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SUSTAINED VENTRICULAR ARRHYTHMIAS: THE STORM HAS ARRIVED

ARRITMIAS VENTRICULARES SUSTENTADAS: A TEMPESTADE CHEGOU

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ABSTRACT

One of the biggest challenges in the care of patients in emergency units is the treatment of ventricular arrhythmias, particularly when sustained and relapsing, as they are difficult to treat and are associated with high mortality. The main mechanism involved in the maintenance of ventricular tachycardias is the mechanism of reentry, due to myocardial scars secondary to various structural heart diseases. The electrical storm may be serious when it occurs in patients with automatic defibrillators, causing multiple corresponding shocks outside the hospital setting. In these cases, admission to hospital is necessary, where specific and stepwise treatment measures are performed, ranging from clinical management to specific interventions, such as programming of electronic devices, and electrophysiological or surgical interventions.

Keywords: Arrhythmias, Cardiac; Tachycardia, Ventricular; Defibrillators; Heart Diseases.

RESUMO

Um dos grandes desafios no atendimento dos pacientes nas unidades de emergência é o tratamento das arritmias ventriculares, principalmente, quando sustentadas e recorrentes, pois são de difícil tratamento e estão associadas à alta mortalidade. O principal mecanismo envolvido na sustentação das taquicardias ventriculares é o mecanismo de reentrada, devido às cicatrizes miocárdicas secundárias a diversas cardiopatias estruturais. A tempestade elétrica pode ser séria quando ocorre em portadores de desfibriladores automáticos, provocando múltiplos choques correspondentes fora do ambiente hospitalar. Nesses casos é necessária a internação hospitalar, onde medidas específicas e escalonadas de tratamento são realizadas, indo desde o manejo clínico até intervenções específicas, como programação de dispositivos eletrônicos, intervenções eletrofisiológicas ou cirúrgicas.

Descritores: *Arritmias Cardíacas; Taquicardia Ventricular; Desfibriladores; Cardiopatias.*

INTRODUCTION

One of the greatest challenges in the care of patients in emergency units is the treatment of ventricular arrhythmias, especially when sustained and recurrent, as they are difficult to treat and are associated with high mortality.

Ventricular tachycardia (VT) is clinically defined as sustained when the duration is longer than 30 s, or when associated with hemodynamic instability and requiring immediate interruption.[1] Electrical storm (ES) is defined as the occurrence of 3 or more separate episodes of sustained VT in a period of 24 h, each requiring interruption. VT is considered incessant when sustained despite repeated attempts at interruption.[1]

The primary mechanism involved in sustained VT is reentry, due to myocardial scarring secondary to various structural heart diseases; less frequently, sustained VT is due to intrinsic pacemaker disorders or triggered activity, caused by electrolyte imbalance or metabolic disorders, or even due to genetically determined diseases in individuals with seemingly normal hearts. The electrocardiogram (ECG) patterns during VT, whether monomorphic or polymorphic, can suggest its etiology and the prognosis.[1]

ES can be dramatic when it occurs in patients with automatic defibrillators, causing multiple corresponding shocks outside the hospital environment. In these cases, prompt hospitalization is required for specific measures and stepwise treatment, preferably by an integrated heart team with cardiologists, intensivists, and electrophysiologists for coordinated and optimized action.

This section will discuss the main concepts, differential diagnosis of wide QRS tachycardia, clinical treatment, and specialized approach to sustained ventricular arrhythmias, with a focus on emergency management.

Concepts in ventricular tachycardia

VT is defined as a sequence of 3 or more heart beats arising from the ventricles, with a frequency between 100 and 250 beats per minute.[1,2] VT can be classified as sustained or non-sustained, monomorphic or polymorphic, paroxysmal or incessant, and endocardial or epicardial, according to

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electrophysiological presentation.[1,2] VT can be observed in the evolution of heart diseases and sometimes occurs in clinical situations where structural heart disease cannot be identified. The prognostic significance is also variable and must take into account peculiarities specific to each context in which this arrhythmia presents. VT is almost always associated with the presence of structural heart disease. When there is no evidence of structural heart disease (approximately 15% of cases), a broad spectrum of possibilities arises, and one must evaluate the presence of changes in the QT interval, family history, and use of drugs, as well as the morphology of the tachycardia on the acute ECG, which may suggest the origin.[1] The recognition VT on the ECG is critical in the formulation of diagnostic hypotheses and therapeutic planning.

Sustained VT (SVT) is defined as an event with a minimum of 3 ventricular complexes, with duration greater than 30 s, and/or associated hemodynamic instability.[1,2] It may be present in seemingly normal hearts or in structural heart disease.

VT in seemingly normal hearts may be summarized as:

1. Repetitive monomorphic VT
2. Sustained or incessant monomorphic tachycardia
   - Idiopathic right ventricular outflow tract origin
   - Idiopathic left ventricular origin
3. Polymorphic tachycardia associated with long QT interval.
   - Congenital long QT Syndrome
   - Drug-induced congenital long QT Syndrome
4. Polymorphic tachycardia with normal QT interval
   - Triggered by extrasystole with short coupling interval
   - Catecholaminergic VT
   - Brugada Syndrome
5. Polymorphic tachycardia with ultra-short QT interval

VT that occurs in an emergency environment is usually secondary to a scarred substrate with reentry in patients with preexisting structural heart disease (ischemic and nonischemic cardiomyopathy).[1,3]

Finally, the concept of ES should be considered. ES is a term in common usage, but is defined in an arbitrary manner,[1] e.g., the occurrence of 3 or more potentially malignant episodes of ventricular arrhythmia within 24 h, requiring emergency intervention with drugs, cardioversion, or an implantable cardioverter defibrillator (ICD) (overstimulation or shock). ES is a severe event, in the majority of cases leading to hospitalization in an intensive care unit (ICU), and may be responsible for a dramatic presentation, especially in patients with an ICD, when repetitive shocks occur. In addition to the risk of harm and discomfort, repetitive shocks can result in premature failure of the ICD. Pacing is appropriate and effective in the diagnosis of ES (more than 3 events in 24 h) caused by the ICD. It is also important to identify inappropriate therapy for arrhythmias that do not need treatment, such as supraventricular tachycardia or oversensing of artifacts by the ICD.[4-6] In most cases, ES is an emergency that requires hospitalization in the ICU. Repeated shocks are uncomfortable and can cause severe psychological stress.

DIFFERENTIAL DIAGNOSIS OF VENTRICULAR TACHYCARDIA

Stable VT should be distinguished from other wide-QRS tachycardias. The recognition of tachyarrhythmia is a decisive factor for the most accurate and optimized approach in the emergency room. Advances in programmed electrical stimulation of the heart (electrophysiological study) have improved understanding of the origin and mechanisms of tachycardia, with increased accuracy in correct interpretation of the 12-lead ECG.

Tachycardia that occurs during cardiopulmonary resuscitation, including polymorphic VT and ventricular fibrillation, will not be discussed in this section.

The differential diagnosis of regular tachycardia with wide QRS complexes (greater than 0.12 s) implies a systematic approach of the ECG, which allows differentiation between supraventricular tachycardia with aberrant conduction, VT, and supraventricular tachycardia with antegrade conduction through an accessory pathway (Figure 1).

In contrast to regular narrow-QRS tachyarrhythmia, in which paroxysmal tachycardia is independent of etiology, the etiological diagnosis in patients with regular tachyarrhythmia and a wide QRS guides therapy and prevents inappropriate use of antiarrhythmic drugs. From a practical point of view, if the emergency physician does not have extensive experience in differentiating tachyarrhythmias, one must consider the entire spectrum of "undefined" QRS tachycardias as VT. Moreover, if tachycardia occurs in a hemodynamically unstable patient, it is assumed that, until proven otherwise, treatment should be as for VT. It has been shown that when emergency physicians must distinguish between VT and supraventricular tachycardia with aberrancy, errors occur in more than 50% of cases,[7] most often when true VT is treated with drugs that would be used for supraventricular tachycardia with conduction aberrancy.[8]

Nevertheless, some algorithms using configuration and/or morphology criteria for the QRS complex have been published,[9-10] with the aim of reducing errors. None of these algorithms are sensitive and specific in 100% of cases; however, some authors have demonstrated greater chances of success when the algorithm is more visual and direct, or when a more global, inclusive, or "holistic" approach based on all criteria is used.[11-13] The Brugada algorithm enables differential diagnosis between supraventricular tachycardia with conduction aberrancy and VT, using a sequence of 4 consecutive steps.[9] We suggest the mnemonic: "*RS - 100 – decoupling- morphological*". If the criterion for VT is met in the first step, no further analysis is necessary. If, however, the diagnosis of VT cannot be made after the 4 basic steps are applied, the tachycardia and QRS complex abnormalities must be related to conduction aberrancy. The sensitivity and specificity of these criteria were analyzed in 554 wide-QRS

Wide QRS tachycardia –Stable
Possible scenarios

- Monomorphic VT
- Polymorphic VT
- SVT with conduction aberrancy
- Preexcitation antidromic tachycardia
- Undefined wide-QRS complex tachycardia

Figure 1. Possible scenarios in the presence of stable, wide-QRS tachycardia. When in doubt, treat as VT.

tachycardia recordings and reached values of 99% and 96%, respectively, for the diagnosis of VT.

Regarding the first step proposed by the criteria of Brugada et al.,[4] the first question is whether a complex RS can be identified in any of the precordial leads (V1-V_6). If such a complex cannot be identified, the diagnosis of VT will be established. If RS complexes in these leads are present, the interval between the beginning of the R wave and the nadir of the S-wave should be measured. If this interval is greater than 100 ms in any of the precordial leads, the diagnosis of VT is also confirmed. However, when this interval is less than 100 ms, the third step is applied. The third step takes into account the presence of atrioventricular dissociation, which alone allows the diagnosis of VT. The occurrence of fusion beats also confirms the presence of atrioventricular dissociation and diagnosis of VT. Finally, if these 3 steps do not provide a diagnosis, the next step is the analysis of classic morphological criteria. These must be present in leads V1 and V_6, and should demonstrate the presence of right and left bundle-branch block. For practical purposes, the presence of a Q wave in lead V_6, regardless of the type of bundle-branch block, virtually excludes the possibility of supraventricular tachycardia with aberrancy or antidromic atrioventricular tachycardia (accessory pathway).

Another 4-step algorithm was proposed by Vereckei et al.[10] This uses a single RV lead, in which the presence of a wide R wave would confirm the diagnosis of VT in the majority of cases.

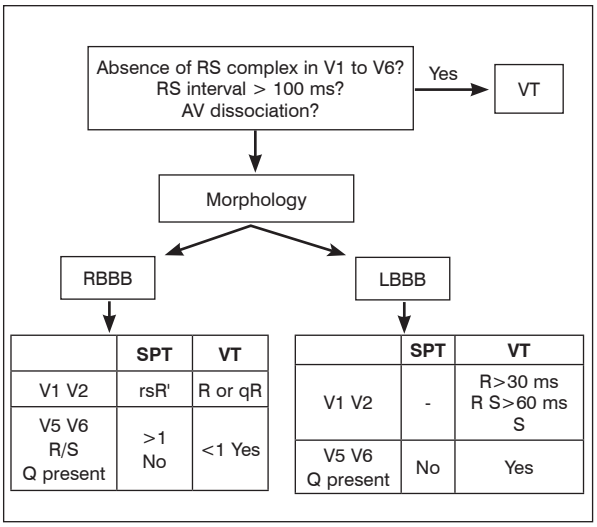
A simplification of these 2 algorithms and the morphological criteria are summarized in Figures 2 and 3, with examples of an ECG using these criteria in Figure 4.

EMERGENCY MEDICAL TREATMENT

After a wide-QRS tachycardia is identified, a diagnosis of VT or generic, wide-QRS complex tachycardia of uncertain origin is most safely treated with electrical cardioversion (class I). If electrical cardioversion is not successful, cannot be performed, or is not used (unavailable and/or the patient is nonfasting and "stable"), the drug of choice is intravenous amiodarone.

Approximately 30% of patients with an ICD will at some point develop ES, a life-threatening syndrome, with clinical presentation of recurrent episodes of ventricular arrhythmias within a short period of time, followed by appropriate shocks by the ICD.[14-15] The lifelong incidence of ES varies according to the population studied, and is 10-58% in patients with an ICD for secondary prevention, and 4-7% when used for primary prevention.[14-17]

In cases of ES, sedation should always be considered; this helps to reduce sympathetic tone. The correction of possible comorbidities (e.g., cardiac insufficiency, fluid and electrolyte imbalance, coronary insufficiency) is crucial for the control of the arrhythmia. Intravenous (IV) infusion of amiodarone in a loading dose (up to 5 mg/kg as a bolus and infusion of 900-1,200 mg IV in 24 h for up to 4 days, up to a maximum of 2,200 mg in the first 24 h) is the procedure of choice in cases of recurrent arrhythmia, except in the rare condition of congenital long-QT syndrome. The medication must be maintained at a dose of 600 mg orally after control of the arrhythmia (24 h without pacing). The combined use of amiodarone with beta-blockers is an



	SPT	VT
V1 V2	rsR'	R or qR
V5 V6 R/S Q present	>1 No	<1 Yes

	SPT	VT
V1 V2	-	R>30 ms R S>60 ms S
V5 V6 Q present	No	Yes

Figure 2. Simplification of the Brugada and Vereckei criteria for differential diagnosis of wide-QRS tachycardia.

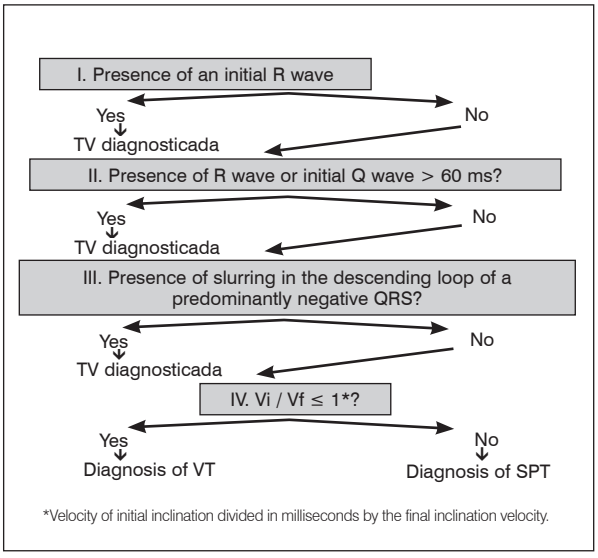


Figure 3. Simplification of the Brugada and Vereckei criteria for differential diagnosis of wide-QRS tachycardia.

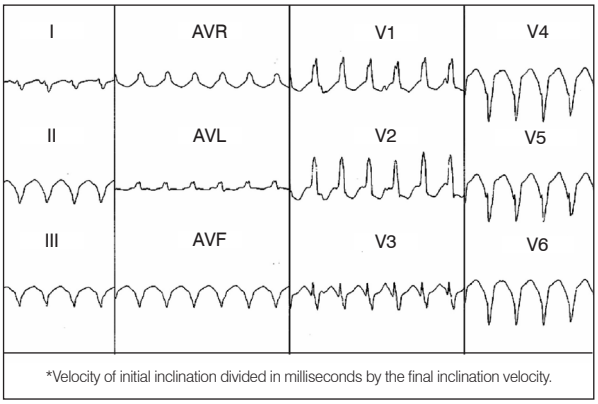


Figure 4. Example of stable, sustained tachycardia with RBBB morphology. Both the first step in the Vereckei algorithm (pure R in aVR), and the third and fourth steps in the Brugada algorithm (AV dissociation; R monophasic in V1 and Q wave in V6), confirm VT, in this case, secondary to Chagas cardiomyopathy.

option, both for acute control and prevention of relapse.[15-17] In a study to determine the optimal beta-blocker, Chatzidou et al.[16] randomly and prospectively assessed 60 patients with an ICD and ES, in a 1:1 randomized, double-blind treatment protocol using propranolol (40 mg orally every 6 h) versus metoprolol (50 mg orally every 6 h). Any secondary causes for the initial presentation were excluded, and all patients were already using amiodarone. The authors demonstrated that patients treated with propranolol had a shorter hospitalization time, with significant reduction in arrhythmic events and number of ICD shocks in the first 48 h. These results clearly indicate that propranolol was a better antiarrhythmic drug than metoprolol for the acute treatment of ES in patients who are already using amiodarone.

The use of group I antiarrhythmic agents, such as propafenone and quinidine, should be avoided, since some studies have shown that these drugs act as independent factors in the onset of ES.[15] However, quinidine has been useful in the control of repetitive tachyarrhythmia in Brugada syndrome.[18] In refractory cases, in which the control of tachyarrhythmia with antiarrhythmic agents is not possible, VT substrate ablation shows good results, especially when using electro-anatomic mapping and an epicardial technique.[19]

NON-PHARMACOLOGICAL TREATMENT

The catheter intervention approach should always be considered when initial pharmacological measures and adjustment of electronic devices are not effective. Almost all patients with ES have structural heart disease and varying degrees of ventricular dysfunction, which can often be the cause of worsening arrhythmia, as in cases of advanced cardiac insufficiency, coronary artery disease, and reinfarction. The therapeutic approach may include circulatory support for patients in cardiogenic shock, hemodynamic intervention in stent occlusion, or even sympathetic blockade.

However, some cases of ES can occur in the absence of demonstrable structural heart disease and may require a more specialized approach, since some of these cases are due to rare channelopathies. To demonstrate their complexity, we reported a case of ES with recovery from cardiac arrest triggered by EV with ultrashort coupling, in which several episodes of spontaneous TdP-type polymorphic VT occurred. After several failed treatment attempts, isoproterenol infusion was performed until the heart rate decreased to 100 bpm, and was able to suppress ventricular arrhythmia this was followed by implantation of an electrode in the right atrium, with stimulation at a rate of 85 bpm in the acute phase.[20] The use of high-dose verapamil and ICD implantation were subsequent therapeutic procedures. Thus, differentiated and specific measures may be necessary to interrupt ES.

The use of catheter ablation in VT should consider the risks and benefits of the procedure, patient characteristics, technical conditions, and operator experience; the electrophysiology laboratory should be able to perform both endocardial and epicardial approaches.[21,22] The risk-benefit ratio is favorable in cases of monomorphic VT, including VT interrupted by ICD shock that recurs despite antiarrhythmic therapy, particularly in cases of ES that are not clinically controlled.[22,23]

Other options in specialized centers include attempts at sympathetic block, e.g., renal denervation via catheter and sympathectomy, with promising results.[24-27] In patients with ES, bilateral sympathectomy seems to be more beneficial than left sympathectomy. The beneficial effects of bilateral sympathectomy go beyond the acute period, with absence of ICD shocks in about 48% of patients and significant reduction in the number of shocks in 90%.[26,27]

CONCLUSION

Most patients who present with sustained VT or ES have structural heart disease and the substrate of scar-related reentry is the most prevalent. Etiological and electrocardiographic diagnosis is critical for emergency and long-term treatment strategies, which include clinical management (amiodarone, beta-blockers, sedation, control of electrolytes, correction of precipitating factors, etc.) as well as specific interventions, e.g., programming of electronic devices (pacemakers and defibrillators), electrophysiological intervention (endocardial and/or epicardial ablation), and specific intervention for sympathetic blockade (renal denervation, sympathectomy).

CONFLICTS OF INTEREST

The author declares that he has no conflicts of interest in this work.

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