, implanted cardiac devices, altered blood pressure, angina, MI, palpitations, rheumatic fever, endocarditis, anxiety or panic disorder, syncope, vertigo, seizures, anemia, nutritional changes, infection, electrolyte imbalances, endocrine dysfunction
   6. Family history: Heart disease, anemia, orthostasis
   7. Social history: Nicotine consumption (e.g., cigarettes, chewing tobacco, pipe smoking), illicit drug use (e.g., amphetamines, marijuana), caffeine intake, energy drink consumption

C. Signs and symptoms
   1. Sensations and awareness of heart beating, fluttering, skipping, or pounding
   2. Chest tightness or discomfort
   3. Shortness of breath
   4. Presyncope: Nausea, chest discomfort, headache, diaphoresis, palpitations, dyspnea, and paresthesias more likely associated with dysrhythmias
   5. Dizziness, vertigo
   6. Tinnitus
   7. Weakness
   8. Anxiety
   9. Pounding in neck

D. Physical examination: Focus on neurocardiac examination. Typically, the patient is not experiencing palpitations during the examination. An examination is useful in defining potential cardiovascular abnormalities and indications of dysrhythmia.
   1. Cardiac
      a) Apical heart rate: Fast or irregular
      b) Pulse deficit: Difference in the rate between the apical and radial pulse (radial lower); more indicative of premature beats than a rapid pulse
      c) Pulsus alternans or other irregularity: Variable peripheral pulses indicative of low or inconsistent cardiac output; may indicate heart failure
      d) Heart sounds: Possible gallops or murmurs present in valvular conditions, septal defects, heart failure, MI, pregnancy, or hyperthyroidism
      e) JVD/pulsations: Evidence of heart failure with venous congestion
      f) Peripheral pulse: Increased or decreased heart rate, irregular heart rhythm
      g) Orthostatic heart rate or blood pressure: Heart rate greater than 20 bpm higher value when sitting than when lying down; blood pressure greater than 20 mm Hg lower value when sitting than when lying down
   2. Neurologic: Sensory perceptual difficulties exacerbated when sitting or standing (e.g., vertigo, dizziness, tinnitus, presyncope)
      a) Observe patient standing up straight after bending over. This may elicit sensation if related to dysrhythmia.
      b) Observe patient lying down. Sensation may end if related to dysrhythmia.

IV. Diagnostic tests (Abi Khalil et al., 2017; Giada & Raviele, 2018; Quan, 2019; Reed et al., 2019; Sakhi et al., 2019; Wexler et al., 2017)
   A. Laboratory
      1. CBC to identify anemia and assess blood volume
      2. Blood chemistry
         a) Electrolytes to evaluate fluid and electrolyte imbalances that can cause dysrhythmias and to assess blood volume status
         b) BUN and creatinine to assess for kidney disease, risk for fluid excess
      3. Thyroid function tests: Free T_4 (thyroxin) and TSH to assess for thyroid disorders
      4. Cosyntropin test to evaluate for adrenal insufficiency
      5. Blood cultures to assess for sepsis, as indicated
      6. Blood levels of medications known to affect heart rate and rhythm, as indicated
   B. Radiology: Usually not indicated
   C. Other
      1. ECG rhythm strip in leads II, aVF, V_1, and 12-lead ECG: Clearly defines a particular portion of the P, QRS, and T waves, intervals, and their directional deflection

that helps to discern one rhythm disturbance from another; usually initial test to assess for clear cardiac pathology
   a) Prolonged QT increases risk for lethal ventricular dysrhythmia.
   b) Shortened PR suggests risk for re-entrant tachycardia.
   c) If the rhythm is not always present, an exercise stress test may be performed in an attempt to trigger the rhythm disturbance. If triggered by exercise, this test has increased value.
2. Echocardiogram may be used to identify clinical effects of a dysrhythmia, presence of valvular disease, presence of cardiomyopathy, or heart failure.
3. 24-hour Holter monitor provides 24 hours of continuous rhythm monitoring, which is particularly helpful if rhythm disturbance is intermittent. The patient also keeps a diary that will aid in identifying clinical symptoms directly related to rhythm disturbance. This test is used when patients describe palpitations and cardiac rhythm disturbances cannot be confirmed.
4. Event monitoring may be used with Holter monitoring to reduce the amount of tape that must be processed. Event monitoring only records when certain cardiac rhythm criteria are met or when the patient triggers a record button. Some event monitoring is linked to a central monitoring facility that can prompt emergency interventions PRN. Newer smartphone-operated single lead rhythm documentation may be a valuable first assessment.
5. Implantable loop recorders have proven helpful in diagnosing the etiology of intermittent dysrhythmias when Holter monitoring has not been definitive. Implanted recorders can run for extended periods, enhancing the potential for dysrhythmia detection.
6. Electrophysiologic testing is used for life-threatening dysrhythmias and enables the physician to map the source of dysrhythmia. It also provides an avenue for direct destruction of the dysfunctional cell pathway.
   a) Testing can be helpful for establishing the origin, signs, and symptoms when the patient has multiple dysrhythmias.
   b) Ablative therapy (e.g., laser, electrocautery, cryotherapy) can provide an alternative to medications.
7. Tilt table test is performed when palpitations are accompanied by dizziness or syncope. This test is a more accurate method of diagnosing orthostasis than bedside evaluation (see Appendix D).

V. Differential diagnosis (Aşkın et al., 2017; Wilken, 2016)
   A. Adrenal disease
   B. Aortic dissection type A, aneurysm
   C. Cardiac valve disease
   D. CAD: Angina, unstable angina, MI (see Chapters 40 and 49)
   E. Cardiomyopathy: Dilated, hypertrophic (Chapter 41)
   F. Endocarditis (see Chapter 46)
   G. FMS (see Chapter 101)
   H. Fluid overload
   I. Heart tumors
   J. Hypertensive urgency
   K. Hypoxia/respiratory failure
   L. Malignancy involving heart: Myxoma, hemangioma, lymphoma
   M. Rheumatoid condition involving heart: Autoimmune myocarditis, sarcoid, scleroderma, SLE
   N. Congenital conduction abnormalities: Wolff-Parkinson-White syndrome, Brugada syndrome
   O. PE (see Chapter 33)

P. Hyperthyroidism (see Chapter 157)
Q. Electrolyte imbalances (see Chapters 152–156)

VI. Treatment: Dependent on the etiologic factors, patient's tolerance, and planned treatment goals (Barley & Lawson, 2016; Giada & Raviele, 2018; Wells & Tonkin, 2016; Wexler et al., 2017; Wilken, 2016)
A. Do not treat palpitations if all of the following are true.
1. The cause of rhythm is known and treatable.
2. Significant clinical symptoms are absent, signaling cardiac instability.
3. Rhythm is unlikely to disintegrate into a life-threatening and unstable rhythm.
B. Low-risk features to treat and evaluate may occur over time.
1. Isolated infrequent palpitations not associated with exercise or waking from sleep
2. Absence of syncope
3. Normal ECG
C. Emergency management of palpitations
1. Provide supplemental oxygen and IV fluids if not contraindicated. Many etiologies of palpitations respond well to one of these general therapies.
2. Determine the patient's cardiac instability and need for emergency medical care (e.g., chest pain, hypotension, dyspnea, hypoxemia, mental status changes).
3. If associated with rapid pulse and heart rate, have patient perform a vagal maneuver.
D. Correct underlying etiology or risk factors, if known.
1. Implant devices such as pacemakers to assess function or control dysrhythmia.
2. Discontinue medications that may increase risk of dysrhythmias.
3. Eliminate nicotine. Alcohol and caffeine should be consumed in moderation.
4. Regulate metabolic rate. Control fevers and other factors that increase metabolic rate (e.g., stress, pain).
5. Correct vascular volume disturbances. Administer fluids or diuretics.
6. Correct electrolyte imbalances (see Chapters 152–156) or nutritional deficits.
7. Supportive therapy for orthostatic hypotension may include increased salt intake or compression wrapping of the lower legs.
8. Treat dysrhythmias according to defined standards (see Chapter 45).
9. Treat endocarditis (see Chapter 46) or primary cardiac diseases, such as hypertension (see Chapter 47), MI (see Chapter 49), cardiomyopathy (see Chapter 41), and heart failure (see Chapter 42), according to defined standards.
E. Evidence-based principles for care
1. Recommendations are all consensus based.
2. All patients are evaluated for ischemic etiology.
3. Nonspecific ECG abnormalities should not be considered normal and warrant investigation.
4. Syncope or autonomic dysfunction warrants tilt-table testing.
5. Evidence of heart failure or structural disease warrants echocardiogram evaluation.
F. Supportive care
1. Assist the patient in identifying triggers for symptoms and altering lifestyle (e.g., rest patterns; consumption of alcohol, caffeine, or nicotine) to reduce risk.
2. Provide patient education regarding orthostasis management or administration of antidysrhythmic medications.
3. Herbal agents are believed to abrogate palpitations (e.g., thiamine, coenzyme Q).
4. Perform safety assessment of the patient and living circumstances based on severity of symptoms and probable etiology or risk of serious adverse effects. Act on this safety assessment to modify living conditions or supervision of ADLs.

VII. Follow-up (Gale & Camm, 2016)
   A. Patients with untreated palpitations that resolve spontaneously may not need formalized follow-up.
   B. Patients should avoid cardiac stimulants (e.g., caffeine, alcohol, tobacco, antihistamines) indefinitely.
   C. Patients with high-risk features associated with syncope should be evaluated promptly by a cardiologist.
   D. Patients requiring medical treatment for dysrhythmias may have routine laboratory and specific cardiac tests performed on a periodic basis depending on the type and etiology of the dysrhythmia.
      1. Management of the dysrhythmias may require an electrophysiologist or cardiologist.
      2. Antiarrhythmic medication serum levels may need to be monitored.
      3. Electrolyte levels should be maintained at high-normal when the patient has experienced palpitations or is at risk for dysrhythmias (e.g., potassium greater than 4 mEq/L, magnesium greater than 2 mEq/L).

VIII. Referrals (Barley & Lawson, 2016; Owens et al., 2016)
   A. Cardiologist: For evaluation of dysrhythmias of unknown origin or when refractory to treatment; symptoms and specific diagnosis will determine whether to consult a dysrhythmia specialist or interventional cardiologist.
   B. Neurologist: For assessment and management of syncope or orthostasis
   C. Pharmacist: To advise patients on the correct administration of antidysrhythmics; many of these agents have altered absorption with food, pH levels, or other concomitant medications.
   D. Dietitian: To assist the patient with revising eating habits to decrease the risk of dysrhythmias (e.g., avoiding caffeine), supplementing electrolytes, or altering salt intake (more if orthostasis, less if hypervolemic)
   E. Psychologist or psychology support professional: For cognitive behavioral approaches to symptom distress management, which have been effective for treatment of nonemergent etiologies of palpitations
   F. Tobacco cessation or alcohol rehabilitation programs, if appropriate

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