

Understanding Cardiac Arrhythmias

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1 Introduction

Cardiac arrhythmias refer to disturbances in the normal electrical activity of the heart, leading to irregular heart rhythms. These abnormalities can present as an excessively slow heart rate (bradyarrhythmia), an excessively fast heart rate (tachyarrhythmia), or an irregular rhythm. While some arrhythmias may be benign and asymptomatic, others can cause significant morbidity and even be life-threatening. The ability to recognize and appropriately manage arrhythmias is a crucial skill in both general and specialized medical practice.

The study of cardiac arrhythmias is vital because they are prevalent in a variety of clinical settings, including emergency medicine, cardiology, and internal medicine. Arrhythmias can be triggered by various underlying conditions such as electrolyte imbalances, ischemic heart disease, structural abnormalities, and genetic predispositions. Understanding their mechanisms, clinical manifestations, and treatment options is essential for improving patient outcomes and preventing complications such as stroke, heart failure, and sudden cardiac death.

2 Cardiac Electrophysiology Overview

The heart's electrical activity is generated and propagated through a specialized conduction system that ensures synchronized contraction of the atria and ventricles. This system comprises the sinoatrial (SA) node, atrioventricular (AV) node, bundle of His, and Purkinje fibers. The SA node, located in the right atrium, serves as the primary pacemaker, generating impulses that travel through the atria to the AV node. The AV node functions as a gatekeeper, delaying impulse transmission to allow ventricular filling before propagation through the bundle of His and Purkinje fibers, ensuring efficient ventricular contraction.

Disruptions in this conduction system can lead to arrhythmias through various mechanisms, including enhanced automaticity, reentry circuits, and triggered activity. Enhanced automaticity occurs when non-pacemaker cells gain the ability to generate spontaneous action potentials, leading to ectopic beats. Reentry circuits, a common cause of tachyarrhythmias, result from abnormal looping of electrical impulses that continuously activate cardiac tissue. Triggered activity, such as early and delayed afterdepolarizations, arises from abnormal ion channel function, contributing to arrhythmogenesis.

The development of arrhythmias is influenced by intrinsic cardiac factors, systemic conditions, and external triggers. Structural heart disease, ischemia, electrolyte imbalances, and genetic mutations can predispose patients to arrhythmic events. Additionally, factors such as autonomic nervous system imbalances, drug effects, and metabolic abnormalities play a crucial role in modulating cardiac excitability and conduction. Understanding these electrophysiological principles is fundamental to diagnosing and managing various cardiac arrhythmias effectively.

3 Classification of Cardiac Arrhythmias

Cardiac arrhythmias can be classified based on their impact on heart rate, their site of origin, and their underlying electrophysiological mechanisms. This classification is essential for guiding diagnosis and treatment decisions.

Bradyarrhythmias, characterized by a heart rate of less than 60 beats per minute, typically result from impaired impulse generation in the sinoatrial node or blocked conduction through the atrioventricular node. Common causes include sinus bradycardia, where the SA node generates impulses at a slower rate, and various degrees of AV block, which interfere with impulse transmission to the ventricles. Junctional escape rhythms may also arise when the AV node or lower conduction system takes over as the heart's pacemaker due to failure of the SA node.

Tachyarrhythmias, defined by a heart rate greater than 100 beats per minute, are classified into supraventricular and ventricular arrhythmias based on their site of origin. Supraventricular tachyarrhythmias include atrial fibrillation, atrial flutter, and AV nodal reentrant tachycardia (AVNRT), which originate above the ventricles. These arrhythmias often result from reentry circuits within the atria or AV node. In contrast, ventricular arrhythmias, such as ventricular tachycardia and ventricular fibrillation, arise from the lower chambers of the heart and are more likely to cause hemodynamic instability, potentially leading to sudden cardiac arrest.

Based on Heart Rate

- Bradyarrhythmias (<60 bpm):
 - Sinus bradycardia
 - AV blocks (first, second, and third degree)

- Junctional escape rhythms
- Tachyarrhythmias (>100 bpm):
 - Atrial tachycardias (atrial fibrillation, atrial flutter, supraventricular tachycardia)
 - Ventricular tachycardias (ventricular tachycardia, ventricular fibrillation)

Based on Origin

- Supraventricular arrhythmias:
 - Sinus tachycardia, atrial fibrillation, atrial flutter
 - AV nodal reentrant tachycardia (AVNRT)
 - Wolff-Parkinson-White (WPW) syndrome
- Ventricular arrhythmias:
 - Premature ventricular contractions (PVCs)
 - Ventricular tachycardia (monomorphic/polymorphic)
 - Ventricular fibrillation
 - Torsades de Pointes (associated with prolonged QT interval)

4 Clinical Manifestations

The clinical manifestations of cardiac arrhythmias vary widely depending on the type, severity, and underlying cause of the rhythm disturbance. Some arrhythmias remain asymptomatic and are discovered incidentally during routine examinations, while others can cause debilitating symptoms or even life-threatening complications.

4.1 Symptoms

- Palpitations: A common symptom where patients describe sensations of rapid, irregular, or forceful heartbeats. These may occur in paroxysms or persist over time and are often associated with supraventricular or ventricular tachyarrhythmias.
- Dizziness and Syncope: Arrhythmias that significantly reduce cardiac output, such as bradyarrhythmias or ventricular tachycardia, can lead to inadequate cerebral perfusion, resulting in dizziness, near-syncope, or complete loss of consciousness.
- Fatigue and Weakness: Chronically slow heart rates or frequent ectopic beats can lead to reduced cardiac efficiency, causing patients to experience persistent fatigue and reduced exercise tolerance.
- Chest Pain and Dyspnea: Myocardial oxygen demand may exceed supply during episodes of tachyarrhythmias, especially in patients with underlying coronary artery disease, leading to angina-like chest pain and shortness of breath.
- Sudden Cardiac Arrest: Life-threatening arrhythmias such as ventricular fibrillation can cause an immediate cessation of effective cardiac output, leading to sudden cardiac arrest and death if not promptly treated.

4.2 Signs

- **Irregular Pulse:** A hallmark of atrial fibrillation and other irregular arrhythmias, an irregularly irregular pulse may be detected on physical examination.
- **Variable Blood Pressure:** Hypotension may occur with bradyarrhythmias or high-rate tachycardias, whereas hypertension can be seen in stress-induced arrhythmias.
- **ECG Abnormalities:** The electrocardiogram (ECG) provides crucial insights into arrhythmia diagnosis, revealing variations in P-wave morphology, PR interval abnormalities, widened QRS complexes, or prolonged QT intervals indicative of different arrhythmic conditions.

Recognition of these clinical features is critical in differentiating benign arrhythmias from those requiring urgent intervention. Early identification and appropriate management can prevent complications and improve patient outcomes.

5 Diagnosis of Cardiac Arrhythmias

The diagnosis of cardiac arrhythmias relies on a combination of clinical assessment, electrocardiographic evaluation, and advanced diagnostic tools. A thorough history and physical examination help identify potential triggers, risk factors, and associated symptoms, guiding further diagnostic testing.

Electrocardiography (ECG) Electrocardiography (ECG) is the cornerstone of arrhythmia diagnosis. A standard 12-lead ECG provides crucial information about heart rate, rhythm, and conduction abnormalities. Specific findings such as prolonged PR intervals, wide QRS complexes, or irregular R-R intervals help distinguish different types of arrhythmias. In cases where arrhythmias are intermittent, ambulatory ECG monitoring methods such as Holter monitoring (24-48 hours) or event recorders (used over weeks to months) can help capture transient episodes.

Electrophysiological Studies (EPS) In patients with unexplained syncope or persistent arrhythmias resistant to standard evaluation, an electrophysiological study (EPS) may be conducted. This invasive procedure involves inserting catheters into the heart to map electrical activity and identify abnormal conduction pathways. EPS is particularly useful in diagnosing reentrant arrhythmias and guiding catheter ablation therapy.

Laboratory and Imaging Tests Certain laboratory tests can assist in identifying underlying causes of arrhythmias. Electrolyte imbalances (such as hypokalemia or hyperkalemia), thyroid dysfunction, and cardiac enzyme elevations in myocardial infarction cases are commonly evaluated. Additionally, echocardiography and cardiac MRI can help assess structural abnormalities, valve diseases, and cardiomyopathies contributing to arrhythmic disorders.

Wearable and Implantable Monitors For patients with infrequent yet clinically significant arrhythmic events, long-term monitoring devices such as implantable loop recorders (ILRs) provide extended observation over months to years. These devices automatically detect and record abnormal rhythms, aiding in the diagnosis of cryptogenic arrhythmias or unexplained syncope.

A comprehensive approach combining clinical evaluation, ECG interpretation, and advanced diagnostic tools ensures accurate identification and appropriate management of

cardiac arrhythmias, improving patient outcomes and reducing associated morbidity and mortality.

6 Treatment Approaches

The treatment of cardiac arrhythmias is tailored to the type of arrhythmia, its severity, and the patient's overall clinical condition. Management strategies include pharmacological therapies, interventional procedures, and device-based treatments.

Pharmacological Treatment Antiarrhythmic medications are classified according to the Vaughan-Williams system, which categorizes drugs based on their effects on cardiac ion channels and conduction pathways:

- Class I (Sodium Channel Blockers): Used for rhythm control by reducing excitability (e.g., Flecainide, Lidocaine).
- Class II (Beta-Blockers): Slow heart rate and reduce sympathetic activity (e.g., Metoprolol, Propranolol).
- Class III (Potassium Channel Blockers): Prolong action potential duration to prevent reentry (e.g., Amiodarone, Sotalol).
- Class IV (Calcium Channel Blockers): Reduce AV nodal conduction (e.g., Verapamil, Diltiazem).
- Other agents: Digoxin (used for rate control in atrial fibrillation) and anticoagulants (for stroke prevention in atrial fibrillation).

Non-Pharmacological Interventions

- Electrical Cardioversion: Used for rapid restoration of sinus rhythm in atrial fibrillation and flutter.
- Catheter Ablation: A curative option for certain arrhythmias like AVNRT and atrial flutter, where targeted destruction of arrhythmogenic tissue prevents recurrence.
- Pacemakers: Implanted in patients with symptomatic bradyarrhythmias to maintain an adequate heart rate.
- Implantable Cardioverter Defibrillators (ICDs): Indicated for high-risk patients to prevent sudden cardiac death from ventricular tachyarrhythmias.

Timely intervention and appropriate selection of treatment modalities improve patient outcomes and prevent long-term complications associated with arrhythmias.

7 Special Considerations

- Arrhythmias in special populations:
 - Elderly (increased risk of atrial fibrillation, bradyarrhythmias)
 - Athletes (risk of sinus bradycardia, ventricular arrhythmias)
 - Pregnant women (hemodynamic changes affecting arrhythmia prevalence)
- Arrhythmias in structural heart disease:
 - Post-myocardial infarction (risk of ventricular arrhythmias)
 - Heart failure (association with atrial fibrillation and sudden cardiac death)

8 Summary and Take-home Messages

- Cardiac arrhythmias arise from disruptions in normal cardiac conduction.
- Diagnosis is primarily based on ECG findings and clinical correlation.
- Treatment strategies depend on the arrhythmia type, severity, and patient stability.
- Advanced therapies include catheter ablation, pacemakers, and ICDs for high-risk patients.