SEVEN publicações acadêmicas
EM PARCERIA COM: IEMS UNIASSELVI

CARDIAC RHYTHM DISORDERS: MECHANISMS, ELECTROCARDIOGRAPHIC PATTERNS, DIAGNOSIS AND MANAGEMENT IN THE MAIN ARRHYTHMIAS

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ABSTRACT

The study analyzes heart rhythm disorders in emergencies, addressing pathophysiological mechanisms, electrocardiographic patterns, diagnosis and therapeutic conducts for tachy and bradyarrhythmias, aiming to improve the prognosis with precise interventions. The literature review, based on recent articles, highlights the importance of ECG and early identification to reduce complications.

Keywords: Cardiac Arrhythmias. Cardiogram.

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INTRODUCTION

As in many organs of the body, the heart has a unique and fundamental function. The cardiovascular system is exclusively responsible for conducting blood flow to the tissues for an adequate distribution of nutrients and oxygen. For this function to occur properly, there is a range of physiological mechanisms that need to be in harmony so that errors do not occur and that allow achieving the goal (DE ABREU et al., 2022).

For the goal to be achieved, it is necessary that the cardiac function is adequate, allowing an adequate stimulus and conduction of the impulse. Not only does the electrical function need to be adequate, but also the contractile mechanical function so that the blood flow is coordinated in the heart chambers in a cyclical and orderly manner (DE CASTRO LISBOA et al., 2025).

An inadequate functioning of the heart can be conditioned by mechanical situations such as valvular dysfunctions, dilated or hypertrophic cardiomyopathies or congenital alterations. There are also dysfunctions due to ischemic alterations such as acute myocardial infarction causing cell death, infectious alterations such as myocarditis and endocarditis. And electrical conduction alterations, which are the alterations responsible for cardiac arrhythmias (NAGATA et al., 2021).

Known as heart rhythm disorders, or arrhythmias, they represent a diverse group of changes in the heart's electrical activity that can compromise its hemodynamic function and oxygenation. These changes can manifest asymptomatically or cause significant symptoms, such as palpitations, dyspneas, chest pain, dizziness, syncope, and, in severe cases, cardiorespiratory arrest (SCARCELLA et al., 2023).

The correct recognition of the electrocardiographic patterns of arrhythmias and the main clinical complaints is essential for an accurate diagnosis and appropriate intervention, preventing fatal complications (CANNAVAN et al., 2021).

OBJECTIVE

The general objective of this study is to analyze heart rhythm disorders in emergency situations, addressing the main clinical indications, pathophysiological mechanisms, electrocardiographic patterns, diagnosis and the conducts used to provide a better conduct and favor the patient's prognosis with appropriate therapy. The aim is to provide a comprehensive and practical view for application in the clinical context.

The specific objectives

1. Determine the main mechanisms causing heart rhythm disorders
2. Describe your electrocardiographic patterns of the main types of arrhythmias

3. Address the diagnostic methods needed for the most common arrhythmias
4. Discuss clinical and therapeutic approaches for the management of tachyas and bradyarrhythmias.

METHODOLOGY

Considering that theoretical studies are an indispensable basis for field and laboratory research, we opted for conceptual deepening and search for official data on the object of study, allowing the knowledge of reality as well as the possibility of critical reflection on the subject within the scope of the Brazilian reality.

Based on the understanding of Creswell (2007) for whom the Literature Review is configured as a preliminary stage of scientific studies, then the research is a Literature Review in which articles published in the National Library of Medicine (Pubmed), Virtual Health Library (VHL), Web of Science, Lilacs and Capes Journals were used as the basis of the study by descriptors obtained by the Health Sciences Descriptors (DeCS) of the VHL.

This is a literature review of articles published in the National Library of Medicine (Pubmed), Virtual Health Library (VHL), Web of Science, Lilacs and Cape Journals.

For the selection of articles, the following steps were followed: (I) search for articles in the databases; (II) reading of titles and abstracts, with analysis according to the eligibility criteria and; (III) full-text analysis of the papers, including in the systematic review only those required by the inclusion criteria and did not meet any of the exclusion criteria.

The following inclusion criteria were selected: (1) studies involving the main mechanisms causing cardiac rhythm disorders; (2) studies that had as their object of study electrocardiographic patterns of the main types of arrhythmias, as well as their diagnostic methods and conducts; (3) articles published in the last 4 years. There were no restrictions on sample size or foreign language.

As exclusion criteria, articles were excluded that: (1) were published before 2020; (2) studied situations that do not include the management of patients with arrhythmias in emergency situations; (3) duplicates; (4) had no direct relationship with the diagnosis, electrocardiographic patterns, or management of the patient with arrhythmia in clinical practice.

DEVELOPMENT

Arrhythmia is a condition defined by alteration of rhythm and/or heart rate without other physiological justification, such as tachycardia with adrenergic discharge or in situations of physiological bradycardia such as when the patient is sleeping. Arrhythmias are interpreted by

the analysis of a single-lead electrocardiographic tracing. A relevant piece of data to interpret arrhythmias and define the conducts is the characterization of the severity of the arrhythmia in question, that is, to determine if this patient is stable or if it is an arrhythmia with patient instability, thus having to be managed in the emergency room immediately (CANNAVAN et al., 2021).

Heart rhythm disturbances can be classified into bradyarrhythmias and tachyarrhythmias, depending on the heart rate. Bradyarrhythmias represent arrhythmias with a frequency of less than 50 beats per minute, can be represented by sinoatrial nodal dysfunction and atrioventricular blocks, while tachyarrhythmias encompass supraventricular and ventricular tachycardias (DE SOUZA et al., 2023).

Tachyarrhythmias can be classified in 3 main ways, by their pathophysiological mechanism (abnormal automatism or reentry), by rhythm (irregular or regular), or by their QRS (narrow or wide) (ARCOVERDE FILHO et al., 2022).

Arrhythmias can be caused by alteration of cardiac automatism, by alteration of atrioventricular conduction, leading to an acceleration or deceleration of the automatic structures of the heart, such as the sinus node, in situations of sinus tachycardia or bradycardia, AV node or the myocardium itself, situations that would no longer be physiological conductions. Ectopic beats or depolarizations can also arise from atrial electrical disturbances, AV junction or ventricles, thus generating abnormal rhythms, such as atrial or ventricular tachycardia. through re-entry mechanisms or by a combination of these factors (DE ABREU et al., 2022).

Another pathophysiological mechanism is atrioventricular conduction disorders, which can be accelerated, as in Wolff-Parkinson-White Syndrome, or slow, as in Atrioventricular block. Another possible cause is the combination of the two mechanisms, which can generate alterations such as atrial extrasystole with Atrioventricular block or atrial tachycardia with 3:1 AV block (SCARCELLA et al., 2023).

Therefore, the pathophysiological mechanisms of arrhythmias include abnormal automatism, triggered activity, and reentry. Recognition of these mechanisms is essential for the choice of appropriate treatment. The electrocardiogram (ECG) is the main diagnostic tool, allowing the identification of characteristic patterns such as missing or altered P waves, wide or narrow QRS complexes, and prolonged PR intervals (NAGATA et al., 2021).

Tachyarrhythmias were also classified according to their rhythm, with regular rhythm and narrow QRS, i.e., less than 120 milliseconds: sinus tachycardia, nodal reentry tachycardia, atrioventricular tachycardia, focal atrial tachycardia, and atrial flitter. Regular tachycardias with wide QRS levels greater than 120 milliseconds include monomorphic ventricular tachycardia,

supraventricular tachycardia with conduction aberrancy, and antidromic atrioventricular tachycardia (DIAS et al., 2023).

Irregular rhythm tachycardias with narrow QRS are represented by atrial fibrillation, multifocal atrial tachycardia, atrial flutter, or atrial tachycardia with variable AVB. And irregular rhythm tachycardias with wide QRS are polymorphic ventricular tachycardia, torsades de pointes, and atrial fibrillation with pre-excitation (BRANDÃO et al., 2023).

The electrocardiographic tracing is provided through the electrocardiogram, an exam for reading the electrical activity of the heart. Physiologically it shows the process of depolarization, repolarization, and transmission of the electrical impulse from the heart. The cardiac conduction system is composed of the following structures: sinus node, atrioventricular (AV) node, bundle of His, right and left branches, Purkinje fibers, and cardiomyocytes (ARCOVERDE FILHO et al., 2022).

These structures have the intrinsic characteristic of automatically depolarizing to a frequency of their own, and any external stimulus is unnecessary to generate this depolarization. The ability of these structures to emit a stimulus and automatically depolarize is called chronotropism. They also have the function of conducting the cardiac electrical stimulus, which is called dromotropism. Batmotropism is the condition in which the heart fiber can be excited by an external stimulus. Inotropism, on the other hand, refers to the ability of heart cells to contract the myocardial fiber (DE ABREU et al., 2022).

The physiological electrocardiographic tracing is represented by the P wave, which comprises the electrocardiographic recording of atrial depolarization, physiologically determined by the automatic depolarization of the sinus node. After the P wave, we have the PR interval, which represents the interval between the onset of the P wave and the onset of the QRS, which corresponds to the physiological delay in the conduction of the electrical wave generated in the atrial depolarization within the AV node. This delay can have a maximum duration of 0.2 seconds to be considered physiological (JACQUES et al., 2025).

The QRS complex represents the electrical depolarization of the ventricles and has a maximum physiological duration of 0.12 seconds. Physiological ventricular depolarization occurs by progression of the electrical stimulus through the junctional region, the right and left branches, and the Purkinje fibers, until it reaches the heart muscle. And the T wave represents ventricular electrical repolarization (BRANDÃO et al., 2023).

Atrial fibrillation is a narrow QRS arrhythmia, with irregular RR and absence of P waves, since there is no adequate command sent by the sinus node, consequently there is no adequate atrial depolarization. It is the most frequent cardiac arrhythmia, responsible for 30% of

hospitalizations due to heart rhythm alterations. It is classified into 3 types, the paroxysmal, which is self-limiting, the episodes last less than a week and in general less than 24 hours. There is also persistent where its duration is greater than 7 days, and can be maintained indefinitely until the patient is cardioverted. And the third type is permanent, where its duration is greater than 1 year, and is refractory to cardioversions (DE ABREU et al., 2022).

The diagnosis of fibrillation is usually based on clinical practice, and confirmed through an electrocardiogram with these characteristics. Their therapy is initially determined whether the patient is clinically stable or unstable. The signs of clinical instability for arrhythmias are dyspnea, anginal pain, fainting, decreased level of consciousness. If one of these signs or symptoms are present, I classify the patient as unstable. Therefore, the conduct should be electrical cardioversion of 50 to 100 J. If the patient is stable, my goal is heart rate control with beta-blockers or verapamil, dithiazem or digoxin, and anticoagulation to prevent cerebral thrombotic events (DE ARAÚJO SAMPAIO et al., 2024).

Anticoagulation therapy should be given to patients at high risk of embolization, such as patients with atrial fibrillation and atrial flutter. And we must stratify the risk of this patient through CHADSVASC, and depending on the risk, anticoagulation should be performed. If I identify a patient with stable atrial fibrillation, I should know the time of onset of atrial fibrillation. If it appeared in less than 48 hours, I must wait until the 48 hours are complete, as there may be spontaneous reversal of AF during this period. If there is a need to cardiovert this patient with less than 48 hours of AF, it is necessary to perform heparinization 6-12 hours before (PORTA-SÁNCHEZ et al., 2021).

If AF is already present for another 48 hours, cardioversion can be performed even in the stable patient, but it is necessary to prove that there are no thrombi in the atrium, for this it is necessary to perform a tranesophageal echocardiogram. If thrombi are present, or if the test is not performed, previous anticoagulation with warfarin should be performed for 4 weeks before cardiovery, and the INR should be in the ranges of 2.0-3.0. And after the procedure, the use of the anticoagulant should continue for at least another 4 weeks (JACQUES et al., 2025).

Supraventricular tachythymia is a narrow QRS arrhythmia, with regular R-R, there may be no P wave, and if there is it may be negative and is at the end of the QRS. Its diagnosis is made by the electrocardiographic pattern. And its treatment depends on the patient's stability or instability. If a stable patient, the first approach is to perform vasovagal maneuvers, such as carotid sinus massage or modified Valsalva maneuver, if there is no improvement in the condition I can do up to 3 cycles of adenosine, the first with 6 mg, the second with 12 mg and the third with 12 mg. Calcium channel blockers such as verapamil, diltiazem and metoprolol can

also be used. If the patient is unstable, the conduct is electrical cardioversion of 50-100J (DIAS et al., 2023).

Atrial flutter is a regular R-R narrow QRS arrhythmia, without the presence of P-wave, negative F-waves in IBD, IBD, and aVF with a morphology of serrated lines or "saw teeth". There is a substitution of P waves for others called F waves. Classically, flutter travels with a ventricular rate of 150 bpm. Among its main causes are atrial overloads, valvular heart disease, hyperthyroidism, and alcohol. Its treatment is immediate electrical cardioversion for cases of patients with hemodynamic instability. And anticoagulation should be performed with the same recommendations as atrial fibrillation (DE SOUZA et al., 2023).

Sinus tachycardia is an affectation characterized by sinus rhythm with a heart rate greater than 100 beats per minute, with regular R-R, and the presence of positive P waves preceding the QRS complexes. Among its main causes are fever, anemia, heart failure, physical exercise, emotions, hyperthyroidism. And its therapy is based on the treatment of its cause (BRITO et al., 2025).

Tachyventricular is a wide and aberrant QRS arrhythmia, it can have a single morphology or be polymorphic. It is mainly caused by structural heart disease, with coronary heart disease being the most common. Its therapy includes electrical cardioversion in monomorphic cardioversion. Polymorphic tachyventricular defibrillation should be performed (JACQUES et al., 2025).

Bradycardias are heart rate changes of less than 50 beats per minute. They can be caused by drugs such as beta-blockers, calcium channel blockers, digitalis and amiodarone. Increased vagal tone, hypothyroidism, disease of the sinus node conduction system, infiltrative or infectious diseases of the heart (FERRARI et al., 2024).

Sinus bradycardia corresponds to arrhythmia with a frequency of less than 50 beats per minute, and does not require specific treatment. If symptomatic, consider atropine, isoproterenol, or pacemaker. And it is always necessary to seek a reversible cause (BALÓN et al., 2024).

1st degree atrioventricular block is represented by a tracing with a PR interval greater than 200 ms, and the P wave is always followed by QRS. The treatment is the same as for sinus bradycardia (BALÓN et al., 2024).

2nd degree atrioventricular block, mobitz 1 is represented by an electrocardiographic tracing with increasing PR interval until conduction failure. Therapy is given with atropine 0.5 - 1.0 mg IV if symptomatic. On the other hand, 2nd degree atrioventricular block, mobitz 2 is represented by an electrocardiographic tracing for a constant PR interval until conduction failure.

In most cases, it does not respond to atropine and pacemaker implantation may be necessary (BALÓN et al., 2024).

3rd degree atrioventricular block is represented by a tracing with atrioventricular dissociation, with regular PP and RR intervals, and treatment is done by pacemaker implantation in most cases (BALÓN et al., 2024).

FINAL CONSIDERATIONS

Heart rhythm disorders are potentially serious conditions that require an accurate diagnosis and an effective therapeutic approach. The understanding of the underlying mechanisms and the correct interpretation of the electrocardiographic patterns are fundamental for a correct diagnosis and an adequate management conduction.

Treatment varies according to the nature of the arrhythmia, and may involve maneuvers, pharmacological measures, procedures such as cardioversion and defibrillation, as well as the placement of implantable devices. Early identification and timely intervention are essential to reduce the morbidity and mortality associated with these conditions.

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