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Therapeutic approach to stable tachycardias

Kamilla Kristina Faleiros Sá Gomes de Almeida[1], Marcela Gattass Luchesi[2], Victor Nogueira de Jesus[3], Taysa Horn da Cruz[4], Roberto Guilherme Rosa Morais[5], Ronaldo Marcelo Taques[6].

ABSTRACT

This article aims to clarify possible doubts about the therapeutic approach of stable tachycardias in medical practice, therefore, the study on the treatment of tachyarrhythmias, a heart rhythm disorder characterized by rapid heartbeat, usually above 100 bpm, will be approached in a clear and objective way. These arrhythmias can be classified into two types: supraventricular arrhythmias, which originate above the bundle of His, and ventricular arrhythmias, which begin in the ventricles. Various factors, such as heart disease, emotional stress, substances such as drugs.

Keywords: Heart rate, Quality of life, Tachycardia.

INTRODUCTION

This article aims to clarify possible doubts about the therapeutic approach of stable tachycardias in medical practice, therefore, the study on the treatment of tachyarrhythmias, a heart rhythm disorder characterized by rapid heartbeat, usually above 100 bpm, will be approached in a clear and objective way. These arrhythmias can be classified into two types: supraventricular arrhythmias, which originate above the bundle of His, and ventricular arrhythmias, which begin in the ventricles. Several factors, such as heart disease, emotional stress, substances such as drugs, caffeine, and alcohol, can trigger tachyarrhythmias. Depending on the severity, such conditions can cause symptoms such as palpitations, dizziness, fainting and, in extreme cases, even sudden death. Prompt diagnosis and treatment is crucial to prevent serious complications and improve patients' quality of life. In this work, therapy will be contemplated, in an objective way, for stable tachycardias, facilitating understanding.

OBJECTIVE

The initial approach to major tachyarrhythmias in medical practice plays a crucial role in the effective management of these conditions. Given the diversity of cardiac arrhythmias, it is essential to adopt an evidence-based therapeutic approach. This article aims to outline the treatment of the main tachyarrhythmias found in clinical practice, contributing to a better understanding and a more effective approach to these conditions in an objective way.

[1] University of Cuiabá – Mato Grosso
[2] University of Cuiabá – Mato Grosso
[3] University Center of Várzea Grande – Mato Grosso
[4] University of Cuiabá – Mato Grosso
[5] University of Cuiabá – Mato Grosso
[6] University of Cuiabá – Mato Grosso

METHODOLOGY

For the conception of this document, we proceeded to the exegesis of patterns of expanded synopses, as well as to the search of relevant literature and databases updated on the appropriate therapeutic management of stable tachycardias in the practical setting. Subsequently, a discussion was engendered among the authors about the formulation of a standard that would amalgamate the analyses undertaken, aiming to improve the understanding of the therapeutic approach to these clinical conditions.

DEVELOPMENT

It is a fundamental task to differentiate between unstable and stable tachycardias. In cases of unstable tachycardia, it is crucial that treatment be initiated immediately by means of synchronized electrical cardioversion, a theme that will not be addressed in the current study. On the other hand, in stable tachycardias, there is time for further evaluation and identification of the specific type of tachyarrhythmia, enabling appropriate and personalized treatment for each patient. Therefore, differentiating between stable and unstable tachycardias is crucial to ensure effective treatment and reduce the risks of serious complications for each patient.[1-4 years]

When it comes to stable tachycardias, the approach should follow two subsequent criteria: QRS duration, which can be classified as wide (equal to or greater than 0.12 seconds) or narrow (less than 0.12 seconds), and QRS regularity, which can be identified as regular or irregular. This evaluation is important to establish the origin of the tachyarrhythmia and define the most appropriate treatment for each specific case. Therefore, the analysis of these criteria is essential for the diagnosis and choice of appropriate therapies in patients with stable tachycardias.[2,5,7]

Narrow and regular QRS: Listed below are the possibilities included in this segment and a brief description of the conditions listed, which:

Sinus tachycardia is an arrhythmia in which the heart accelerates its heart rate above 100 beats per minute, due to an excessive stimulation of the sinus node, which is the heart's natural pacemaker; atrial tachycardia occurs when there is an abnormal focus of electrical activity in the walls of the atria, resulting in a rapid and irregular heart rhythm; Atrial flutter is an arrhythmia in which the atria contract rapidly, causing a high and regular heart rate, and can be identified by the characteristic pattern on an electrocardiogram; Nodal Reentrant tachycardia (NRT) occurs when there is an abnormal electrical pathway in the atrioventricular node, which allows the re-entry of the electrical impulse and causes tachycardia, and Atrioventricular Reentry tachycardia (VAT) is an arrhythmia in which an accessory electrical pathway occurs, allowing the re-entry of the electrical impulse.

However, in many cases, sinus tachycardia can be secondary to other triggering factors such as infections, hydroelectrolyte disorders, emotional stress, hypoxia, among others. It becomes crucial that the

underlying cause is treated appropriately. For example, if the cause is an infection, treatment will include administering antibiotics, while in the case of hyperthyroidism, treatment will involve therapy to control thyroid function. Only after the identification and treatment of the triggering factor will it be possible to control sinus tachycardia effectively.[2,7,8]

When it is not possible to identify the type of rhythm or if the tachycardia is stable, the initial approach is to perform vagal maneuvers, which aim to stimulate the vagus nerve and lead to bradycardia, that is, a reduction in heart rate. The most commonly performed vagal maneuvers are the carotid sinus massage and the Valsalva maneuver. In carotid sinus massage, the doctor applies gentle pressure to the carotid sinus (located on the side of the neck), which can lead to a vagal response and decreased heart rate. In the Valsalva Maneuver, the patient is instructed to exhale forcefully against the closed glottis, for example, blow a syringe trying to push the plunger or exhale against the back of the hand. This maneuver increases intra-abdominal pressure and stimulates the vagus nerve, which can lead to a reduction in heart rate. These maneuvers can be effective in some cases to stop tachycardia and restore the heart's normal rhythm, but they should be performed carefully and only by trained professionals, as they can cause side effects such as hypotension and syncope.[7.9]

Carotid sinus massage may be contraindicated in patients with carotid atherosclerosis due to the risk of embolization, which is the release of thrombotic material into the bloodstream. For this reason, this maneuver should be performed with caution in elderly patients over 65 years of age, with carotid murmurs, or with several risk factors for atherosclerosis, such as diabetes mellitus, systemic arterial hypertension, and smoking. It is important to note that these contraindications do not apply to all cases and that the physician must evaluate each patient individually before deciding to perform the maneuver. In some cases, it may be necessary to resort to other therapeutic options, such as antiarrhythmic medications or electrical cardioversion.[7.9]

In 2015, a randomized clinical trial was published and showed that the modified Valsalva maneuver may be an effective therapeutic option to reverse some tachyarrhythmias. In this technique, the patient is positioned in a sitting position and is instructed to perform a forced exhalation for 10 to 15 seconds. Then the patient is laid down with their legs elevated at a 45-degree angle for another 15 seconds. This sequence leads to increased intrathoracic pressure and vagus nerve stimulation, which can help reduce the heart rate. The results of this study showed that the modified Valsalva maneuver was able to successfully reverse some tachyarrhythmias, such as paroxysmal supraventricular tachycardia, in more than 40% of cases. In addition, the technique was well tolerated by the patients and did not present serious side effects.[2.3]

If vagal maneuvers are not effective in reversing tachyarrhythmia, adenosine administration is often considered as a next therapeutic option. Adenosine is a drug that acts directly on adenosine receptors

in the atrioventricular node (AVN), inhibiting its electrical conduction and reducing heart rate. The drug has a peak effect and lasts very quickly, usually within seconds. Adenosine should be administered as an IV bolus, with *fush* of saline and elevation of the limb to rapidly reach the central circulation. The initial dose is 6 mg and can be repeated with higher doses. According to the ACLS 2020 guidelines, an additional dose of 12 mg of adenosine can be given if the first dose is not effective, while the European Supraventricular Tachycardia Guideline 2019 allows for an additional third dose of 18 mg. However, it is important to note that the use of adenosine is contraindicated in patients with bronchospasm, such as those who have asthma attacks or exacerbated COPD.[2-4]

Finally, if there is no response to adenosine, antiarrhythmic drugs should be administered to control heart rate, the most commonly used in the emergency room:

Metoprolol by intravenous administration, which is a 5mg beta-blocker with a maximum dose of 15 mg. It is important to evaluate for the possibility of bronchospasm and heart failure before administering the drug, as they contraindicate.[7]

Diltiazem is a calcium channel blocker that is administered intravenously at a dose of 0.25 mg/kg. It should not be used to treat patients with congestive heart failure and left ventricular dysfunction.[7]

Verapamil can be given as an intravenous bolus in doses of 5 to 10 mg and is a calcium channel blocker. However, it is contraindicated in patients with congestive heart failure and left ventricular dysfunction.[7]

Deslanoside is a cardiac glycoside that can be administered intravenously as a bolus, with a dose of 0.4 to 0.8 mg. However, its use should be avoided in patients with impaired renal function, as there is an increased risk of digitalis intoxication.[7] Signs of digitalis poisoning are nausea, vomiting, tachyarrhythmias and xanthopsia (yellowish vision).

Amiodarone should be taken intravenously with a dose of 150 to 300 mg diluted in saline over 10 minutes. This medication acts as a blocker of potassium, calcium, and sodium channels.[7]

Outpatient treatment includes radiofrequency ablation of the anomalous beam (VAT), dual nodal pathway (NRT) or macro entry circuit (atrial flutter) depending on the symptoms and risk of sudden death associated with WPW Syndrome. The decision to perform ablation depends on the characteristics of the anomalous bundle in question.[8,9,10]

In patients with pre-excitation, the use of drugs that decrease conduction through the AV node should be avoided, as this may increase the risk of conduction through the anomalous bundle and, consequently, the risk of sudden death. The most indicated antiarrhythmic drugs to prevent recurrence of supraventricular tachycardias in patients with pre-excitation are Propafenone and Procainamide, but their use is contraindicated in patients with coronary artery disease or structural heart disease.[11]

Beta-blockers and calcium channel blockers are proscribed drugs and increase mortality in cases of atrioventricular reentrant tachycardia.[7]

Patients with advanced age and renal dysfunction are more susceptible to poisoning by digitalis antiarrhythmics, whose clinical presentation may include gastrointestinal symptoms (such as nausea, vomiting and abdominal pain), neurological symptoms (such as confusion and weakness) and visual symptoms (xanthopsia, characterized by the visualization of yellowish objects), as well as several types of arrhythmias (such as bradycardia, atrial tachycardia and ventricular extrasystoles in patients with chronic diseases). biggeminism). Management of this condition involves drug discontinuation, rehydration, evaluation of possible associated fluid and electrolyte disturbances (such as hypokalemia and hypomagnesemia), and, in refractory cases, the use of anti-digoxin antibodies.[7,12,13]

Narrow and irregular QRS: In these cases, the main diagnostic hypothesis is Atrial Fibrillation (AF), however, in rare cases, atrial flutter, variable atrioventricular block, or multifocal atrial tachycardia should be considered as differential diagnoses. Below is the discretion of an FA.[7]

Atrial fibrillation (AF): occurs due to multiple chaotic re-entries of small waves within the atria. However, sometimes an ectopic focus is triggered within venous structures adjacent to the atria (such as the pulmonary veins). In this way, the atria do not contract and the AV conduction system is bombarded by various electrical stimuli, leading to an inconsistency of impulse transmission and irregular ventricular rate, which is usually found at the frequency limits of tachycardia. This pathology presents on the ECG in an irregular rhythm, with absence of "p" wave and tremor at baseline ("f" wave).[7.9]

AF can be classified according to its clinical presentation as follows: Paroxysmal when it is reversed spontaneously or with medical intervention within 7 days; persistent duration of more than 7 days and permanent in cases in which attempts to revert to sinus rhythm will no longer be instituted.

Valvular AF occurs in the presence of moderate to severe mitral stenosis and with mechanical valve prosthesis.

The initial treatment should be to control the heart rate with antiarrhythmic drugs (such as Metoprolol, Delanoside and Amiodarone), being careful with amiodarone, as it can revert to sinus rhythm. Verapamil and Diltiazem should not be used in patients with left ventricular dysfunction or heart failure (as previously stated).[7]

The decision on the reversion to sinus rhythm should take into account the time of symptom onset, due to the risk of thromboembolism. In patients with AF lasting less than 48 hours, electrical or chemical cardioversion (with amiodarone) can be performed without the need for prior anticoagulation, evaluating the CHA2DS2-VASc score. Amiodarone is effective in reversing and maintaining sinus rhythm.[7.10]

For patients with AF of longer duration, anticoagulation should be initiated for at least 3 weeks prior to attempting cardioversion, followed by a minimum of 4 weeks of anticoagulant treatment. One

option, if available, is to perform a transesophageal echocardiogram, and in the absence of atrial thrombi, synchronized electrical cardioversion can be safely performed.[8-11 months]

The indication of anticoagulation for a longer period of time should take into account comorbidities and the patient's age, and should be performed by scales, such as the acronym CHA2DS2-VASc. In the presence of 2 or more points, anticoagulation is indicated.

CHA2DS2-VASc score: C for heart failure (1 point); H for hypertension (1 point); A2 age ≥ 75 years (2 points); D for diabetes (1 point); S2 of previous stroke or transient ischemic attack (2 points); V for coronary or peripheral vascular disease (1 point); Age from 65 to 74 years (1 point) and Sc female (1 point).

The point classifications were: CHA2DS2-VASc of 0: anticoagulate with heparin (during conversion), without the need for subsequent anticoagulation; CHA2DS2-VASc of 1: use clinical judgment and consider an antiplatelet agent (aspirin) and CHA2DS2-VASc ≥ 2: anticoagulation is indicated, which can be done with direct oral anticoagulants or warfarin. Antiplatelet agents (such as ASA and clopidogrel) do not reduce the risk of VTE, so they are not indicated in this case.

The risk of bleeding should also be assessed using the HAS-BLED score. In case of a score ≥ 3 points, the patient is at high risk of bleeding and should be monitored more frequently.

HAS-BLED score: we have the H of uncontrolled hypertension or systolic blood pressure ≥ 160; Abnormal renal or hepatic function alteration (creatinine > 2.6 or dialysis or cirrhosis and significantly increased bilirubins or transaminases); S of previous stroke; B of antecedents or predisposition to bleeding; L of labile INR; And of elderly people > 65 years of age and D of drug use.

Anticoagulant therapy traditionally involves coumarin drugs (warfarin), which inhibit vitamin K-dependent clotting factors (II, VII, IX, and X) related to the extrinsic coagulation pathway.

Its effect should be monitored by prothrombin time (assessed by the *International Normalized Ratio* – INR). In general, after initiation of warfarin, a prothrombin time control should be ordered in approximately 5 days. These drugs, however, have several drug and food interactions, which makes it difficult to keep the patient in the therapeutic range (INR between 2 and 3).

In the last decade, oral anticoagulants with direct action on clotting factors have become available, without the need to monitor clotting times and with fewer drug interactions. However, patients with moderate or severe mitral stenosis or mechanical valve prosthesis should be anticoagulated with warfarin, and these new drugs are contraindicated.[7]

Regarding the mechanism of action of the new oral anticoagulant drugs, dabigatran inhibits factor IIa, while rivaroxaban, apixaban, and edoxaban inhibit coagulation factor Xa.[7.10]

The patient's renal and hepatic function should be checked before starting treatment with the new anticoagulants. During treatment, renal function should be monitored regularly and liver function

occasionally for factor Xa inhibitor medications. At the end of the day, the rule that new blood thinners do not require control is not completely true, because although it is not necessary to monitor the anticoagulation itself, it is essential to monitor kidney function regularly.

The recommended doses of the different oral anticoagulants are: Warfarin: start at 5 mg/day or 2.5 mg/day if elderly patient or < 60 kg. Measure the INR from the third day. The full effect of warfarin requires at least 7 days of use (factor II half-life). Dabigatran: 150 mg 2 times/day. For patients at risk of bleeding, 110 mg 2 times/day may be used. Rivaroxaban: 20 mg 1 time/day. Apixaban: 5 mg 2 times/day. For patients at risk of bleeding, 2.5 mg 2 times/day and Edoxaban: 60 mg/day.[7]

In patients with AF, there is no evidence that the rhythm control strategy (reversion to sinus) is superior to heart rate control.

In outpatient management, there is the possibility of ablation, which is indicated for very symptomatic patients (with frequent palpitations). In AF, interventional therapy (ablation) is not superior to the use of antiarrhythmic drugs in reducing harsh outcomes, such as mortality, HF, and strokes, but it can improve symptoms and quality of life.[4,5]

Wide and regular QRS: in these cases the main hypothesis is monomorphic ventricular tachycardia (about 80% of the time this is the diagnosis found). Supraventricular tachycardia (already previously described) with wide QRS can be found in situations of conduction aberrance or pre-excitation; however, these are rarer situations and, in order to differentiate them, electrocardiographic criteria (such as Brugada and Vereckei, among others) are necessary, which are not easy to apply in an urgent and emergency environment for non-specialists.[2,4,7] Below is a summary of monomorphic ventricular tachycardia:

Monomorphic ventricular tachycardia (VT): these are those with a single abnormal focus or reentrant route and identical-looking and regular QRS complexes. Ventricular tachycardias can be classified into sustained and non-sustained. Sustained VT occurs for more than 30 seconds or generates hemodynamic instability; and those that are not sustained occur for less than 30 seconds and do not generate instability.[7]

The patient, although stable, has the potential for rapid clinical worsening, with a high probability of degeneration to the rhythm of arrest. For this reason, it should be monitored in an emergency room. Initial treatment is a trial of chemical cardioversion with amiodarone.

In the absence of response, the next step is synchronized electrical cardioversion.

Patients should be investigated for the presence of myocardial ischemia, and suggestive ECG findings and markers of myocardial necrosis (troponin) should be evaluated.

ACLS 2020 allows adenosine administration to be performed for patients with large, regular QRS tachyarrhythmia who are hemodynamically stable. This recommendation is based on the fact that a small

percentage of patients may be presenting with supraventricular tachycardia with conduction aberrancy, for which adenosine could be sufficient to lead to reversion to sinus rhythm.[7]

Wide and irregular QRS: these are rare cases, and consist of polymorphic ventricular tachycardias, better described below:

Polymorphic ventricular tachycardias: have several different foci or pathways and variable and irregular QRS complexes. There is a specific type of polymorphic tachycardia, called *Torsades de Pointes* in which the QRS complexes increase and decrease, as if there is a change in the axis or "twisting of the tips".[7]

In general, patients with wide and irregular QRS are unstable or on the verge of instability (drowsy, borderline blood pressure). For this reason, the immediate approach is defibrillation (with maximum load), considering that, due to the complete disorganization of the rhythm, it is not possible to synchronize the shock with the R wave.

Correction of electrolyte disturbances (especially hypokalemia and hypomagnesemia) should be performed. Prevent subsequent episodes with amiodarone or lidocaine. As a second line of treatment, beta-blockers (e.g. Esmolol or Metoprolol) may be used. If the cause is ischaemic, initiate measures for acute coronary syndrome.[3,4,8]

In the specific case of Torsades de Pointes, the most effective treatment is magnesium sulfate. If there is no response, defibrillation should be carried out. All patients should receive magnesium, even if magnesium dosing is normal, to maintain elevated serum levels with a target of 3.5-5 mg/dL until withdrawal of the causative agent. Mg dosing should be done every 6 hours. If Mg > 7 mg/dL, stop the infusion. If 5-7 mg, decrease infusion by half. The loading dose is 10% magnesium sulfate 2 g IV in 10 min and the maintenance dose is 10% magnesium sulfate 1-4 g/hour, as it acts by decreasing the QT interval. If the patient remains refractory to the measurements, the option is to increase the heart rate (HR) to decrease the QT interval. It can be used in the infusion of chronotropic medications (e.g. adrenaline or dobutamine); or electrical chronotropism with transcutaneous or transvenous pacemaker. The HR target is 100 to 110 bpm. Aggressively treat hypokalemia, with a target serum K of 4.5 mEq/L.[7,9,12]

Magnesium sulfate has anticonvulsant properties, with a central nervous system depressant effect (with analgesic and sedative potential). It blocks neuromuscular transmission. It also acts as a vasodilator, reducing blood pressure and heart rate. It is still used for arrhythmias (as above) and cerebral edema. Its mechanism is not fully elucidated, but it seems to be due to antagonism to the action of calcium.[7,8]

This arrhythmia is related to conditions that increase repolarization time, which on the ECG is identified by increased QT interval, such as congenital causes (long or idiopathic QT syndrome), metabolic causes (such as hypokalemia, hypocalcemia, and hypomagnesemia), antiarrhythmics (such as

amiodarone, sotalol), antibiotics (such as quinolone, macroids, azole antifungals), psychotropic (such as halopedirol, tricyclic antidepressant), and anticholinergic drugs (such as poisoning by organophosphates).[7]

FINAL THOUGHTS

In summary, understanding the management of stable tachyarrhythmias is of crucial relevance in daily medical practice, given its association with severe complications, including heart failure, stroke, and sudden death. In this context, it is imperative that healthcare professionals, including us, future doctors, are able to identify and treat such conditions effectively. It is also critical to recognize that the treatment of stable tachyarrhythmias has advanced significantly over the years, with the advent of new pharmacological therapies and ablation techniques, requiring us to stay up-to-date to provide the best possible care to our patients. Ultimately, a thorough understanding of the management of stable tachyarrhythmias can not only contribute to the prevention of adverse complications, but also to the substantial improvement of the quality of life of affected individuals. Therefore, it is essential that we dedicate continuous efforts to study and update in this field, as demonstrated throughout this article.

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