Cardiac arrhythmia is an abnormal heartbeat, either the heart is beating too fast, too slow, or in an irregular manner. A healthy heart beats in a coordinated and regular way. Electrical and chemical impulses develop into specialized cells. These impulses stimulate the heart muscle cells (myocytes) to contract. When the heart is irritated or damaged, these impulses can develop spontaneously in the atria or the ventricle. Some spontaneous contractions are normal, but certain contraction patterns are very dangerous and can result in permanent heart damage, stroke, or death.

Recap: A Healthy Heart

**Myocardial cells** are striated muscle cells that are similar to skeletal muscle. They differ because they have only one nucleus and many more mitochondria. These cells make up the walls of the atria and ventricles of the heart. They are electrochemically connected to adjacent cells by gap junctions and intercalated disks. Channels allow electrolytes such as sodium, potassium, and calcium to flow between cardiac cells and allow the heart to work in unison. In contrast, skeletal muscle cells lack intercalated discs and act individually.

**Pacemaker cells** are modified cardiomyocytes. There are many groups of pacemaker
cells throughout the heart. They have the ability to spontaneously develop a cardiac action potential, called automaticity. The electrical impulse is propagated throughout the heart by conductive fibers and travels from the sinoatrial (SA) node to the myocardial cells of the right and left atria, then through the atrioventricular (AV) node, to the His-Purkinje fiber system and into the left and right ventricles. The electrical impulse stimulates the myocardial cells to contract.

![Electrical conduction system of the heart](https://via.placeholder.com/150)

**Sinoatrial (SA) node**

In a healthy heart, electrical impulses start at the SA node, the primary node. It is located in the right atrium. The blood supply for the SA node comes from the right coronary artery in approximately 60% of the population and the left coronary artery in 40% of the population. An occlusion in the downstream coronary artery may result in damage to the node. The SA node spontaneously depolarizes approximately 100 times per minute.

Fibers of the autonomic nervous system connect to the SA node. Sympathetic stimulation (epinephrine, norepinephrine) increases the intrinsic depolarization rate while parasympathetic inhibition (acetylcholine) decreases this rate. Usually, the parasympathetic inhibition signal is the stronger of the two, resulting in a heart rate between 60-100 beats per minute. The depolarization impulse passes through the right and left atrium through internodal tracts of conducting fibers and myocardial muscle cells into the AV node that is located in the interatrial septum.

**Atrioventricular (AV) node**

At the AV node, the impulse pauses briefly due to slow conduction velocity. This gives the ventricle an opportunity to fill before contraction. The AV node blood supply comes from the right coronary artery in 80% of the population. The autonomic nervous system regulates the conduction velocity through the AV node. If the primary pacemaker
(the SA node) is defective or damaged, the AV node becomes the pacemaker. It spontaneously depolarizes 40-60 times per minute, but this impulse is usually overwhelmed by the electrical impulse from the SA node. The impulse continues from the AV node to the Bundle of His-Purkinje fibers.

Bundle of His and Purkinje fibers

The atria and ventricles are electronically isolated by the annulus fibrosus. The **Bundle of His** is the only **electrically conductive track** that connects the two sections of the heart. The Bundles of His travel down the interventricular septum. It separates into the left and right bundle branches located in the walls of the left or right ventricles. Its blood supply comes from the left anterior descending artery.

The impulse continues down left or right bundles into the Purkinje fibers, the terminal branch of the conduction system. The left branch depolarizes first, resulting in left ventricle contraction followed by the right ventricle contraction. The Bundle of His spontaneously depolarize at a rate of 30-40 times per minute, but the normal electrical impulse from the SA node usually overwhelms this signal.
Definition of Arrhythmia

An arrhythmia is an irregular heartbeat due to a disorder of impulse production, impulse conduction, and in some instances, both.

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Pathophysiology of Arrhythmias

Disorder of impulse production

The heart contains many pacemaker cells with the ability to produce an electrical impulse. Under normal circumstances, the SA node controls the rate of electrical impulse production. Occasionally, the other nodes (AV node, Bundle of His) generate an electrical impulse as well, resulting in brief disorganization in the carefully regulated contraction process of the heart. This produces an arrhythmia.

In a healthy heart, the automaticity of the pacemaker cells can be manipulated by the autonomic nervous system, electrolyte imbalances, and medications (beta-blockers, calcium channel blockers, and much more). The parasympathetic nervous system slows the heart by releasing acetylcholine at the SA node. The sympathetic nervous system increases the heart rate by releasing catecholamines (epinephrine and norepinephrine) at the SA node.

If the SA node degenerates and loses function, then the AV node will take over as a pacemaker, but at a much slower rate. Also, the myocytes, non-pacemaker muscle cells of the heart, can develop an electrical impulse that will stimulate the heart to contract. This is a rare and dangerous occurrence.

Disorder of impulse conduction

Normal conduction delay occurs at the AV node. The electrical impulse pauses for a brief moment, which allows the ventricles to fill with blood before continuing down the His-Purkinje system into the walls of the left and the right ventricles, stimulating contraction. Pathologic conduction delay can also occur due to medication (B-blockers, calcium channel blockers, etc.) or tissue damage. The conduction fibers can be damaged to the degree where no signal passes through, called a block.

Reentry is another disorder of impulse. There is a polarity in electrical impulse propagation and conduction. The electrical impulse moves down the heart as tissue depolarizes and enters a refractory period. However, if an isolated group of muscle tissue does not receive the electrical impulse and does not depolarize in a controlled manner, then they may depolarize spontaneously and activate surrounding tissue. This would result in an additional contraction and an arrhythmia.

A slow heart rate of fewer than 60 beats per minute (bpm), or bradycardia, results from a primary pacemaker dysfunction or a conduction block in the AV node or The His Bundles.

A fast heart rate of greater than 100 bpm, or tachycardia, results from an abnormal pacemaker or a reentry impulse.

Classification of Arrhythmias

There are many ways to classify arrhythmias. Here is a simplified method.

Premature beats

These arrhythmias are relatively common and usually benign and asymptomatic. Occasionally a patient will feel a palpitation or fluttering of the heart. Premature beats are
further divided into a premature atrial contraction that originates in the upper chamber and premature ventricular contractions in the lower chamber.

Supraventricular arrhythmia

These arrhythmias originate in the atria or AV node. These arrhythmias result in tachycardia. Supraventricular arrhythmias include atrial flutter, atrial fibrillation, multifocal atrial tachycardia, Wolff-Parkinson-White syndrome, and paroxysmal supraventricular tachycardia.

Ventricular arrhythmia

These arrhythmias are very dangerous and require immediate medical care. They originate in the ventricle and also result in tachycardia. Ventricular arrhythmias include Torsade de Pointes, ventricular fibrillation, and ventricular tachycardia.

Bradyarrhythmias

A slow heartbeat is known as bradycardia or bradyarrhythmia. This can occur in healthy individuals, such as professional athletes. Heart blocks can be placed in this category.

Symptoms of Arrhythmia

There are many different types of arrhythmias. Many arrhythmias are asymptomatic. However, palpitations, the awareness of an abnormal heartbeat, is the most common symptom of arrhythmias.

This can be sensed as a flutter or pressure in the chest. If arrhythmia results in heart dysfunction, the patient may experience dyspnea or syncope. Some arrhythmias are lethal within minutes, such as ventricular fibrillation.
Diagnosis of Arrhythmia

Auscultation can occasionally detect an arrhythmia. Most arrhythmias can be diagnosed based on **12 lead electrocardiogram** (ECG). A 12 lead ECG study is non-invasive but requires experience to interpret the result. It briefly measures the electrical signals the heart emits during the depolarization of the cardiac muscle. A **Holter monitor** (portable 12 lead ECG) is used to detect infrequent or occasional arrhythmias. It is worn for about 24 hours and constantly monitors the heart.

A few arrhythmias (such as first degree SA block) require an **electrophysiology study** that is much more invasive and involves sedation and placing a catheter into the heart, usually through the arteries of the groin. Some arrhythmias only occur when the patient is under stress. A **treadmill (or chemical) stress test** is required to provoke arrhythmia, which is then recorded on ECG.

Some arrhythmias are completely benign. Some arrhythmias are potentially lethal, and some require an immediate response to prevent death. **Atrial fibrillation**, a supraventricular arrhythmia, allows blood to stagnate in the heart and form clots. These clots could result in an embolic stroke. A transesophageal echocardiogram is required for any patient in atrial fibrillation to ensure clots have not formed during the arrhythmia and before returning the patient to conversion to normal sinus rhythm.

Ventricular fibrillation requires an immediate response, such as defibrillation, to prevent degeneration into asystole, cardiogenic shock, and death.

**References**


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