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USE OF ANTI-ARRHYTHMIC DRUGS IN STABLE TACHYARRHYTHMIA

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Abstract: Introduction: Tachyarrhythmias comprise a group of heart rhythm disorders whose heart rate is greater than or equal to 100-110 beats per minute. When very high, the greater the relationship with signs and symptoms of instability. These cases of unstable tachyarrhythmias have a formal indication for electrical cardioversion (EVC). For stable ones, however, it is valid to carefully evaluate case by case and indicate from vagal maneuvers to pharmacological measures with antiarrhythmics. Since it is still necessary to know more about chemical cardioversion, the current literature review aims to compendium the use of antiarrhythmic drugs in stable tachyarrhythmias. **Methodology:** The present research was carried out through a critical review of bibliographic literature with analysis and discussion. At first, there was no time interval or source language delimitation. The main selection criterion was based on the quality of the information, excluding old materials that disagreed with current guidelines for the management of tachyarrhythmias. Finally, the content was synthesized and summarized in texts and tables in order to facilitate dynamic reading and comprehension. **Results:** The mechanism of tachycardia helps in deciding the type of treatment. There are two main mechanisms of tachycardia: automatism or reentry. Stable tachycardias can also be reversed with CVE or antiarrhythmic drugs. In this scenario, it is possible to first define the tachyarrhythmia to decide, then, the best drug to be used, considering its contraindications. **Conclusions:** The use of antiarrhythmic drugs in stable cases presents a wider range of possibilities, requiring a more detailed diagnosis and a broad pharmacological knowledge of this drug class. In this sense, outlining the therapeutic options allows both to update the scientific literature on the subject, as well as to facilitate the review of the

first therapeutic line of a disorder that usually presents itself in the condition of emergency care.

Keywords: Stable tachyarrhythmias, Antiarrhythmics, cardioversion.

INTRODUCTION

Cardiac morphofunctionality provides the ability to generate electricity on a regular and continuous basis in a phenomenon known as automatism. Under normal conditions, the physiological rhythm of the heart is determined by the sinus node, which fires about 80,000 to 120,000 heartbeats a day – at a rate of 60 to 80 times per minute. This rate of depolarization allows it to control, at first, the cardiac rhythm, despite the existence of self-excitable cells, the atrioventricular node, the bundle of Hiss and the fibers of Purkinje.

To define sinus rhythm, at least 3 criteria are evaluated: morphology and orientation between 0º and 90º, P waves with the same morphology, positive on D2 and preceding each narrow QRS. When there is alternation in these criteria, it is a heart rhythm disorder.

In this context, tachyarrhythmias make up a group of heart rhythm disorders whose heart rate is greater than or equal to 100-110 beats per minute. When very high, there is a greater relationship with signs and symptoms of instability, such as hypotension (SBP < 90 mmHg) or circulatory shock (perfusion disturbance), anginal chest pain, altered level of consciousness and/or dyspnea associated with pulmonary congestion.

These cases of unstable tachyarrhythmias have a formal indication for electrical cardioversion (EVC). For stable ones, however, it is valid to carefully evaluate case by case and indicate from vagal maneuvers to pharmacological measures with antiarrhythmics. Classically, antiarrhythmic drugs were divided into four groups (**Table 1**).

GROUP 1

Drugs from this group depress most of the electrophysiological properties of the myocardial fiber. Decrease Na+ entry into the cell, decrease conduction velocity, decrease automatism, increase refractory period duration, increase action potential duration. They are represented by Quinidine, Procainamide and Disopyramide. Still in this Group, there are also those that have the same mechanism of action, but with greater kinetics, such as Lidocaine, Phenytoin and Tocainide.

GROUP 2

The drugs of this Group have the direct membrane effects of Group I and also act through their anti-adrenergic effect - they produce a competitive and reversible block of the actions that catecholamines exert via cardiac beta-adrenergic receptors. In healthy myocardium, beta-blockers only depress excitability, conduction velocity (does not change QRS), and atrial and ventricular refractory periods. In pathological myocardium, this class exerts beneficial effects ranging from antianginal properties to rhythmic control. They are represented by Propranolol, Metoprolol and Atenolol.

GROUP 3

It is characterized by the blockade of several potassium outflow currents, with an increase in the refractory period and vasodilator and antianginal properties. They are represented by Amiodarone, Sotalol and Ibutylide.

GROUP 4

These drugs block calcium channels in a cardioselective manner, which at the cardiac level is responsible for the excitation-contraction coupling of sinus and atrioventricular node depolarization, maintenance of phase 2 of the action potential

and coronary vascular tone. All these drugs have antihypertensive and antianginal properties. They are represented by Verapamil and Diltiazem.

OTHERS

These are drugs that do not participate in the aforementioned antiarrhythmic groups, but have shown a certain role in restoring heart rhythm. Adenosine, for example, activates an outflow of K+ and inhibits cAMP-stimulated Ca2+ inflow. The increase in K+ flux hyperpolarizes the membrane potential of the sinus and atrioventricular nodes and inhibits their automatic activity, while in atrial cells it shortens the membrane potential. Magnesium salts, in turn, are considered because hypokalemia increases the automaticity of ectopic pacemakers and causes the appearance of early postpotentials that predispose ventricular arrhythmias, such as Torsades de Pointes). In addition, hypokalemia facilitates the appearance of signs of digitalis intoxication.

Seeing that, it is still necessary to know more about chemical cardioversion, the current literature review aims to compendium the use of antiarrhythmic drugs in stable tachyarrhythmias.

METHODOLOGY

The present research was carried out through a critical review of bibliographic literature with analysis and discussion. Initially, material on the topic addressed was sought in books, journals and scientific publications linked to the virtual health library, journals and electronic databases Medline (National Library of Medicine, USA), Lilacs (Latin American and Caribbean Literature in Sciences, Health) and Scielo (Scientific Electronic Library Online).

For that, the keywords were used (Stable tachyarrhythmias. Antiarrhythmics. Cardioversion). At first, there was no time interval or source language delimitation. The main selection criterion was based on the quality of the information, excluding old materials that disagreed with current guidelines for the management of tachyarrhythmias. Finally, the content was synthesized and summarized in texts and Tables in order to facilitate dynamic reading and comprehension.

RESULTS

The mechanism of tachycardia helps in deciding the type of treatment. There are two main mechanisms of tachycardias:

Groups	Action mechanisms	Main drugs
Group IA	They block sodium channels	Quinidine, Procainamide, Disopyramide
Group IB	They block sodium channels + have higher kinetics	Lidocaine, Phenytoin, Tocainide
Group II	Beta blockers	Propranolol, Metoprolol, Atenolol
Group III	They block potassium channels and elevate the refractory period	Amiodarone, Sotalol, Ibutilide
Group IV	They block cardioselective calcium channels	Verapamil and Diltiazem
Others	They do not belong to the above Groups	Adenosine, digitalis and magnesium

Table 1. Vaughn-Williams classification of antiarrhythmic drugs.

automatism, in which a group of myocardial cells take command of cardiac depolarization with accelerated heart rate and may be secondary to autonomic, electrolyte or pharmacological alterations; or reentry, in which there is a circuit that allows the circulation of the cardiac stimulus, with at least two pathways.

In automatism, electrical cardioversion or short-acting drugs such as adenosine are of little use, since after the effect of the procedure, the same group of cells resumes the cardiac rhythm.

On re-entry, myocardial stimulation may occur at each cycle leading to tachycardia. For reentry to continue to occur, perfect timing is required, and any factor that alters the conduction velocity or the refractory period of the pathways can interrupt it, such as antiarrhythmics.

The biggest problem with the use of antiarrhythmics is the possibility that they can generate or worsen cardiac arrhythmias. The problem is magnified by considering that the risk of proarrhythmic effects is greatest in patients who may benefit most from these drugs (eg, with ischemic heart disease, intracardiac conduction disturbances, or ventricular dysfunction).

Patients with stable tachycardias must immediately undergo a 12-lead ECG. Other complementary exams will depend on the clinical picture and the electrocardiographic alteration in question. For example, the ECG may show sinus tachycardia and the clinical picture indicates only an anxiety disorder. On the other hand, the patient may also have the same sinus tachycardia, but he/she has signs/symptoms suggestive of pulmonary embolism.

Stable tachycardias can also be reversed with CVE or antiarrhythmic drugs. The current trend is for only one antiarrhythmic drug to be used to avoid the proarrhythmic effect of drugs; in case of failure in the reversal, CVE is indicated. In this scenario, it is possible to first define the tachyarrhythmia (Table 2) and then decide the best drug to

Narrow QRS tachycardias without a P wave	
Variable RR interval	Atrial fibrillation
Constant RR interval; there are no P waves, but there are F waves ("in the mountains")	Atrial *Flutter*
Constant RR interval with no visible atrial depolarization	Junctional, paroxysmal supraventricular (nodal reentry), or orthodomic atrioventricular (AV) tachycardia
Narrow QRS tachycardias with P wave	
PR interval smaller than RP interval	Sinus tachycardia (ST) or atrial tachycardia (TA)
PR interval equal to RP interval	This could be the situation above with 1st degree AV block, but if the HR is close to 150 bpm, consider the possibility of a 2:1 atrial flutter, in which half of the F waves are covered by the QRS
PR interval greater than RP interval	PR interval < 0.08 second: paroxysmal supraventricular tachycardia due to nodal reentry PR interval for > 0.08: orthodromic atrioventricular tachycardia
Wide QRS tachycardias	
About 80% of these arrhythmias are ventricular tachycardias (VT), and the other 20% are supraventricular (SVT) with conduction aberration.	

Table 2. Diagnosis of tachyarrhythmia.

be used, considering its contraindications (Table 3).

SINUS TACHYCARDIA

In principle it is secondary and does not need specific treatment. The triggering cause, such as fever, anxiety or pain, must be investigated and corrected.

ATRIAL TACHYCARDIA

It is usually secondary to extracardiac diseases, such as pneumonia and chronic obstructive pulmonary disease, and to medications. CVE is often ineffective, as autofocus resumes rhythm after myocardial depolarization by CVE. If specific treatment is required, diltiazem or verapamil is recommended. beta blocker is an option. Rarely, amiodarone may be necessary.

JUNCTIONAL TACHYCARDIA

It responds well to drugs capable of blocking the NAV, such as beta-blockers or calcium channel blockers.

SUPRAVENTRICULAR TACHYCARDIA

The objective is to perform instantaneous parasympathetic discharge in atrioventricular node! It must start with a vagal maneuver, such as Valsalva, compression of the carotid sinus after auscultation, among others. If not effective, consider adenosine (6mg bolus with the possibility of doubling the dose on a second attempt). If contraindicated, opt for Verapamil. If still persistent, evaluate electrophysiological study and cure with ablation.

ATRIAL FIBRILLATION

It has an important relationship with the formation of intracavitary thrombi and subsequent embolization. Therefore, it can be reversed up to 48 hours after its onset, while the risk of embolism is very low. After 48 hours of arrhythmia (or if the date cannot be specified), HR must be controlled with drugs that block the atrioventricular node (verapamil, diltiazem or beta-blockers) and the patient must be anticoagulated with warfarin. With 3 weeks of effective anticoagulation (INR = 2.0 to 3.0), the *flutter* and keep the patient anticoagulated for another 4 weeks. An alternative to anticoagulation prior to ECV is to request a transesophageal echocardiogram; in the absence of a thrombus, we can proceed with CVE. After cardioversion, the patient must be anticoagulated for 4 weeks.

ATRIAL FLUTTER

The atria may not contract effectively during the arrhythmia, with the possibility of intracavitary thrombus formation and subsequent embolization. Although there is no evidence as consistent as that of atrial fibrillation (AF), Flutter can be reversed in a similar way to AF: anticoagulation for 3 weeks, CVE and anticoagulation for another 4 weeks. Alternatively, a transesophageal echocardiogram can be performed and, if there is no thrombus, CVE and anticoagulation for another 4 weeks can be performed.

Flutter does not respond well to chemical cardioversion by most available drugs.

MONOMORPHIC VENTRICULAR TACHYCARDIA

When stable, it can be treated with drugs with antiarrhythmic activity in the ventricles (preference for amiodarone or procainamide; sotalol is an option).

POLYMORPHIC VENTRICULAR TACHYCARDIA

Pharmacological treatment to prevent arrhythmia recurrence will depend on the QT interval during sinus rhythm or if

there is a very recent ECG. If normal QT interval: IV amiodarone and beta-blocker are recommended. Magnesium is not helpful. If prolonged QT interval, such as *Torsades de Pointes*, it is necessary to use antiarrhythmics, treat associated electrolyte disturbances, and withhold potential triggering drugs (antiarrhythmics, quinolones, etc.). CVE is of little help due to the intermittent nature of the arrhythmia. The administration of 2 g of magnesium sulfate and the passage of a temporary pacemaker to accelerate the baseline HR (even if the patient is not bradycardic) are the recommended measures.

It is worth mentioning that, with rare exceptions, when the chosen drug does not control the arrhythmia or if the maximum dose produces a high incidence of adverse reactions, the antiarrhythmic can be modified or associated with another with different properties from the first one. The association of a drug from group IB with AI or IC, for example, potentiates its effectiveness and allows reducing the dose and the incidence of undesirable effects of each one of them. On the other hand, the association of HF with AI is not recommended, as it produces greater depression of intracardiac conduction and increases the incidence of proarrhythmogenic effects.

CONCLUSIONS

Electrical cardioversion is a well-known procedure indicated in the presence of unstable tachyarrhythmias. However, the use of antiarrhythmic drugs in stable cases presents a wider range of possibilities, requiring a more detailed diagnosis and a broad pharmacological knowledge of this drug class.

In this sense, outlining the therapeutic options (Table 4) allows both to update the scientific literature on the topic, and to facilitate the review of the first therapeutic line of a disorder that usually presents itself in the condition of emergency care.

Drugs	Contraindications
Procainamide	Atrioventricular blocks, hypotension unrelated to dysrhythmia, hypersensitivity
Beta blockers	Sinus bradycardia, atrioventricular block, congestive heart failure, bronchial asthma
Amiodarone	Atrioventricular block (major)
Verapamil	Association with beta-blockers (additive effect), atrioventricular blocks

Table 3. Contraindications of the main antiarrhythmic drugs.

Stable tachyarrhythmia	Therapeutic option
Atrial tachycardia	Diltiazem, verapamil or beta-blocker
Sinus tachycardia	To correct cause
Junctional tachycardia	Beta-blockers or calcium channel blockers
Supraventricular Tachycardia	Vagal maneuver followed by adenosine if necessary
Acute atrial fibrillation	Amiodarone or synchronized CVE
AF of indeterminate date or atrial flutter	HR control ((diltiazem, verapamil or beta-blocker)
Monomorphic Ventricular Tachycardia	Amiodarone or Procainamide or CVE
Polymorphic Ventricular Tachycardia	Magnesium and pacemaker

Table 4. General therapeutic regimen in the main stable tachyarrhythmias.

After the electrocardiogram in hemodynamically stable patients, the regularity of the arrhythmia and QRS duration must be evaluated, contraindications must be individualized and, finally, the best antiarrhythmic drug for the case must be instituted.

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