v. 2, n. 13, 2022

International Journal of Health Science

MAIN ELECTROCARDIOGRAPHIC CHANGES

Ana Isabel Machado de Freitas
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0003-0506-0507

Úrsula Maria Moreira Costa Burgos
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0001-7234-4046

Maylla Fontes Sandes
Universidade Tiradentes, Aracaju
http://orcid.org/0000-0002-5511-0253

Angela Santos Lima
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0001-9730-2934

Catharina Garcia de Oliveira
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0001-6015-7323

Bruno José Santos Lima
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0002-5898-3746

Gabriel Dantas Lopes
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0002-9743-6825

Mariana Alma Rocha de Andrade
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0002-3516-6997

Fernanda Bastos Santos
Universidade Tiradentes, Aracaju
https://orcid.org/0000-0003-4040-1901

Manuela Azevedo Vieira
Universidade Tiradentes, Aracaju
http://lattes.cnpq.br/2674575184852071

Abstract: Introduction: The cardiac conduction system is formed by the sinoatrial (SA) node, interatrial and internodal bundles, atrioventricular (AV) node, bundle of His (atrioventricular) and Purkinje fibers. In the SA node, rhythmic impulses are generated that pass through the internodal pathways, reaching the AV node. In it, the impulse is conducted to the bundle of Hiss to finally reach the Purkinje fibers that will stimulate the entire cardiac ventricle. When evaluating an ECG, it is necessary to pay attention to both the way the stimulus is being conducted, that is, the morphology of the emitted waves, as well as the rhythm and frequency. **Methodology:** A systematic review of books on how to evaluate the ECG and its alterations was carried out, in addition to physiology treaties and guidelines on the same. This article aims to list the electrocardiographic changes in the main cardiac arrhythmias. **Results:** Cardiac arrhythmia is a condition identified by abnormalities or lack of rhythm in the heartbeat. Monitoring systems capture a non-stationary physiological signal formed by a sequence of waves that reflect the electrical activity of the heart. In electrophysiology, the genesis of cardiac arrhythmias is related to changes in both the formation and conduction of the cardiac electrical impulse. Bradyarrhythmias are disturbances of the heart rhythm that accompany the decrease in HR and result either from the reduction in the generation of the electrical stimulus, or from the disturbance of conduction by the cardiac tissue. Tachyarrhythmias are defined when the HR is above 100bpm, whether sinus or not. **Conclusion:** The ECG is a practical and low-cost exam, being a complementary exam to the clinical history and exam in emergency rooms and outpatient consultations. Arrhythmias present peculiarities of the electrocardiographic tracings, being necessary to evaluate the same in order to choose the best therapeutic plan and prognosis for the patient.

Keywords: electrophysiology, electrocardiogram, electrocardiographic changes.

INTRODUCTION

The heart's function is to make the body's blood undergo a transformation (venous to arterial, through the lung) so that it oxygenates all cells through systole. For this to occur, the cardiac muscles undergo electrical stimuli that will be represented on the electrocardiogram (ECG).

The cardiac conduction system is formed by the sinoatrial (SA) node, interatrial and internodal bundles, atrioventricular (AV) node, bundle of His (atrioventricular) and Purkinje fibers. In the SA node, rhythmic impulses are generated that pass through the internodal pathways, reaching the AV node. In it, the impulse is conducted to the bundle of Hiss to finally reach the Purkinje fibers that will stimulate the entire cardiac ventricle. (MACHADO, F.J; 2017).

The SA node is located in the superior posterolateral wall of the right atrium (RA). The atrial fibers connect to this and when issuing the impulse, it travels through the atrial musculature and also spreads in the interatrial bundle. The AV node, located in Koch's triangle, is responsible for delaying the transmission of the atrial impulse to the ventricles, which is responsible for atrioventricular synchronism (systole and diastole), (MACHADO, F.J; 2017). Subsequently, it goes to the bundle of Hiss where it enters the ventricular musculature that extends to the cardiac apex. At that moment, it divides into right and left branches that run through the entire endocardium on both sides.

Cardiac cells are submerged in an extracellular medium composed of several ions, including sodium, potassium and calcium. The intracellular medium has a higher

concentration of K+ while the extracellular medium has a predominance of Na+ and Ca2+. This ionic concentration difference is responsible for the transmembrane resting potential. (MACHADO, F.J; 2017).

METHODOLOGY

A systematic review of the literature on physiology was carried out, how to evaluate the electrocardiogram, as well as its alterations. As a reference, the III Guidelines of the Brazilian Society of Cardiology on analyzes and emission of electrocardiographic reports were used, in addition to the search for articles in the Sciello and Pubmed databases in the period 2017/2021 with the following keywords: bradyarrhythmias; tachyarrhythmias; electrocardiogram.

This article aims to show electrocardiographic changes in the main pathologies of cardiac arrhythmias in order to better choose the medical management, whether in medical emergency or outpatient. Therefore, we have a practical and low-cost exam, which helps a lot in the diagnosis and prognosis of patients.

RESULTS

Cardiac arrhythmia is a condition identified by abnormalities or lack of rhythm in the heartbeat. According to the Brazilian Society of Cardiac Arrhythmias, only one type of cardiac arrhythmia, atrial fibrillation, affects around 175 million people worldwide and 2 million in Brazil. (PASSO, S.J.G, et al. 2020).

Experimental evidence shows that heart disease could be diagnosed early, controlled and prevented with the help of continuous monitoring, especially of ECG signals. Monitoring systems capture a non-stationary physiological signal formed by a sequence of waves that reflect the electrical activity of the heart. (PASSO, S.J.G, et al. 2020).

The epidemiology varies according to the type of arrhythmia presented. Atrial fibrillation (AF) is the most clinically relevant presentation and shows a variable prevalence with age. Considering that the incidence of most cardiac arrhythmias tends to increase with advancing age, in view of the aging population, the number of cases of these rhythm disorders will increase. In the case of AF, the prevalence in people under 60 years of age is around 1%, while in people over 80 years of age, it exceeds 8%. In addition, the prevalence is influenced by the sex factor, being more prevalent in men, in the proportion of 2:1. Despite this, AF is more common in women, considering the survival rate related to them. (CARDIOLOGIA, Sociedade Brasileira, 2016a).

Changes in the impulse and propagation of the electrical stimulus and consequent decrease in heart rate (HR) can be observed as part of the aging process and/or disease progression, and are therefore more commonly identified in older individuals. Changes in the sinus node (SN), internodal bundles, atrioventricular node (AV) and other conduction tissue may result in bradycardia, atrioventricular (AV) dyssynchrony or abnormalities in ventricular depolarization (Costa, H.; Azevedo, P.; Carvalho, D.; Candeias, R.; Jesus, I, 2021).

A. BRADYARRHYTHMIAS

They are disturbances of the heart rhythm that accompany the decrease in HR and result either from the reduction in the generation of the electrical stimulus, or from the disturbance of conduction by the cardiac tissue. They are frequent and sometimes considered physiological dysrhythmias, such as some cases of sinus bradycardia in young adults or trained athletes. Its prevalence increases with age, and it is usually associated with sinoatrial disease (ASD) and AV conduction disorders.

The inability to generate or conduct the cardiac electrical impulse may be due to senile degeneration or a primary disorder of the cardiac conduction system (primary etiology); or be associated with several clinical conditions, whether reversible or permanent (secondary etiology); or both (primary etiology aggravated by secondary condition.

The 1st degree atrioventricular block (AVB) will have a widening of the PR interval, that is, greater than 200ms and will be the same at all times. This situation, considered benign, is asymptomatic in patients who have it and presents as possible causes: medications (beta-blockers, amiodarone), diseases of the conduction system, athletes and endocarditis (the abscess formed in the aortic valve can compress the conduction system).

The 2nd degree AV block divides into Mobitz I (Wenchebach phenomenon), when the PR interval progressively increases until the moment when the QRS complex becomes absent in the tracing. This cycle can be repeated for variable periods, when it is possible to notice that the PR interval, after the blocked beat, is the smallest among all, and the one that follows it has the highest percentage increase in relation to the later ones. (Pastore, C.A; et al 2016). In Mobitz II, in which the PQRS is normal and suddenly, the blocked P wave appears, without the QRS. For this reason, Mobitz II has a worse prognosis, since it initially appears normal and suddenly an alteration appears.

The 3rd degree AV block or total atrioventricular block (TAVB) in which there is complete dissociation of the P wave with the QRS complex, the explanation is the unsynchronized muscle contraction of the atria and ventricles. This can be intermittent or permanent. Blocks of supra-Hisian origin

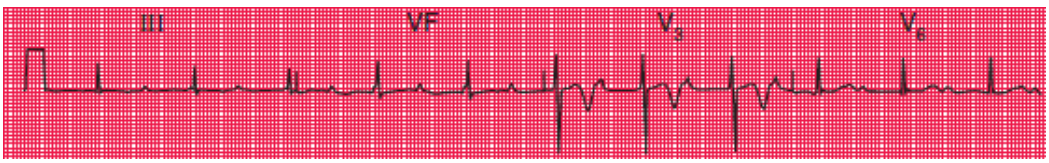

Figure 3.1: BAV of 1º degree. (HAMPTON, J.R.2011)

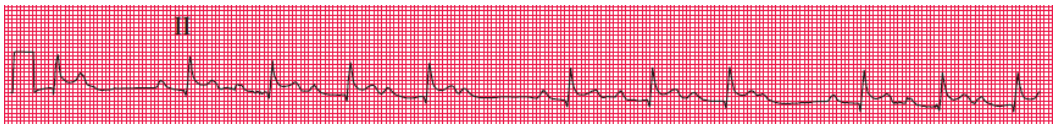

Figure 3.2: BAV 2º grau, Mobitz I. (HAMPTON, J.R.2011)

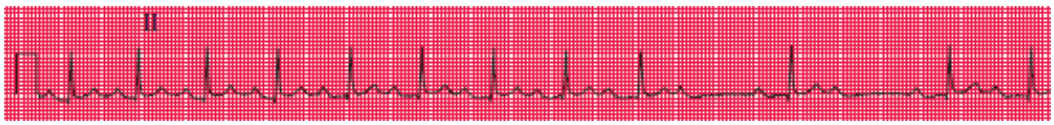

Figure 3.3: BAV 2º grau, Mobitz II. (HAMPTON, J.R.2011)

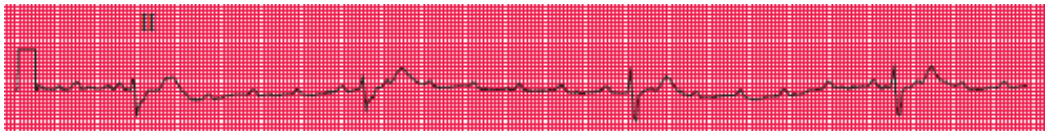

Figure 3.4: BAVT. (HAMPTON, J.R.2011)

present with ventricular leaks similar to the basal ECG, while infra-Hisian origin shows wide QRS complexes as leaks. This being the worst prognosis. (Pastore, C.A; et al 2016).

Atrioventricular block 2:1 is characterized by the situation in which, for every two beats of atrial origin, one is conducted and depolarizes the ventricle, and another is blocked and fails to depolarize the ventricle. Thus, the PP intervals are constant, thus excluding the diagnosis of blocked atrial extrasystoles. (Pastore, C.A; et al 2016).

In advanced or high-grade atrioventricular block, there is AV conduction in less than half of the atrial beats, with a ratio of 3:1, 4:1 or greater. The presence of AV conduction is noted by the constant PR interval on each beat, which generates a QRS. Most of these blocks are located in the intra/infra-His region. (Pastore, C.A; et al 2016).

Paroxysmal atrioventricular block is the sudden and unexpected occurrence of a succession of blocked P waves. When the block occurs from a shortening of the sinus cycle, it is called "phase 3", and when it results from a prolongation of this cycle, "phase 4". (Pastore, C.A; et al 2016).

In addition to the sinoatrial blocks, it is worth emphasizing the concepts of sinus pause and sinus disease. The first refers to a change in sinus automatism, with intermittent interruptions in impulse formation. On the ECG, these pauses are characterized by the absence of a P wave, the duration of which is not a multiple of the baseline PP interval. Regarding sinus disease, we can define it when symptoms of low cardiac output are associated with sinus node dysfunction. In most cases, it results from senile degeneration of the cardiac conduction system, and presents a wide spectrum of clinical and electrocardiographic presentations, including: sinus bradycardia, sinoatrial blocks, sinus arrest, junctional escape rhythm, long pauses after supraventricular extrasystoles, atrial fibrillation with poor ventricular response, cardiac rhythm instability after cardioversion, and brady-tachy syndrome (characterized by episodes of atrial tachyarrhythmias followed by sinus bradyarrhythmias), (MACHADO, F.J; 2017).

B. TACHYARRHYTHMIAS

Tachyarrhythmias are defined when the HR is above 100bpm, whether sinus or not. Those with a narrow QRS complex are the most frequent arrhythmias in the emergency room and on 24-hour Holter exams. They usually present with symptoms of palpitation, but they can also cause syncope in the presence of other cardiovascular comorbidities, such as aortic stenosis, mitral stenosis, ischemic and non-ischemic dilated cardiomyopathy. (MACHADO, F.J; 2017). Most have a good prognosis, being considered benign, not directly associated with risk of sudden cardiac death.

In electrophysiology, the genesis of cardiac arrhythmias is related to changes in both formation (automatism) and conduction (dromotropism) of the cardiac electrical impulse. For an arrhythmia episode to occur, a combination of three factors is necessary: supraventricular or ventricular extrasystole acting on an anatomical substrate that will be modulated by the autonomic nervous system. Impulse formation disorders include hyperautomatism, abnormal automatism (reflects the acquisition of exacerbated automatic capacity of cells that do not normally have a pacemaker function), early postpotential triggered activity (generation of electrical potential in phase 3 of the potential of action, often associated with intrinsic disturbances of ion channels) and late postpotential triggered activity (generation of electrical potential in phase 4 of the action potential, usually associated with intracellular

calcium accumulation). (MACHADO, F.J; 2017).

The dromotropic alterations are among the main mechanisms of action of certain cardiac arrhythmias, they are: anatomical reentry (an electrical circuit that occurs in contiguous areas with distinct conductivity or refractoriness), functional reentry (arrhythmias caused by reentry microcircuits where there is no interval of excitability, that is, it cannot be reversed by "overdrive suppression") and anisotropic reentry (re-entry mechanism associated with the myocardial syncytium – longitudinal and transverse fibers – or the double nodal pathway. (MACHADO, F. J; 2017).

Tachyarrhythmias are divided according to the size of their QRS into: narrow QRS tachyarrhythmias (<120ms) which would be supraventricular and wide QRS tachyarrhythmias (>120ms) which would be ventricular.

Sinus tachycardia, which has a regular rhythm, is the arrhythmia that occurs in times of anxiety, physical exercise, pain, fever, hypotension. As an electrocardiographic change, it includes only the increase in HR. It can be considered inappropriate sinus tachycardia when the heart rate is above 100 beats per minute during wakefulness, with a reduced rate only during sleep. Postural orthostatic tachycardic syndrome (SPOT) is an abnormal or exacerbated behavior of the sympathetic autonomic nervous system (dysautonomia), in which we observe patient intolerance to the orthostatic position. In the tilt test (tilt test) we observed a sudden increase in heart rate above 30 beats per minute in relation to rest or persistence of heart rate above 120 beats per minute in the first 10 minutes of the tilt phase, not associated with arterial hypotension serious. (MACHADO, F.J; 2017).

Atrial tachycardias can be unifocal or multifocal whose arrhythmogenic mechanisms involve abnormal automatism or peripheral reentry.

In unifocal atrial tachycardia, a single region generates the stimulus, which is usually paroxysmal. In the electrocardiographic tracing, it presents a P wave, however, it is not characterized as sinus and its rhythm is usually regular. Regarding the time of crisis, they can be sustained or not sustained if the period is or is not longer than 30 seconds, respectively. (MACHADO, F.J; 2017).

Multifocal atrial tachycardias have a multifactorial mechanism with a paroxysmal character, whether sustained or not. (MACHADO, F.J; 2017). Due to its mechanism, we will find several foci of stimulus and, consequently, presentation of P waves of different morphologies in the same lead.

Atrial fibrillation (AF) is the most common sustained tachycardia in clinical practice. In it, diseases that cause left atrial overload (thyrotoxicosis, hydroelectrolytic disorders, hypertensive heart disease, among others) cause microreentry to generate numerous stimuli, instead of just the SA node generating. On ECG, it will be represented by the absence of P wave, irregular rhythm with irregular R-R interval. The baseline may be isoelectric, with fine or coarse irregularities or a mix of these changes (waves "f"). The occurrence of regular RR intervals indicates the existence of AV dissociation. (Pastore, C.A; et al. 2016). The HR may be within the normal range and when >100 bpm, it is considered high-response AF.

Atrial flutter, unlike AF, is characterized by macro-rents in the right atrium, has a regular rhythm, presence of the F wave and absence of the P wave. A clinical situation that suggests flutter is the presence of a narrow QRS tachycardia associated with a HR of 150 bpm. We can classify atrial flutter as typical or

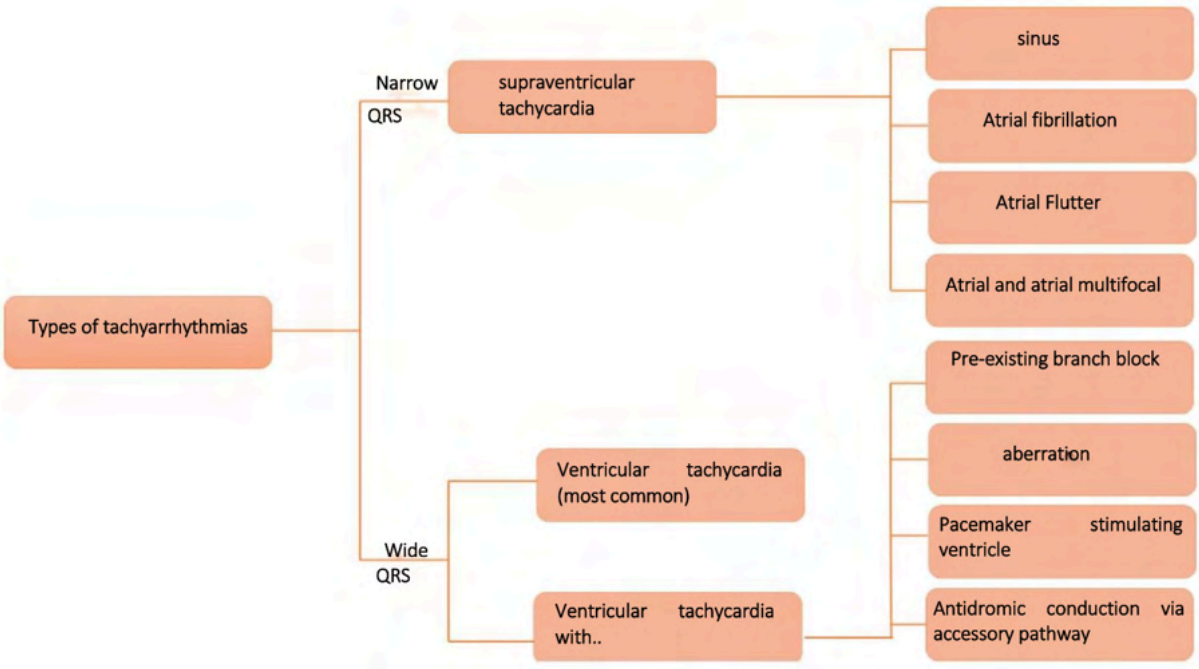

Diagram 3.1: Summary of tachyarrhythmia types.

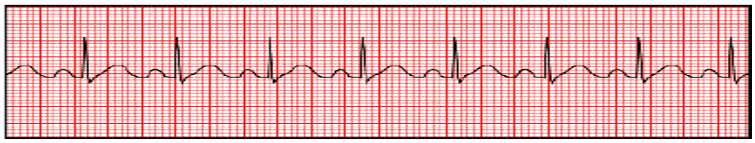

Figure 3.5: Sinus tachycardia. (LAPA, E.C.S. 2017).

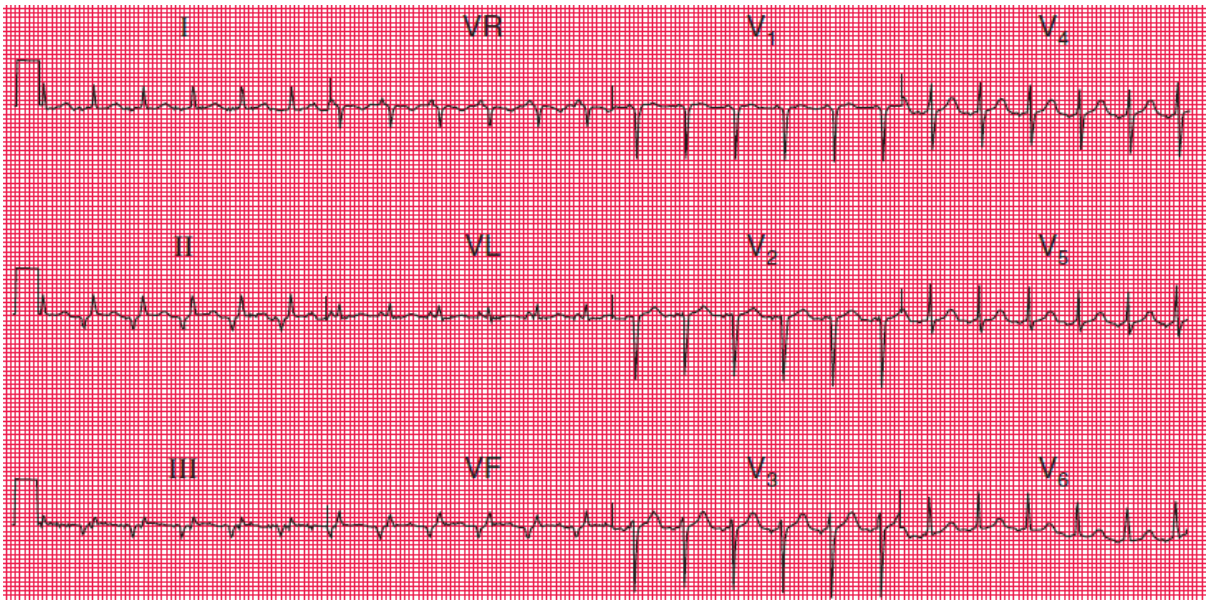

Figure 3.6: Atrial tachycardia with non-sinus P wave. (HAMPTON, J.R.2011).

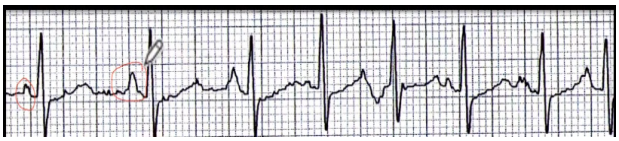

Figure 3.7: Multifocal atrial tachycardia. (LAPA, E.C.S. 2017)

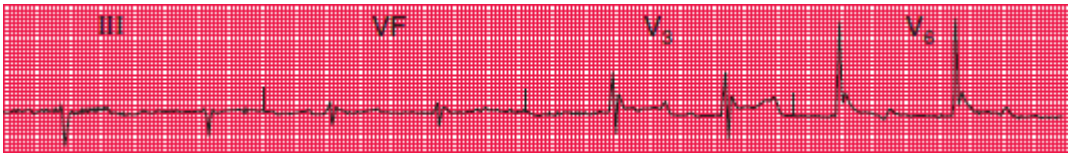

Figure 3.8: FA. (HAMPTON, J.R.2011).

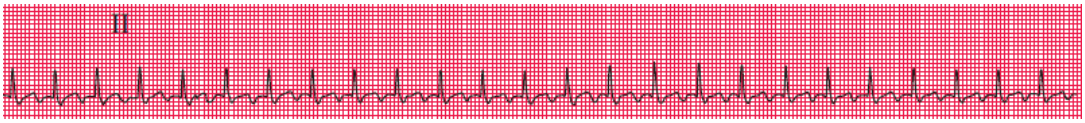

Figure 3.9: Atrial flutter. (HAMPTON, J.R.2011)

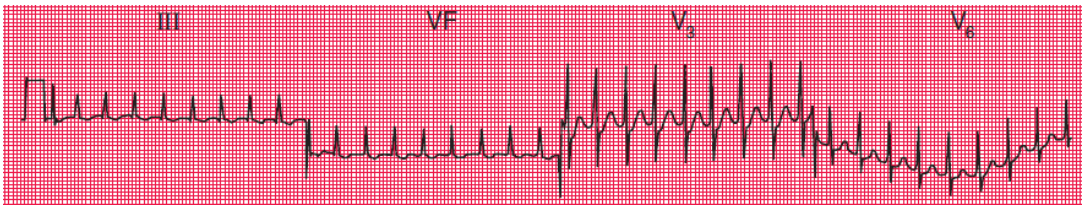

Figure 3.10: Junctional tachycardia. (HAMPTON, J.R.2011)

isthmodependent, whose frequency of F waves varies from 240 to 340 bpm; and atypical or not isthmal whose frequency of F waves varies from 340 to 430 bpm. As for the direction of the reentry circuit, the typical atrial flutter can be classified as common (counterclockwise circuit), whose polarity of the F waves is negative in D2, D3 and avF and positive in V1; and uncommon (hourly circuit) whose polarity of the F waves is positive in D2, D3 and avF and negative in V1.

Focal junctional tachycardia is a replacement or replacement rhythm originating at the AV junction, with QRS of the same morphology and duration as the basal rhythm. (Pastore, C.A; et al. 2016). On the ECG, we observe a progressive shortening of the PR interval, until junctional QRS complexes take over the cardiac rhythm. (MACHADO, F.J; 2017).

Nodal reentry tachycardia is a supraventricular tachycardia whose arrhythmogenic mechanism involves a reentry circuit between two nodal lives, one with slow conduction and short refractory period and the other with fast conduction and long refractory period. Thus, nodal reentry tachycardia is a tachyarrhythmia that uses the AV node as a fundamental part of the electrical circuit. Regarding the electrocardiographic aspects, at rest, the PR interval is normal, since atrioventricular conduction normally follows the fast conduction pathway. For the patient to go into a tachyarrhythmia crisis, a supraventricular extrasystole occurs early, and upon finding the life of rapid conduction of the refractory period, he starts to conduct anterogradely through the slow conduction pathway. As soon as the electrical impulse

reaches the distal portion of the AV node, it finds the fast conduction pathway outside the refractory period, favoring the return of conduction to the atrium in a retrograde manner, thus initiating the atrioventricular reentry mechanism. Therefore, on the ECG it will be a strictly regular RR interval, RP interval < 70ms, pseudo S in D2, D3 and avF and pseudo R in V1. (MACHADO, F.J; 2017).

Atypical nodal reentrant tachycardia, the site of origin and circuit are similar to typical NRT, but the direction of activation is reversed, which is why retrograde atrial activation occurs temporally later, with the characteristic RP interval longer than the PR. (Pastore, C.A; et al. 2016).

Atrioventricular reentry tachycardia (AVRT) via an anomalous or accessory bundle has a strictly regular, paroxysmal RR interval, whose reentry circuit involves the AV node and an accessory pathway. (MACHADO, F.J; 2017). Of the existing types, Wolf-Parkinson-White Syndrome (WPW) will be addressed.

WPW syndrome is a supraventricular tachyarrhythmia, often associated with high cardiac death in the presence of pre-excited atrial fibrillation with high ventricular response. In the electrocardiographic tracing at sinus rest, it will present a short PR interval (<120 ms) and ventricular pre-excitation (delta wave) associated with crises of tachycardic palpitation due to tachyarrhythmia involving the anomalous pathway. (MACHADO, F.J; 2017).

Ventricular tachycardias are those with wide QRS (>120ms), including ventricular tachycardia (VT) which corresponds to 80% of cases. In addition to VT, there are other causes of tachyarrhythmia with wide QRS, indented teeth: beats of supraventricular origin conducted with aberrant atrioventricular conduction (transient or pre-existing); ventricular activation through an accessory bundle observed during supraventricular tachycardia due to antidromic atrioventricular reentry; and supraventricular tachycardia with ventricular pre-excitation. (MACHADO, F.J; 2017).

The main arrhythmogenic mechanism of ventricular tachycardias is reentry, and the triggered activity and changes in automatism are other mechanisms related to the genesis of ventricular tachycardias. (MACHADO, F.J; 2017).

The clinical history is extremely important along with the ECG to help in the definitive diagnosis. Thus, the presence of structural heart disease, history of previous infarction or heart failure, favors the suspicion of a VT. The factors that will determine whether the patient will have a milder or more severe condition are high heart rate, associated underlying heart disease and the presence of left ventricular dysfunction.

Wellens e cols observed that heart rates between 170 and 200 bpm were constantly found in patients with supraventricular tachycardia. In patients with VT, the heart rate was usually between 130 and 170 bpm. Regarding regularity, regular wide QRS tachycardias suggest the diagnosis of VT. The presence of atrioventricular dissociation during wide QRS tachycardia establishes the diagnosis of VT with 100% specificity. (MACHADO, F.J; 2017).

Monomorphic Ventricular Tachycardia (MVT) corresponds to a ventricular rhythm with at least three successive beats, uniform morphology and frequency greater than 100 bpm, it is classified according to its duration as Sustained Ventricular Tachycardia (SVT) or Non-Sustained Ventricular Tachycardia (NSVT), if the arrhythmia period is or is not longer than 30 seconds, respectively. (Pastore, C.A; et al. 2016).

In cases of VT, it is extremely important to differentiate VT from an aberration (tachycardia-induced bundle branch block)

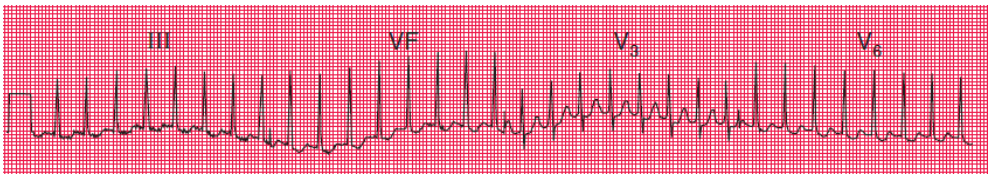

Figure 3.11: Nodal reentry tachycardia. (HAMPTON, J.R.2011)

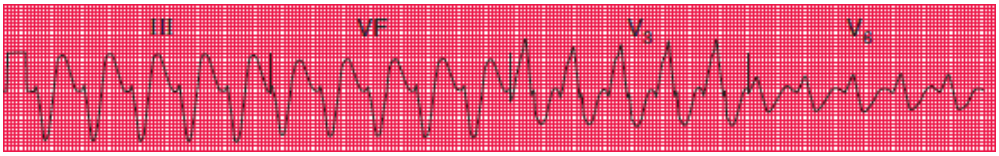

Figure 3.12: TV. (HAMPTON, J.R.2011)

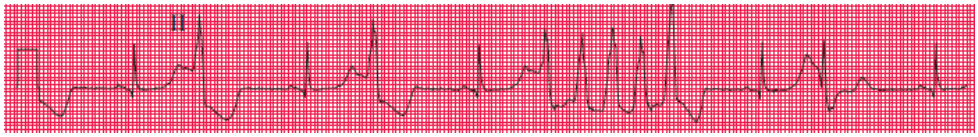

Figure 3.13: *Torsade de pointes.* (HAMPTON, J.R.2011)

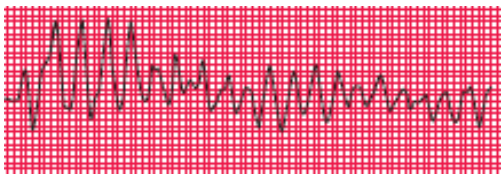

Figure 3.14: FV. (HAMPTON, J.R.2011)

and for that, there are some criteria. The Vereckei criterion says that the presence of an initial R wave in aVR indicates a TV. The Brugada criterion says that the absence of RS in all precordial cells is also suggestive of VT. Finally, the dos Santos criterion reports that the presence of negative QRS in most DI, DII, V1 to V6 makes a diagnosis for VT.

And also as an addition about TV, there is talk of T*orsade de Pointes* (Tdp). It is a polymorphic, wide QRS tachycardia that is usually self-limiting. It is usually preceded by long-short cycles (extrasystole - sinus beat - extrasystole), both in congenital and acquired forms, and is related to the presence of long QT, which may be congenital or secondary to drugs and electrolyte disturbances. (Pastore, C.A; et al. 2016).

Finally, there is ventricular fibrillation, it will be an anarchic rhythm, the patient will be in cardiac arrest and with HR > 300 bpm.

CONCLUSION

As it is a practical and low-cost exam, the ECG fits as a complementary exam to the clinical history and exam in emergency rooms and outpatient consultations.

Arrhythmias are divided into groups according to the HR and peculiarities of the electrocardiographic tracings, being necessary to evaluate the same in order to choose the best therapeutic plan and prognosis for the patient, since behaviors that are not consistent with the alteration can lead to medical iatrogenesis. Therefore, the knowledge of health professionals about the main changes in the electrocardiogram is relevant.

REFERENCES

1. Costa, H.; Azevedo, P.; Carvalho, D.; Candeias, R.; Jesus, I. **Bradiarritmias: abordagem em contexto pré e intra-hospitalar.** Life Saving Scientific. Nº 1, volume 1. Centro Hospitalar Universitário do Algarve. Ago-2021

2. DUBIN, D. **Interpretação rápida do ECG**. 3ª ed. Rio de Janeiro. Editora de publicações científicas LTDA. 2004.

3. GUYTON, A.C. **Tratado de Fisiologia**. 12ª ed. Rio de Janeiro. Elsevier. 2011.

4. HAMPTON, J.R. **ECG 150 Casos Clínicos**. 4ª ed. Rio de Janeiro. Elsevier. 2011.

5. LAPA, E.C.S. **Manual de Eletrocardiografia: Cardiopapers.** Atheneu, 2017.

6. LOURENÇO, O. Potencial de acção no músculo cardíaco: Electrofisiologia Básica. **ANGOMED Portal de Actualidades Médica**, 2014. Disponível em: http://angomed.com/electrofisiologia-basica/ Acessado: 15/02/2021.

7. MACHADO, F.J. **ECG: entendendo eletrocardiograma**. 1ª ed. Aracaju. Infographics Gráfica & Editora. 2017.

8. PASSO, S.J.G.; OLIVEIRA, H.S.; PINTO, R. A.; Montero QUISPE, K. G. M., GUSTI, R.; SOUTO, E. J. P. **Classificação de Arritmias com Paradigma Inter e Intra Paciente utilizando Aprendizagem Profunda.** J. Health Inform. Número Especial. Dezembro 2020.

9. Pastore, C.A; Pinho, J.A; Pinho, C; et al. **III DIRETRIZES DA SOCIEDADE BRASILEIRA DE CARDIOLOGIA SOBRE ANÁLISE E EMISSÃO DE LAUDOS ELETROCARDIOGRÁFICOS**. ISSN-0066-782X. Volume 106, Nº 4, Supl. 1, abril 2016.

10. Sociedade Brasileira de Cardiologia. **II Diretrizes Brasileiras de Fibrilação Atrial**. Arq Bras Cardiol. 2016; 106 (Supl.IV): 1-35. (a).