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Mechanisms, Pathogenesis, Prognosis and Modern Methods of Treatment of Cardiac Arrhythmias

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Annotation: A healthy heart beats in a regular, coordinated manner because electrical impulses in the heart are generated and propagated by myocytes with unique electrical properties that drive consistent and organized contractions of the myocardium. Disorders associated with rhythm and conduction disorders are caused by abnormal formation and/or conduction of these impulses.

Keywords: Heart rhythm, disorder, pathology, pathogenesis, prognosis and treatment.

Introduction: Any cardiac dysfunction, including. Congenital structural abnormalities (e.g., atrioventricular extrasystoles) or functional disorders (e.g., hereditary channelopathies) can cause arrhythmias. Systemic factors contributing to arrhythmias include: electrolyte imbalances (especially hypokalemia or hypomagnesemia), hypoxia, hormonal imbalances (e.g., hypothyroidism, hyperthyroidism), and adverse effects of drugs and toxins (e.g., alcohol, caffeine).

At the junction of the superior vena cava and the upper right atrium is a group of cells called the sinoatrial (SA) or sinus node; the SA node generates the initial electrical impulse of each normal heartbeat. The electrical impulse from these pacemaker cells spreads to neighboring cells, causing the heart chambers to contract in an orderly sequence.

Impulses propagate within the atrium to the atrioventricular (AV) junction via atrial internodal pathways and preferential conduction by unspecialized myocytes. The AV node is located on the right side of the interatrial septum. It has a slow conduction velocity and therefore there is a delay in the transmission of the impulse. The AV delay time affects the heart rate and is modulated by autonomic tone and circulating catecholamines to maximize cardiac output at any given time.

The atria are electrically isolated from the ventricles by a fibrous ring everywhere, except in the anterior septal region. Here is the bundle of His (a continuation of the AV node), which enters

the interventricular septum, where it divides into left and right bundles, which end with Purkinje fibers. The right bundle branch of His conducts impulses to the anterior and apical parts of the right ventricle. The left bundle branch runs along the left side of the interventricular septum. Its anterior part (left anterior branch) and posterior part (left posterior branch) stimulate the left parts of the interventricular septum, which are first activated. Thus, depolarization of the interventricular septum occurs from left to right, with simultaneous excitation of both ventricles from the endocardium to the epicardial surface (see figure Electrical axis of the heart).

The sinoatrial (sinus) node (1) initiates an electrical impulse that passes through the right and left atria (2) and causes them to contract. When the electrical impulse reaches the atrioventricular node (3), it is slightly delayed. The impulse then travels along the bundle of His (4), which divides into the right bundle branch (5), which goes to the right atrium, and the left bundle branch (5), which goes to the left ventricle. The impulse then travels through the ventricles, causing them to contract.

To understand the causes of arrhythmias, it is necessary to have an understanding of the normal physiology of the heart.

Research method and materials: The passage of ions through the myocyte cell membrane is regulated by special ion channels that cause cyclic depolarization and repolarization of the cell, which is called the action potential. The action potential of a working myocyte begins when the cell depolarizes from 90 mV to 50 mV below the diastolic transmembrane potential. At this threshold potential, fast voltage-gated sodium channels open, causing rapid depolarization by reducing the concentration gradient of sodium ions. Fast sodium channels are inactivated and sodium influx stops, but voltage-gated ion channels remain open, allowing calcium to enter through slow calcium channels (depolarization) and potassium to leave through potassium channels (repolarization).

Results: First, these two processes balance and maintain a positive transmembrane potential and prolong the plateau phase of the action potential. During this phase, calcium entering the cell is responsible for the electromechanical coupling and contraction of the myocyte. Eventually, calcium entry ceases and potassium efflux increases, which rapidly repolarizes the cell back to -90 mV. During depolarization, the cell becomes resistant to further depolarization. Further depolarization is not possible (in the absolute refractory period), and after partial but incomplete repolarization, further depolarization is possible but occurs slowly (relative refractory period).

Fast-acting tissues (working atrial and ventricular cardiomyocytes, His-Purkinje system) have a high density of fast sodium channels and are characterized by action potentials.

There is little or no spontaneous diastolic depolarization (and therefore the pacemaker is activated very slowly)

Very fast initial depolarization rate (and therefore fast conduction velocity)

Loss of refractoriness corresponds to repolarization (and therefore the ability to conduct repeated impulses at short refractory periods and high frequencies)

Slow-acting tissues (SA and AV nodes) have and are characterized by a low density of fast sodium channels and action potentials.

Faster spontaneous diastolic depolarization (and therefore faster pacemaker activity)

Slow initial rate of depolarization (and therefore slow conduction velocity)

Loss of delayed refractoriness after repolarization (and therefore long refractory periods and inability to conduct repetitive impulses at high frequencies)

Normally, the SA node has the fastest rate of spontaneous diastolic depolarization, so SA node cells generate spontaneous action potentials at a higher frequency than other cells. Thus, the SA node is the dominant automatic pacemaker in the normal heart. If impulses do not arise in the SA

node, cells with slightly lower automaticity (such as the AV node) take over the role of automatic pacemaker. Sympathetic stimulation increases the activity of pacemaker cells, while parasympathetic stimulation decreases it.

Sinus node cells have an inward sodium-potassium current, called the "strange current," mediated by cyclic nucleotide-activated hyperpolarization-activated channels (HCN channels), which explains their automaticity. Inhibition of this current prolongs the time required for the pacemaker cells to reach critical spontaneous depolarization, thereby reducing heart rate.

The sinus rhythm of the heart in adults at rest is usually between 60 and 100 beats per minute. A slower rhythm (sinus bradycardia) often occurs in young people, especially in athletes, and during sleep. An increase in heart rate (sinus tachycardia) occurs in cases of physical exertion, illness, or strong emotions (by stimulating the sympathetic nervous system and circulating catecholamines).

Typically, a significant daily decrease in heart rate occurs in the morning before waking up. This occurs when the tone of the vagus nerve changes and is especially common among healthy young people. A slight increase in heart rate during inspiration and a decrease in heart rate during expiration are considered a normal variant (respiratory arrhythmia). This variation in heart rate decreases, but does not completely disappear with age. Absolute regularity of sinus rhythm is pathological and occurs in vegetative denervation (for example, in the late stages of diabetes) or other heart diseases that are severe enough to reduce parasympathetic cardiac (vagal) tone and activate sympathetic tone. Thus, heart rate variability indicators are informative indicators of the general condition of the cardiovascular system.

The basic electrical activity of the heart is represented by the electrocardiogram (ECG - a diagram of the cardiac cycle, see figure), although it does not include enough tissue to detect depolarization of the SA node, AV node, and His-Purkinje node. The P wave reflects atrial depolarization. The QRS complex reflects ventricular depolarization, and the T wave reflects ventricular repolarization.

The PR interval (from the beginning of the P wave to the beginning of the QRS complex) reflects the time from the beginning of atrial activation to the beginning of ventricular activation. This interval reflects the slowing of impulse transmission in the AV node. The RR interval (the time between two QRS complexes) represents the ventricular rate. The QT interval (from the beginning of the QRS complex to the end of the T wave) indicates the duration of ventricular depolarization. The normal QT interval is slightly longer in women and even longer in patients with slower heart rates. The QT interval is corrected for the effect of heart rate (QTc). The most common formulas (all intervals in seconds): Rhythm disturbances are caused by disturbances in the formation and / or conduction of impulses.

Bradyarrhythmia results from a decrease in pacemaker function or conduction block, primarily in the AV node or His-Purkinje system.

Most tachyarrhythmias result from a reentrant mechanism; some result from the enhancement of normal or pathological mechanisms of automaticity.

Re-entrance - the circular propagation of an impulse along two interconnected paths with different conductivity properties and refractory periods (see "Mechanism of normal re-entrance").

Here, the mechanism of AV nodal tachycardia is used as an example. Two pathways connect the same points. Pathway A has slow conduction and a short refractory period. Pathway B conducts normally and has a longer refractory period.

I. Normally, the impulse propagates through the A and B pathways (1). Conduction through the A pathway is slower and the impulse arrives at the B pathway already depolarized and therefore refractory (2). The result is normal sinus rhythm.

II. The early impulse finds pathway B refractory and blocked, but can pass through pathway A because its refractory period is shorter. After reaching pathway 2, the impulse continues to travel back and forth up pathway B, where it is blocked by the difficult-to-process tissue until pathway 3. The result is an early supraventricular rhythm with a prolonged PR interval.

III. If the passage through pathway A is slow enough, the early impulse can propagate retrogradely through pathway B, where the refractory period has already ended. If the refractory period of pathway A has also ended, the impulse can re-enter pathway A and travel in a circle, sending an impulse to the ventricle (4) with each cycle and retrogradely to the atria (5), producing a persistent reentrant tachycardia.

The onset of tachycardia is preceded by a non-sinusoidal P wave (P') and a prolongation of AV conduction (long P`R interval).

Under certain conditions, usually due to atrial extrasystoles, reentry can lead to a continuous circulation of the front of the excitation wave, which leads to tachycardia (see “Onset of atrioventricular nodal reentry tachycardia "). The resistance of the tissues to further stimulation prevents the formation of reentry. However, the reentry phenomenon is facilitated by three conditions:

Reducing the refractory period of tissues (for example, when stimulating the sympathetic system)

Conduction pathway dilation (e.g., due to hypertrophy or abnormal conduction pathways)

Slowing of impulse conduction (e.g., during ischemia)

Rhythm and conduction disturbances may be asymptomatic or may cause palpitations (sensation of skipped beats or rapid and forceful heartbeats), hemodynamic symptoms (eg, shortness of breath, chest discomfort, presyncope, fainting), or cardiac arrest. Polyuria is sometimes caused by the release of atrial natriuretic peptide into the bloodstream during prolonged supraventricular tachycardia.

Palpation of the pulse and auscultation of the heart can determine the rate, regularity, or chaos of the ventricles. Palpation of pulse waves in the jugular veins can help diagnose AV block and tachyarrhythmias. For example, in complete AV block, when the AV valves close, the atria contract intermittently, causing large (fast) pulse waves to appear in the jugular veins. There are several other physical signs of arrhythmia.

History and physical examination can identify arrhythmias and suggest possible causes, but diagnosis of arrhythmia requires a 12-lead ECG, and the data obtained during the examination establish the relationship between symptoms and rhythm.

ECG is a systematic approach that allows for interval measurement and detection of subtle abnormalities. Main diagnostic features

Atrial activation rate and frequency

The rate and regularity of ventricular activation

The relationship between the two

Abnormal activation of atrial and ventricular contractions is classified as either regularly irregular or irregularly irregular (no pattern is discernible). Regular, short-term disruption of the regular rhythm (e.g., extrasystole).

A narrow QRS (< 0.12 seconds) indicates a supraventricular origin of the impulse (above the bundle of His).

A wide QRS complex (≥ 0.12 seconds) indicates a ventricular origin of the impulse (below the bundle of His) or a supraventricular rhythm conducted through an accessory conduction

pathway, as in WPW syndrome.

Bradyarrhythmias are characterized by a slowing of the ventricular rate (< 60 beats/min in adults). ECG diagnosis of bradyarrhythmias depends on the presence or absence of P waves, P wave morphology, and the relationship between P waves and QRS complexes.

a. Atrioventricular (AV) block is a partial or complete interruption of the conduction of impulses from the atria to the ventricles. There are 3 degrees of AV block: first, second, and third.

b. In first-degree AV block, a QRS complex is observed after each P wave, but the PR interval is > 0.2 s. First-degree AV block does not cause bradycardia by itself, but is often associated with other conditions.

c. In second-degree AV block, some normal P waves are accompanied by QRS complexes, and some are not. Bradycardia may or may not be present.

d. Third-degree AV block is indicated by a bradyarrhythmia with no correlation between P waves and QRS complexes and a greater number of P waves than QRS complexes; an alternating rhythm may be present

e. node with normal AV conduction (narrow QRS complex)

f. AV node with impaired conduction (wide QRS complex)

g. ventricle (wide QRS complex)

The absence of second- or third-degree AV block is indicated by a regular QRS bradyarrhythmia with a 1:1 ratio between P waves and QRS complexes. P waves preceding the QRS complex indicate sinus bradycardia (if the P waves are normal even in first-degree AV block) or sinus arrest with atrial escape bradycardia (if the P waves are abnormal).

Discussion: P waves following QRS complexes indicate cessation of sinus node activity with replacement by nodal or ventricular rhythm and retrograde atrial activation. Ventricular escape impulse results in a wide QRS complex; A nodal escape complex usually has a narrow QRS (or a bundle of bundles or a wide QRS with pre-excitation).

When the QRS rhythm is irregular, there are usually more P waves than QRS complexes; some P waves produce QRS complexes, while others do not (second-degree AV block). An irregular QRS rhythm with a 1:1 ratio between P waves and subsequent QRS complexes usually indicates sinus arrhythmia, with gradual acceleration and deceleration of the sinus rhythm (if the P waves are normal).

Pauses in the normal QRS rhythm can be caused by P wave block (an abnormal P wave is usually observed after a preceding T wave disturbance or a preceding T wave disturbance), sinoatrial or sinus output block, and 2nd degree AV block.

Tachyarrhythmia is characterized by an acceleration of the ventricular rate (> 100 beats per minute in adults at rest); Tachyarrhythmias can be divided into 4 groups, determined by QRS complexes:

Tachyarrhythmias with irregular narrow QRS complexes include the following 4 rhythms. Differentiation is based on the atrial waves on the ECG, which are best distinguished in the pauses between QRS complexes.

Summary: Atrial fibrillation (AF): Atrial signals on the ECG (usually best seen in lead V1) have a constant, irregular frequency and morphology (> 10 beats per minute).

a. Atrial flutter with variable AV conduction: regular, discrete, uniform atrial impulses (usually best seen in leads II, III, and aVF), without isoelectric intervals, usually > 250 beats/min.

b. True atrial tachycardia with variable AV conduction: regular, discrete, uniform, presence of abnormal atrial signals and isoelectric periods (usually at a rate of < 250 beats/min)

c. Multifocal atrial tachycardia: discrete P waves that vary from beat to beat and have at least 3 patterns

d. Tachyarrhythmias with irregular wide QRS complexes include

e. The above 4 atrial tachyarrhythmias have irregular narrow complexes that are accompanied by bundle branch block or ventricular pre-excitation syndrome.

f. Polymorphic ventricular tachycardia (VT)

g. Differentiation is based on the atrial ECG rhythm and the presence of polymorphic VT with a very high ventricular rate (> 250 beats/min).

h. Tachyarrhythmias with regular narrow QRS complexes include

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