an increased incidence of high-grade atrioventricular block and ventricular arrhythmias. Unlike in acute myocarditis, aggressive immunosuppressive therapy has some benefit and should be initiated in these patients. For this reason, patients with acute heart failure unresponsive to usual care or with accompanying arrhythmias should undergo endomyocardial biopsy for diagnosis. Initial biopsy findings may be negative because of the patchy nature of the inflammation. Patients with giant cell myocarditis often require percutaneous or surgical ventricular support until they recover or need heart transplantation or LVAD placement. If giant cell myocarditis is suspected, prompt transfer to a hospital equipped with mechanical support should be considered because patients can progress from feeling well to moribund within hours.

Tachycardia-Mediated Cardiomyopathy

Tachycardia-mediated cardiomyopathy has been associated with both supraventricular and ventricular arrhythmias. Reversible causes of tachycardia, such as hyperthyroidism, should be ruled out. Importantly, heart rate control improves left ventricular function in these patients. The primary treatments are medications, such as β-blockers, and catheter-directed ablation. In patients with atrial fibrillation associated with rapid ventricular response, there is no evidence that converting to sinus rhythm is more efficacious than controlling the heart rate. In patients with ventricular arrhythmias or frequent premature ventricular contractions, cardiomyopathy is generally thought to develop when the burden of premature ventricular contractions is more than 10,000/day; ablation, especially if the premature ventricular contractions are unifocal, should be considered.

Arrhythmias

Introduction

Arrhythmias are traditionally categorized as supraventricular or ventricular based upon simple electrocardiographic (ECG) findings. Supraventricular arrhythmias originate from the atrium or atrioventricular (AV) node and are characterized by normal-appearing QRS complexes unless complicated by an aberrant ventricular condition. Ventricular arrhythmias originate below the AV node and are characterized by abnormal-appearing and prolonged QRS complexes. Disruptions in rhythm and rate occur in seven basic patterns: early beats, bigeminal beats, grouped beats, pauses, bradycardia, tachycardia, and chaotic rhythms. This section provides an approach to bradycardia and tachycardia and discusses the diagnosis and management of specific rhythm disorders.

Approach to Bradycardia

Clinical Presentation and Evaluation

Bradycardia (heart rate <60/min) may be asymptomatic or associated with symptoms of lightheadedness, syncope, exertional intolerance, dyspnea, and fatigue. It can result from disease in the sinus node, AV node, or His-Purkinje system, or from dysfunction of the autonomic system.

Diagnostic evaluation consists of a thorough history, physical examination, focused laboratory testing (electrolyte levels, thyroid function testing), and resting 12-lead ECG. It is important to identify severe or unstable conduction abnormalities that require urgent intervention. The evaluation should also include investigation for extrinsic and reversible causes of bradycardia, including ischemia, myocarditis, endocarditis, hypothyroidism, electrolyte disturbances, and medication use (especially β-blockers and digoxin). Clues from the history and physical examination may suggest Lyme disease, elevated intracranial pressure, or typhoid as other potential causes of bradycardia. Additional testing may include exercise treadmill testing to assess chronotropic competence and ambulatory ECG monitoring (see Diagnostic Testing in Cardiology).

Sinus Bradycardia

Sinus bradycardia is defined as the presence of sinus rhythm with a heart rate below 60/min. Sinus bradycardia may be appropriate in trained athletes and during sleep. Inappropriate or pathologic sinus bradycardia is most commonly caused by sinus node dysfunction due to age-related myocardial fibrosis. Less commonly, sinus node dysfunction may result from right coronary ischemia; intracranial hypertension; postoperative scarring or fibrosis from cardiothoracic surgery; or infiltrative or inflammatory disorders, such as sarcoidosis. The most common extrinsic cause is medication use.

Atrioventricular Block

AV block may be classified as first degree, second degree, or third degree. First-degree AV block is defined by a delay in AV conduction (PR interval >200 ms). In large cohort studies, first-degree AV block has been associated with an increased risk for atrial fibrillation and all-cause mortality.

In second-degree AV block, only some P waves conduct to the ventricles. Mobitz type 1 second-degree (Wenckebach) AV block is characterized electrocardiographically by a PR interval that progressively prolongs until a beat is dropped (**Figure 10**). Mobitz type 2 second-degree AV block is typified by ECG findings of grouped beating and progressive shortening of the RR intervals (**Figure 11**). The PR interval does not lengthen in Mobitz type 2 before nonconducted atrial beats. When 2:1 block is present, the Mobitz type cannot be determined. Mobitz type 2 AV block usually occurs below the AV node and has a higher risk for progression to complete heart block.

In third-degree AV block, also termed complete heart block, no P waves conduct to the ventricles. AV dissociation is observed on the ECG (**Figure 12**).

Treatment

In patients with symptomatic bradycardia and hemodynamic distress, atropine should be administered. If atropine is

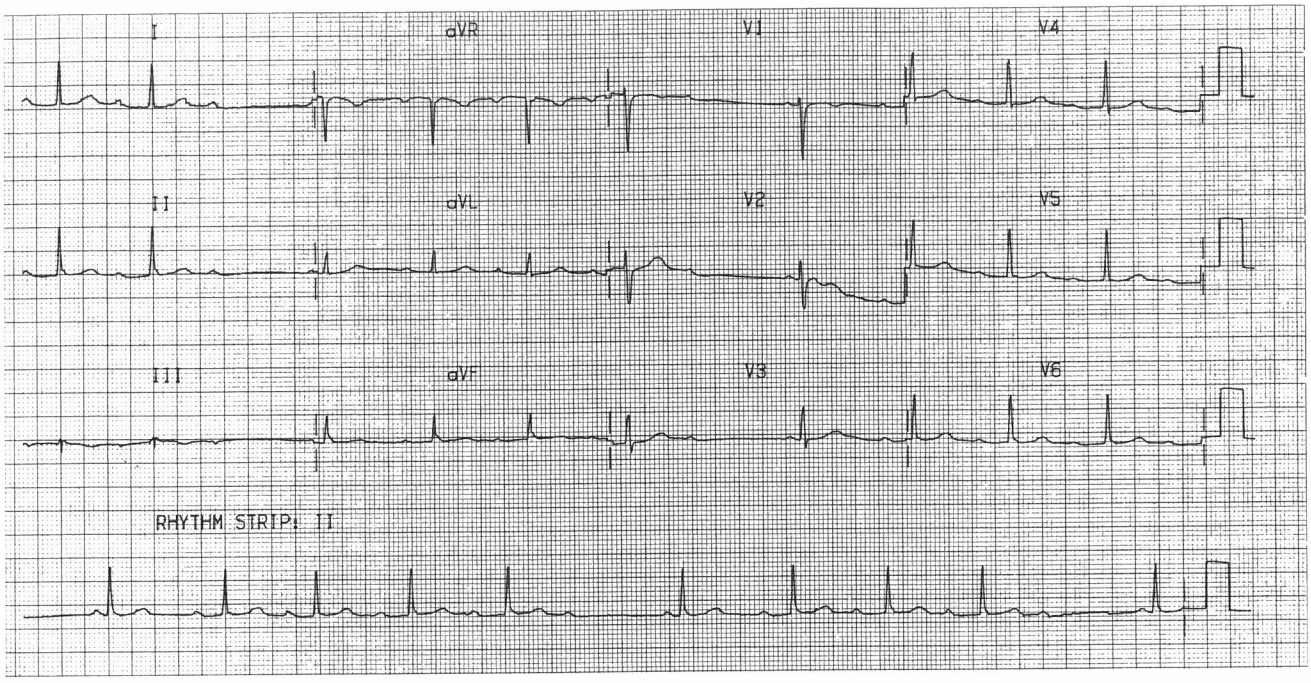

FIGURE 10. Electrocardiogram showing Mobitz type 1 second-degree atrioventricular block (Wenckebach block), which manifests as a progressive prolongation of the PR interval until there is a dropped ventricular beat.

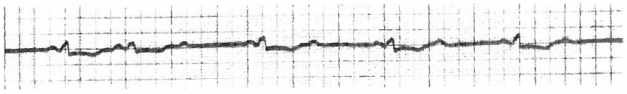

FIGURE 11. Electrocardiogram showing Mobitz type 2 second-degree atrioventricular block. P waves are blocked intermittently, and the PR interval is fixed. Note the wide QRS complexes, which are also more consistent with block below the compact atrioventricular node.

H CONT. ineffective, dopamine or epinephrine infusions can be given until transcutaneous pacing or a temporary pacing wire (preferred) can be placed. Temporary pacing is indicated in cases of hemodynamically unstable bradycardia or asystole. In some unique circumstances, prophylactic temporary pacing may be considered, including in patients undergoing transcatheter aortic valve replacement with high-risk features for heart block (such as right bundle branch block).

In hemodynamically stable patients, reversible and extrinsic causes of bradycardia should always be addressed before more invasive measures, such as permanent pacing, are considered. Common indications for permanent pacing include the following:

- Symptomatic bradycardia without reversible cause
- Asymptomatic bradycardia with significant pauses (>3 seconds in sinus rhythm) or heart rate less than 40/min
- Atrial fibrillation with pauses of 5 seconds or longer
- Alternating bundle branch block
- Asymptomatic complete heart block or Mobitz type 2 second-degree AV block

The various types of implanted cardiac electronic devices, their functions, and their general indications are reviewed in **Table 16**. Patients with left bundle branch block or right bundle branch block with or without a prolonged PR interval do not require permanent pacing because intraventricular conduction delays have a low risk for progressing to complete heart block (1%–3% per year). H

KEY POINT

- Permanent pacing is indicated for symptomatic bradycardia with no underlying reversible cause and in asymptomatic patients who have atrioventricular and infranodal conduction disturbances that have a high risk for progressing to complete heart block or asystole.

Approach to the Patient with Tachycardia

Clinical Presentation and Evaluation

Patients with tachycardia (heart rate >100/min) may be asymptomatic or experience tachypalpitations, a sensation of skipped beats, lightheadedness or dizziness, chest discomfort, dyspnea, exertional intolerance, fatigue, progressive heart failure, near-syncope, or syncope. In asymptomatic patients, tachycardia may be discovered incidentally during routine ECG, monitoring in the setting of hospitalization, or other medical care.

ECG documentation of tachycardia and correlation with symptoms is the key component of the diagnostic evaluation.

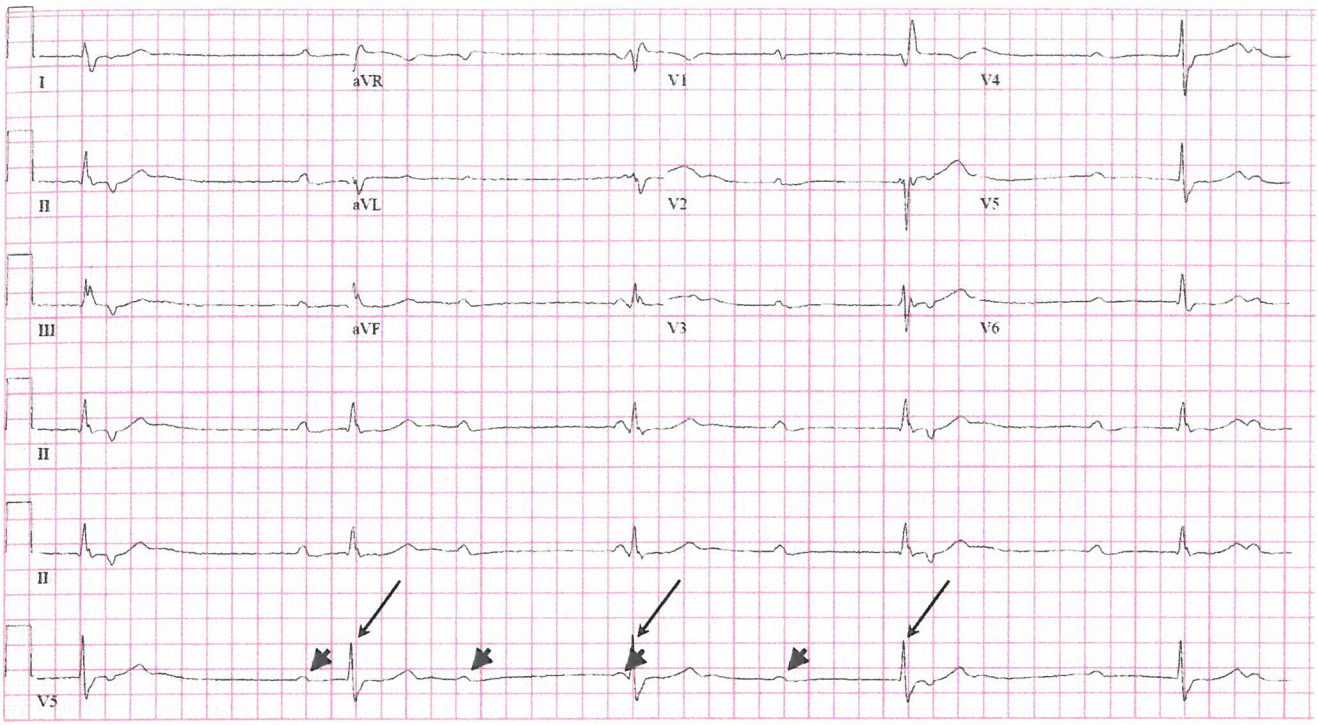

FIGURE 12. In this electrocardiogram, the P waves (*short arrows*) and the QRS complexes (*long arrows*) are not associated with each other, indicating the presence of complete heart block.

TABLE 16. Cardiac Implantable Electronic Devices for Treatment of Cardiac Rhythm Disorders

			Functions		
Device	**Components**	**Indications**	**Pacemaker Function**	**Antitachycardia Pacing**	**Defibrillation**
Transvenous pacemaker	Pulse generator and intravascular leads (single- or dual-chamber)	Sinus node dysfunction, atrioventricular block, nonreversible symptomatic bradycardia	Yes	No	No
Leadless pacemaker	Pulse generator with tines implanted directly into the cardiac chamber; no leads	Atrial fibrillation with bradycardia, paroxysmal sinus node dysfunction (e.g., brief sinus pauses)	Yes (ventricular sensing and pacing only)	No	No
Implantable cardioverter-defibrillator	Defibrillator and intravascular leads (single- or dual-chamber)	Monitoring and treatment of ventricular arrhythmias	Yes	Yes	Yes
Subcutaneous implantable cardioverter-defibrillator	Defibrillator and a single lead that are entirely under the skin (extravascular); no transvenous leads	Monitoring and treatment of ventricular arrhythmias	No	No	Yes
Cardiac resynchronization therapy-pacing (CRT-P)	Pulse generator and intravascular leads, including a pacing lead in the coronary sinus to pace the left ventricle	Restoring electrical synchrony in patients with symptomatic heart failure (left ventricular ejection fraction ≤35% and left bundle branch block)	Yes	No	No
Cardiac resynchronization therapy-defibrillator (CRT-D)	Defibrillator and intravascular leads, including a pacing lead in the coronary sinus to pace the left ventricle	Restoring electrical synchrony between the ventricles in patients with heart failure; monitoring and treating ventricular arrhythmias	Yes	Yes	Yes

After a thorough history and physical examination, a 12-lead ECG should be obtained in all patients with stable tachycardia. A 12-lead ECG recorded during symptoms, although often not possible to obtain, is far superior to most forms of ambulatory monitoring in terms of diagnostic value (see Diagnostic Testing in Cardiology for strategies for selecting an appropriate monitoring device). Thyroid function testing and echocardiography may be considered in selected patients with tachycardia.

Sinus Tachycardia

Sinus tachycardia (sinus rhythm with a heart rate >100/min) is the most common tachycardia and is typically the result of physiologic demand or distress, including exercise, pain, fever, anemia, or anxiety. Diagnostic evaluation and treatment are guided by the underlying cause.

Inappropriate sinus tachycardia (IST) is a disorder characterized by an elevated resting heart rate, with exaggerated increases in heart rate with light activity. The sinus rate typically decreases during sleep, which can be documented with ambulatory ECG monitoring. IST frequently presents in women in their second to fourth decades and appears to be more common in health care professionals. Symptoms vary and can include palpitations, lightheadedness, syncope (or near-syncope), dyspnea, and fatigue. The diagnosis of IST is based on the exclusion of other causes of tachycardia, such as hyperthyroidism, anemia, pheochromocytoma, and structural heart disease. First-line therapy is removal of aggravating factors and exercise therapy. In patients with bothersome and persistent symptoms, pharmacologic therapy with β-blockers, calcium channel blockers, or ivabradine (in refractory cases) can be considered.

Postural orthostatic tachycardia syndrome (POTS) is another condition that often presents with tachycardia. POTS is a form of dysautonomia characterized by orthostatic intolerance and excessive tachycardia, particularly with standing. Diagnostic criteria for POTS include an increase in heart rate of 30/min or more, or an increase to greater than 120/min, within 10 minutes of standing. The diagnosis is often confirmed with tilt-table testing. Behavioral modification, compression stockings, exercise training, and increased fluid intake are important components of therapy. Medical therapy for POTS is highly variable and may include, but is not limited to, β-blockers, fludrocortisone, selective serotonin reuptake inhibitors, midodrine, and pyridostigmine.

Supraventricular Tachycardias

Paroxysmal supraventricular tachycardias (SVTs), including atrioventricular nodal reentrant tachycardia (AVNRT), accessory pathway–mediated tachycardias, and atrial tachycardia, are frequently the cause of palpitations in younger persons. The accessory pathway may result from anterograde conduction, manifesting as a delta wave on ECG or retrograde conduction (so-called concealed accessory pathway). Management of these arrhythmias is discussed later in this chapter.

Other Tachycardias

Older patients with palpitations are more likely to have atrial fibrillation, atrial flutter, or ventricular tachycardia (VT), often due to underlying cardiovascular disease. VT is often associated with hemodynamic compromise; however, some VT can be well tolerated, particularly in patients with normal ventricular function. Evidence of hemodynamic compromise, including syncope, may also be present in patients with atrial tachyarrhythmias. For further discussion of the clinical presentation and management of these conditions, refer to the Atrial Fibrillation, Atrial Flutter, and Ventricular Arrhythmias sections later in the chapter.

KEY POINT

- Sinus tachycardia is the most common tachycardia and is typically the result of physiologic demand or distress, including exercise, pain, fever, anemia, or anxiety.

Antiarrhythmic Drugs

Antiarrhythmic agents are used to treat and suppress arrhythmias. These medications have traditionally been organized according to primary mechanism of action by using the Vaughan-Williams classification system (**Table 17**); however, most antiarrhythmic drugs exert their effects through several mechanisms.

Class I and class III antiarrhythmic drugs are the most effective antiarrhythmic drugs. Class IA agents are indicated for specific conditions (see Table 17). These medications have been associated with ventricular proarrhythmia, sudden death, and increased mortality in patients with coronary artery disease or structural heart disease. Class II agents (β-blockers) and class IV agents (nondihydropyridine calcium channel blockers) are commonly used to inhibit arrhythmia induction and AV conduction in patients with supraventricular or atrial arrhythmias. Class III agents sotalol and dofetilide are used to treat atrial and ventricular arrhythmias. Class III antiarrhythmic therapy should be initiated in an in-patient setting, with regular assessment of the corrected QT (QTc) interval. Ibutilide is an intravenous class III potassium channel blocker that is used for pharmacologic cardioversion of atrial fibrillation.

Amiodarone is among the most effective and commonly used antiarrhythmic drugs. This multichannel blocker is frequently used to treat patients with recurrent VT or atrial fibrillation. Amiodarone has no significant risk for proarrhythmia but is associated with thyroid, liver, lung, and eye toxicities, as well as neurologic side effects. Monitoring thyroid and liver function every 6 months is recommended in patients receiving amiodarone. Patients should also undergo annual pulmonary function testing and ophthalmologic examination. Amiodarone interacts with many drugs, including warfarin, statins, and digoxin. Dronedarone, another multichannel blocker, can be used in patients with intermittent atrial fibrillation and no overt heart failure.

TABLE 17. Antiarrhythmic Medications

Classification	Mechanism of Action	Individual Agents/ Examples	Effects	Use	Side Effects	Contraindications
Class IA	Sodium channel blockade with some potassium channel blockade	Quinidine, procainamide, disopyramide	Decreases speed of depolarization and prolongs repolarization	Preexcited atrial fibrillation (procainamide), Brugada syndrome (quinidine), SVT, atrial fibrillation, ventricular arrhythmias	Anticholinergic effects, including increased heart rate, dry mouth, urinary retention, blurry vision, and constipation Lupus-like syndrome (procainamide)	Ischemic or structural heart disease, second- or third-degree AV block without a pacemaker, prolonged QT interval, advanced kidney impairment
Class IB	Sodium channel blockade	Lidocaine, mexiletine, phenytoin	Decreases speed of depolarization	Ventricular arrhythmias	Headache, dizziness, or other neurologic symptoms Seizures (lidocaine toxicity)	Advanced liver disease
Class IC	Sodium channel blockade	Flecainide, propafenone	Decreases speed of depolarization and shortens repolarization	Atrial fibrillation, SVT, ventricular arrhythmias	Headache, dizziness, or other neurologic symptoms	Ischemic or structural heart disease, sinus node dysfunction, second- or third-degree AV block or bundle branch disease without a pacemaker
Class II	β-Adrenergic blockade	Metoprolol, propranolol, carvedilol, atenolol, bisoprolol	Decreases sympathetic tone; suppresses automaticity, sinoatrial conduction, and AV conduction	Rate control of atrial arrhythmias, SVT, ventricular arrhythmias	Fatigue, drowsiness, dizziness, hair loss, cold hands and feet, depression, erectile dysfunction, bronchospasm	Severe asthma, cardiogenic shock, second- or third-degree AV block, preexcitation
Class III	Potassium channel blockade	Sotalol, dofetilide, ibutilide	Prolongs action potential duration	Atrial fibrillation, atrial flutter, ventricular arrhythmias, pharmacologic cardioversion of atrial fibrillation (ibutilide)	Headache, dizziness, bradycardia, fatigue, dyspnea (sotalol) Headache, dizziness, diarrhea; rarely, torsades de pointes (dofetilide)	CrCl <40 mL/min/1.73 m^2, QTc interval >440 ms, sinus bradycardia <50/min, second- or third-degree AV block without a pacemaker
Class IV	Calcium channel blockade (nondihydropyridines)	Verapamil, diltiazem	Suppresses sinoatrial and AV conduction	SVT, rate control of atrial arrhythmias, triggered arrhythmias (outflow tract VTs)	Dizziness, constipation, dependent edema, nausea	Significant sinus node dysfunction, second- or third-degree AV block without a pacemaker, preexcitation
Multichannel blockers	Several mechanisms, including potassium, sodium, and calcium channel blockade	Amiodarone, dronedarone	Multiple mechanisms, although they act principally by extending repolarization	Atrial arrhythmias, ventricular arrhythmias	Fatigue, dizziness, nausea, vomiting, constipation or diarrhea, tremor	Advanced liver, lung, or thyroid disease (amiodarone) Advanced liver disease, permanent atrial fibrillation, recent decompensated or advanced heart failure (NYHA functional class III-IV) (dronedarone)

(Continued on the next page)

TABLE 17. Antiarrhythmic Medications *(Continued)*

Classification	Mechanism of Action	Individual Agents/ Examples	Effects	Use	Side Effects	Contraindications
Late sodium channel blockers	Late sodium channel blockade	Ranolazine	Shortens action potential duration and prevents calcium overload	Atrial fibrillation, ventricular arrhythmias	Dizziness, nausea, headache, constipation, hypoglycemia	Advanced liver disease, use of strong CYP3A4 inhibitors or inducers
Adenosine receptor agonists	A_1-receptor agonism	Adenosine	Slows or blocks sinoatrial and AV node conduction	Termination of SVT	Flushing, dyspnea, chest pain, hypotension, dizziness, nausea	Severe asthma, cardiac transplantation
Cardiac glycoside	Increases vagal activity	Digoxin	Slows AV node conduction	Rate control of atrial fibrillation	Nausea, vomiting, dizziness, blurry vision and yellow halos, thrombocytopenia	Advanced kidney impairment (requires dose adjustment)

AV = atrioventricular; CrCl = creatinine clearance; CYP3A4 = cytochrome P450 3A4; NYHA = New York Heart Association; QTc = corrected QT; SVT = supraventricular tachycardia; VT = ventricular tachycardia.

Ranolazine, digoxin, and adenosine are excluded from the Vaughan-Williams classification. Ranolazine is used to treat angina and decreases the risk for atrial fibrillation and ventricular arrhythmias. Digoxin is a positive inotropic agent that also increases vagal activity, leading to a lower resting heart rate. It can be used for rate control in patients with atrial fibrillation. Adenosine is used in the acute treatment of arrhythmias to interrupt AV conduction and terminate SVT. Administering adenosine can also help in determining the type of arrhythmia.

Atrial Fibrillation

Atrial fibrillation is defined by the presence of disorganized atrial activity with an irregularly irregular ventricular response on ECG (**Figure 13**). It is the most common

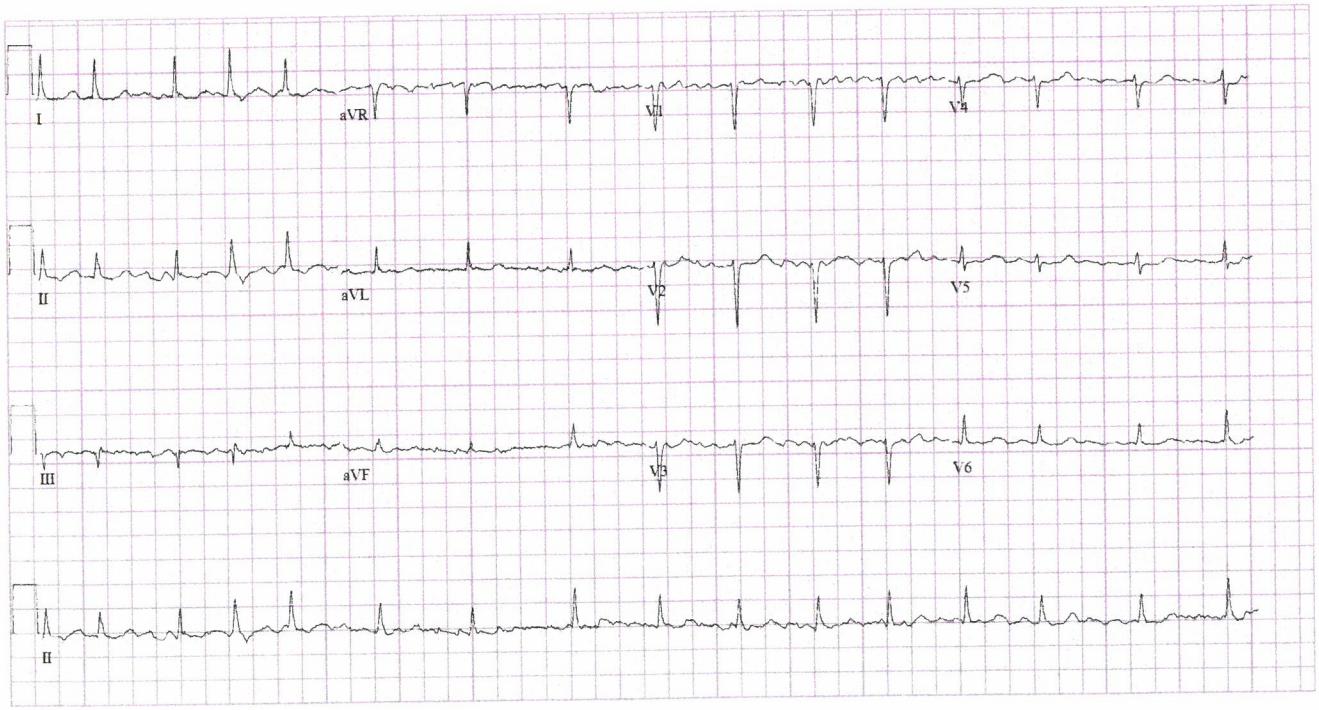

FIGURE 13. Electrocardiogram demonstrating atrial fibrillation. No clear P waves are seen, and the ventricular response is irregular.