Revista da
SOCESP
Sociedade de Cardiologia do Estado de São Paulo

ARRYTHMIAS IN THE EMERGENCY ROOM AND ICU. NARROW QRS TACHYCARDIAS: BASES FOR CLINICAL APPROACH

ARRITMIAS NA SALA DE EMERGÊNCIA E UTI. TAQUICARDIAS DE QRS ESTREITO: FUNDAMENTOS PARA A ABORDAGEM

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Received on 06/28/2018,
Accepted on 08/14/2018

ABSTRACT

Narrow QRS tachycardias are supraventricular in origin. The clinical history, physical exam, and electrocardiogram in the emergency room are the main tools used to manage this condition. Tachycardias that present haemodynamic instability must be promptly reverted through synchronized electrical cardioversion. Those that present haemodynamic stability may be treated with vagal maneuvers or intravenous drugs. If irregular, they may take the form of atrial fibrillation or atrial flutter, and in this case, the duration of the episode and the thromboembolic risk are evaluated to determine not only the need for anticoagulation, but also the treatment strategy, whether through heart rate or rhythm control. The latter may be achieved through the use of drugs (oral propafenone or intravenous amiodarone) or synchronized electrical cardioversion. The role of the clinician in the emergency room is therefore fundamental in ensuring adequate conduct of episodes of supraventricular tachycardia, especially in prevention or prompt intervention in case of haemodynamic deterioration related to the condition.

Keywords: Tachycardia, Supraventricular; Emergency; Management.

RESUMO

As taquicardias de QRS estreito apresentam origem supraventricular. O histórico clínico, exame físico e eletrocardiograma na sala de emergência constituem-se nas principais ferramentas para o tratamento do quadro. As taquicardias que apresentam instabilidade hemodinâmica devem ser, imediatamente, revertidas através de cardioversão elétrica sincronizada. Aquelas que se apresentam como estáveis hemodinamicamente podem, se regulares, ser tratadas através de manobras vagais ou através do uso de fármacos endovenosos. Se irregulares, podem caracterizar fibrilação e flutter atrial, sendo, então, avaliados a duração do episódio e o risco de tromboembolismo para determinar não apenas a necessidade de anticoagulação, mas também a estratégia para tratamento do quadro, seja através do controle da frequência cardíaca ou do controle do ritmo, este último podendo ser alcançado através do uso de fármacos (propafenona oral ou amiodarona endovenosa) ou da cardioversão elétrica sincronizada. Dessa forma, o papel do clínico na sala de emergência é fundamental para garantir a condução adequada dos episódios de taquicardia supraventricular, especialmente, na prevenção ou pronta intervenção em caso de deterioração hemodinâmica relacionada ao quadro.

Descritores: *Taquicardia Supraventricular; Emergência; Tratamento.*

INTRODUCTION

Narrow QRS tachycardias are cardiac rhythm disorders characterized by a heart rate > 100 beats per minute (bpm) and narrow QRS complexes (QRS < 120 ms). They originate in any cardiac structure situated above the bifurcation of the bundle of His (sinoatrial node, atrial myocardium, atrioventricular node, or bundle of His), activate the ventricles by the His-Purkinje system (HPS), and have narrow QRS complexes. All narrow QRS tachycardias are supraventricular in origin.[1]

The prevalence of supraventricular tachycardia (SVT) in the general population is 2.29 for every 1,000 people; the incidence is approximately 89,000 new cases per year. SVT is responsible for approximately 50,000 visits in the emergency room per year.[2,3]

Symptomatic SVTs are medical emergencies that require immediate attention due to the potential for related hemodynamic deterioration. In this scenario, the role of the doctor in the emergency room is of fundamental importance for ensuring the correct diagnosis and proper case conduct, thereby minimizing potential complications and adverse outcomes.

http://dx.doi.org/10.29381/0103-8559/20182803276-85

CLASSIFICATION, EPIDEMIOLOGY, AND ELECTROCARDIOGRAPHIC CHARACTERISTICS

Electrocardiography is the main diagnostic tool for evaluating tachyarrhythmias in the emergency room. This tool allows one to classify tachycardias as narrow QRS (<120 ms) or wide QRS (≥120 ms) complexes. All narrow QRS tachycardias are, by definition, supraventricular. Wide QRS (≥ 120 ms) tachycardias may be of supraventricular or ventricular origin (Figure 1).[1]

Approximately 10% of patients with SVT have intraventricular conduction aberrations consisting of either left bundle branch block or frequency-dependent bundle branch block. These cases are encompassed in the differential diagnoses of wide QRS tachycardias (≥ 120 ms), including ventricular tachycardia, pre-excited SVT, atrioventricular reentrant tachycardia (AVRT) in Wolff-Parkinson-White syndrome (WPWS), and tachycardia mediated or conducted by a cardiac pacemaker.[1]

Other diagnostic criteria, such as RR interval regularity, presence and morphology of P-waves, P:QRS ratio, and the relationship between the RP/PR intervals (Figure 2), among others, can allow the specific diagnosis of narrow QRS tachycardia[1] as long as they are used systematically according to the algorithm in Figure 3.

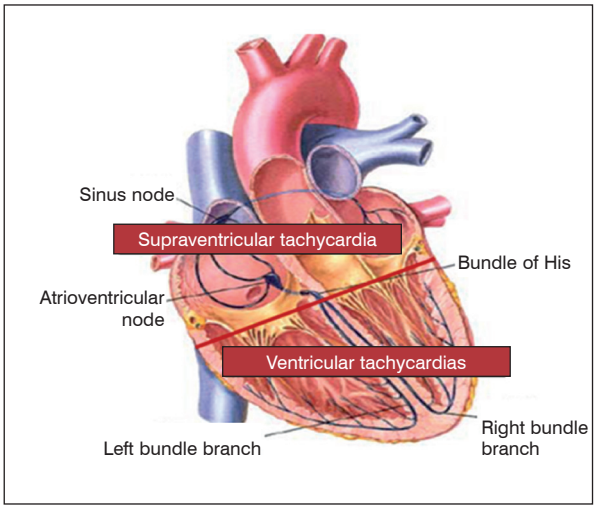

Figure 1. Classification of tachycardia according to supraventricular or ventricular origin.
Taquicardias Supraventriculares = supraventricular tachycardia

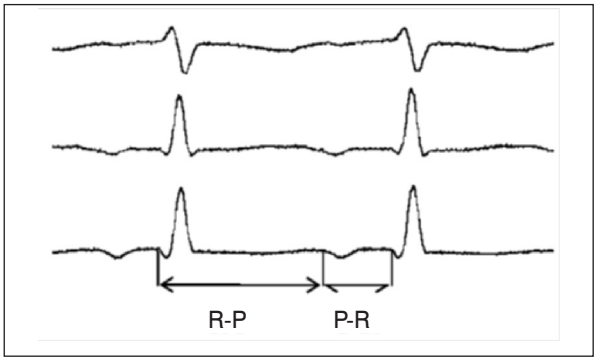

Figure 2. RP and PR intervals. In the differential diagnosis of PSVT, the relationship between them allows one to divide them into short RP tachycardias (RP < PR) and long RP tachycardias (RP > PR).

Regularity is defined as <10% variation between RR intervals. The main tachycardias with a regular RR interval are atrioventricular nodal reentrant tachycardia (AVNRT), AVRT in WPWS, and atrial tachycardia (AT) .[4] These tachycardias are characterized by sudden onset and termination and, therefore, receive the generic denomination of paroxysmal supraventricular tachycardia (PSVT).[4] Sinus tachycardia is an important differential diagnosis of PSVT, mainly in patients with commonly associated clinical conditions (fever, anemia, or hyperthyroidism, for example) and when there is a clearly defined paroxysm. The presence of sinus P waves (positive in the inferior leads and negative in aVR) preceding each QRS complex defines the diagnosis. The main arrhythmia that evolves with irregularity between the RR intervals is atrial fibrillation (AF). However, atrial flutter (AFL) and AT with variable atrioventricular conduction and multifocal AT may also present this irregularity.[5]

Porter et al. reported an approximate prevalence of the major PSVT of 60% for AVNRT, 30% for AVRT, and 10% for AT in patients who underwent an electrophysiological study (gold standard for diagnosis) between 1991 and 2005 (Figure 4).[5]

AVNRT is characterized by a narrow QRS complex tachycardia, regular RR, P waves that are not visible (because they are masked by the QRS complex) or visible as retrograde P in their final portion (pseudo-R' in V1, pseudo-S in inferior leads, and final notch in aVL) defining an RP range < PR and RP < 70 ms. In the typical form (90%), atrioventricular anterograde conduction occurs by the slow pathway, while the ventricular-atrial retrograde conduction occurs through the fast pathway (Figures 5 and 6). In the atypical form (10%), anterograde conduction occurs through the fast pathway and retrograde conduction through the slow pathway. Therefore, what we observe in this case is a long RP tachycardia (RP > PR)[1,2] as shown in Figure 7.

In a report of a Brazilian service that evaluated 256 patients undergoing an electrophysiological assessment,[6] Filgueiras Medeiros et al. corroborated the main electrocardiographic predictors of a diagnosis of AVNRT, namely the presence of pseudo-S in the lower leads and the presence of pseudo-R› in lead V1. In addition, age > 60 years and female sex were statistically significant parameters as clinical predictors of the occurrence of AVNRT.

AVRT, which occurs in patients with WPWS, is characterized by the presence of an accessory pathway connecting the atrial to the ventricular myocardium. Conduction through the accessory pathway can be anterograde (atrioventricular) or retrograde (ventriculoatrial). In patients who present with antegrade conduction by an accessory pathway, the ECG in sinus rhythm can present the classic clinical findings of ventricular pre-excitation: short PR interval, QRS complex with initial slurring (delta wave), and secondary changes in ST segment and T wave. These isolated findings define the electrocardiographic pattern of WPWS (Figure 8). In symptomatic patients, it defines WPWS. In the orthodromic form (90%), the stimulus is conducted to the ventricles by the AV node returning to the atria by the accessory pathway (Figure 9 A). The ECG shows a narrow QRS tachycardia, regular RR, retrograde P waves located in the ST segment or T wave, and an RP interval < PR (Figure 9B). In the antidromic form, the stimulus is conducted to the ventricles by the accessory pathway returning to the atria by the AV

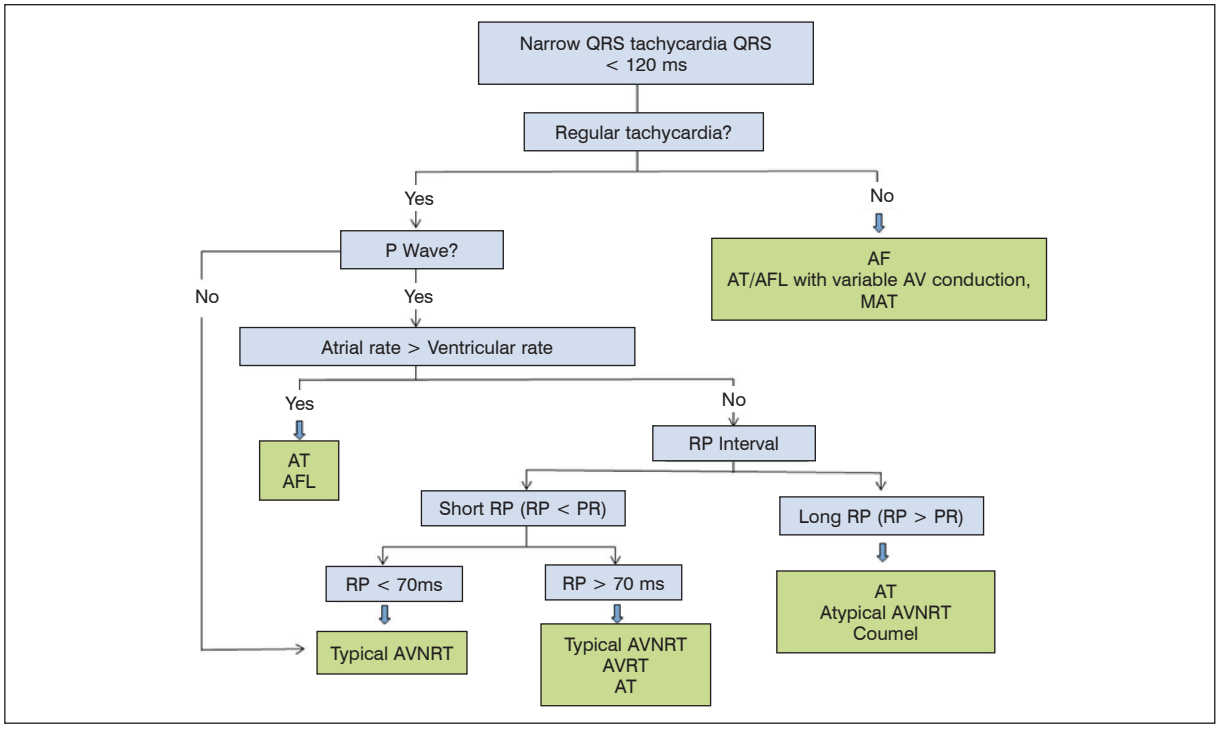

Figure 3. Algorithm for the differential diagnosis of narrow QRS tachycardias. AF, atrial fibrillation; AFL, atrial flutter; TA, atrial tachycardia; MAT, multifocal atrial tachycardia; AVNRT, atrioventricular nodal reentry tachycardia; AVRT, atrioventricular reentry tachycardia.

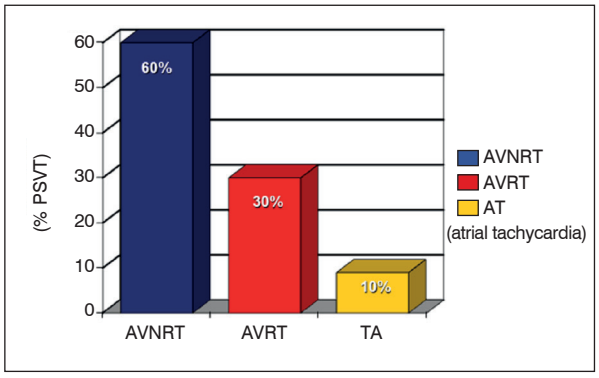

Figure 4. Approximate prevalence of major PSVT.[5]

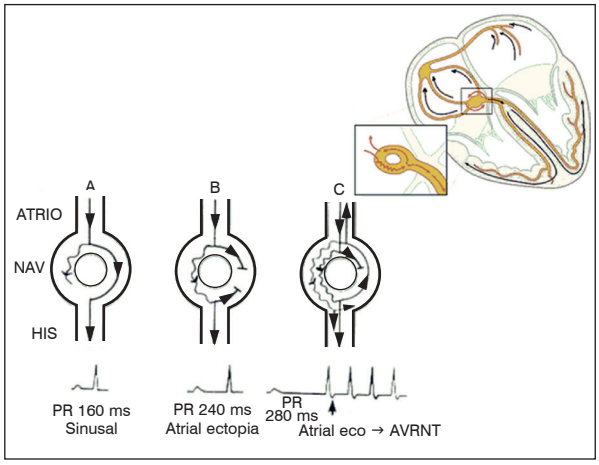

Figure 5. Reentry mechanism of typical AVNRT.

node. In this case, the electrocardiographic pattern is a tachycardia with a wide QRS.[1,2]

AF is the most common chronic arrhythmia, with age being the main predisposing factor. AF may occur in healthy individuals without any disease (*lone AF*), but is more common in the presence of structural heart disease. It can also be related to reversible or transitory factors such as thyrotoxicosis or after cardiac surgery. Due to the predisposition to the formation of thrombi in the left atrial cavity or in the atrial appendage, AF is associated with increased risk of thrombo-embolism events, particularly stroke. An absence of P waves can be noted on the ECG and the presence of small oscillations at baseline, with variable amplitude and morphology, called F waves, and an irregularly irregular RR interval (Figure 10). From an electrophysiological point of view, it is characterized by chaotic disorganized atrial depolarization, with multiple wave fronts in the atrial myocardium (450 and 700 cycles per minute) but without effective atrial contraction. The ventricular rate achieved is highly variable and may be higher or lower depending on the refractory period of the AV node. Excessively fast frequencies can cause ischemia, myocardial dysfunction, or hemodynamic instability.[7-9]

Cavotricuspid isthmus (CTI)–dependent AFL, the most common (90% cases) type, can be divided into typical or counter-clockwise FLA and reverse typical or clockwise FLA according to the direction of the wave front in that circuit. CTI-independent or atypical FLA is less common (10%) and can occur, for example, in surgical scars in the late postoperative period after the correction of congenital and acquired cardiopathies. In the typical counter-clockwise FLA ECG, a baseline with P-waves in the form of saw teeth, negative in inferior leads and positive in V1 with a frequency of 250 to 350 cycles per minute and absence of isoelectric line, is observed (Figure 11).

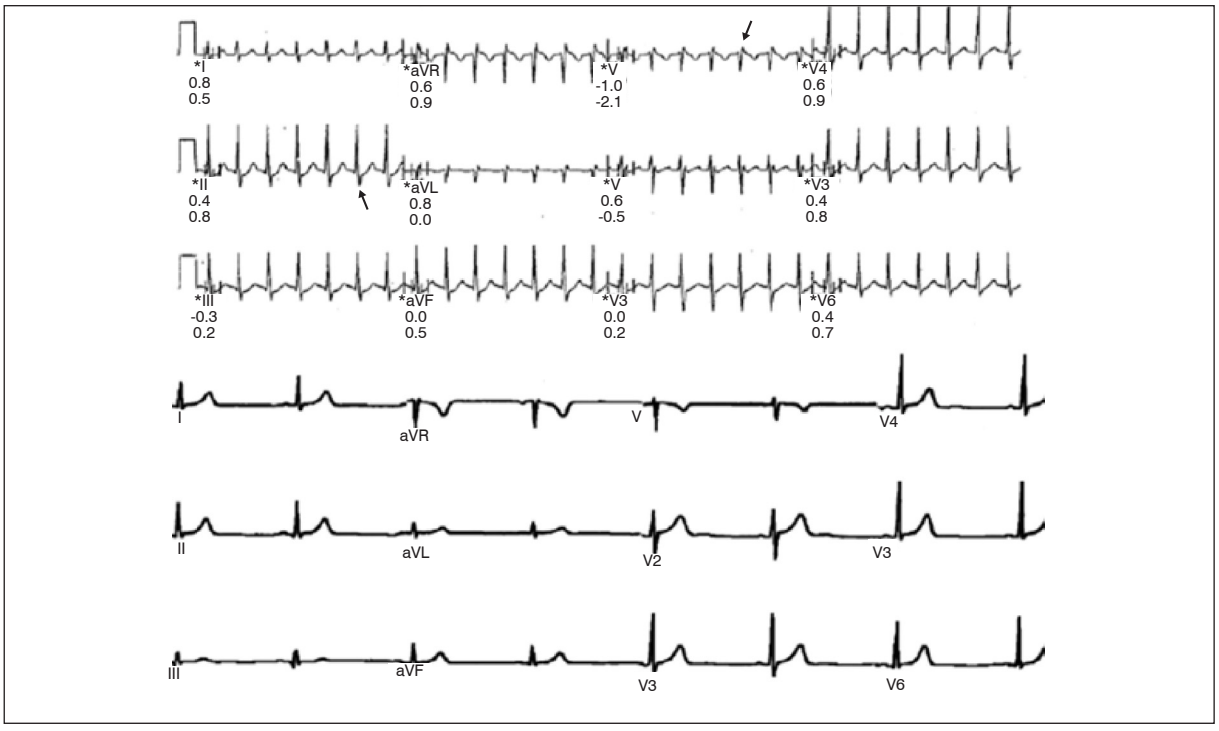

Figure 6. A 67-year-old woman entered the emergency room with palpitations. Electrocardiogram findings were suggestive of typical AVNRT. Note the presence of pseudo R in V1 and pseudo-S in the Inferior leads during tachycardia (arrows). After reversal with adenosine, note the absence of changes in sinus rhythm.

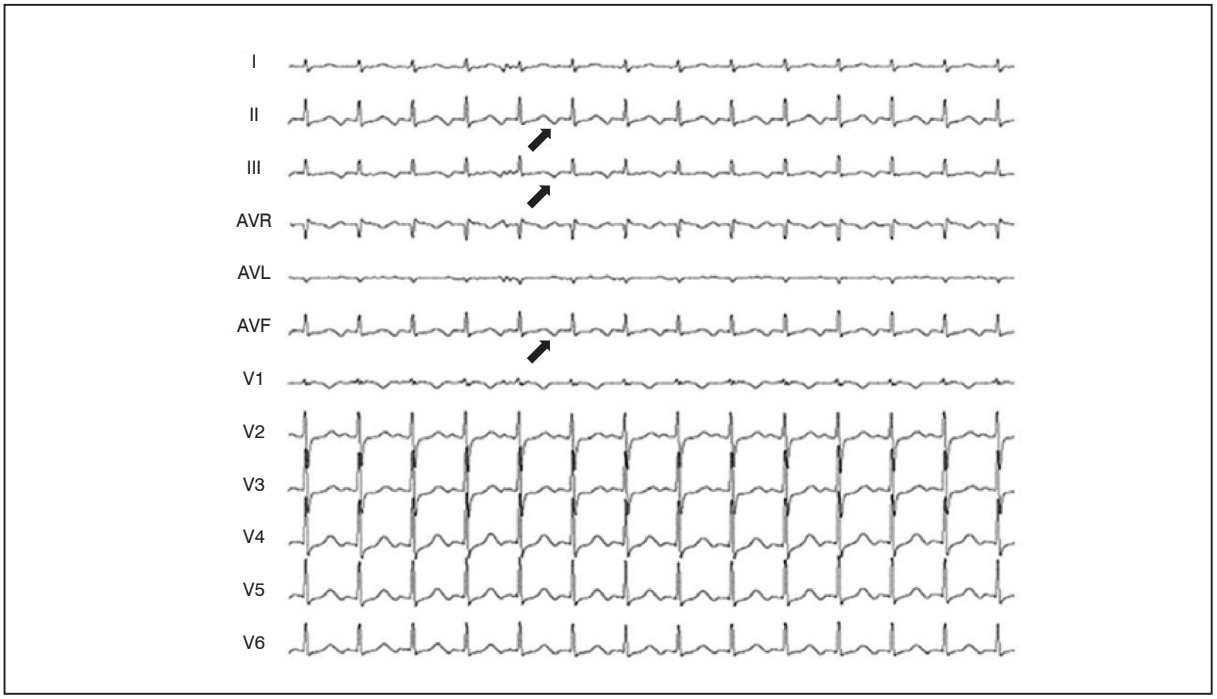

Figure 7. A 52-year-old man entered the emergency room with a 2-hour history of tachycardia palpitations. Electrocardiogram findings were suggestive of atypical AVRNT. Observe the negative retrograde P wave in the Inferior leads (arrows) and an RP > PR interval.

In the typical reverse or clockwise FLA, the ECG shows F waves with a sinusoidal format and positive F waves in the inferior leads and negative in V1 and frequency of 250–350 cycles per minute. The heart rate in AFL will depend on the degree of AV block (more common 2:1 conduction, resulting in a heart rate of 150 bpm) and ventricular rhythm is usually regular but may be erratic when AV conduction is variable. In cases in which the ventricular rate is too high, one can use vagal maneuvers or adenosine that increase the degree of AV block and facilitate visualization of the F waves. This is seen more frequently in patients with COPD or structural cardiopathies and is not very common in normal hearts.[7-9]

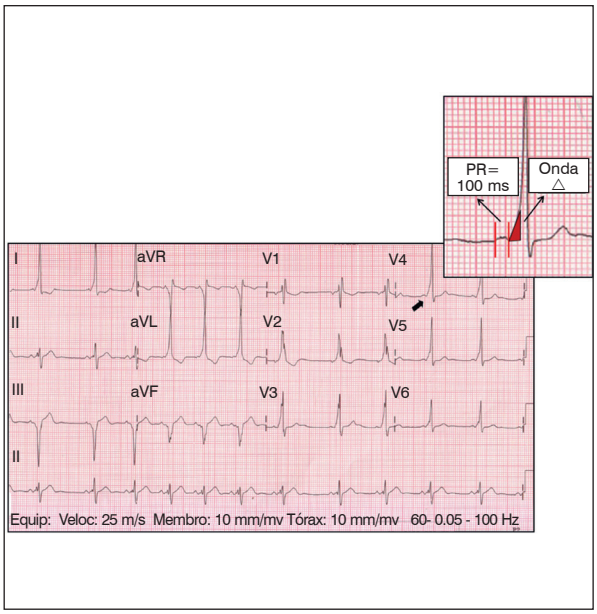

Figura 9. A 21-year-old man demonstrating Wolff-Parkinson-White pattern on the electrocardiogram. Note the short PR interval and the delta wave (arrow and with details in the enlarged image).

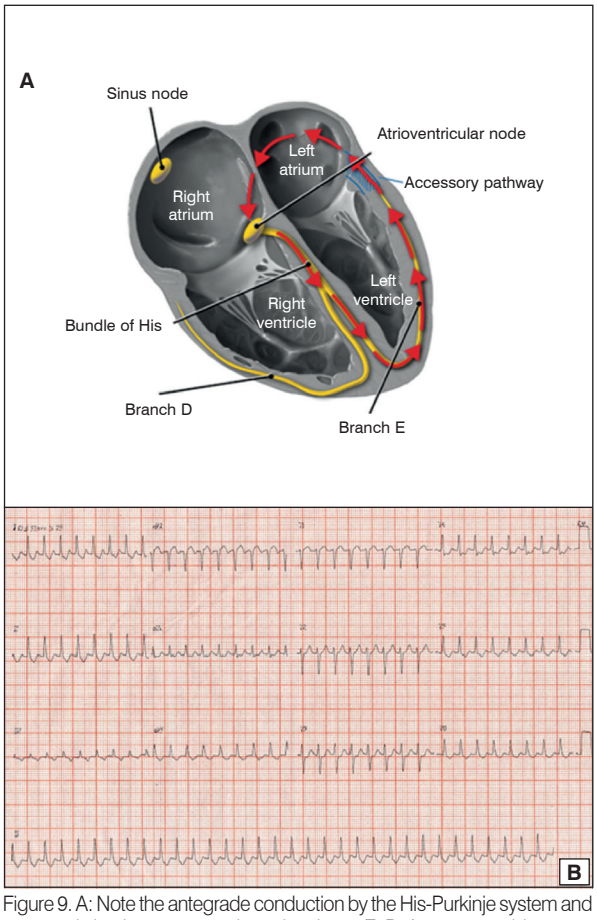

Figure 9. A: Note the antegrade conduction by the His-Purkinje system and retrograde by the accessory lateral pathway E. B: An 18-year-old woman with a history of palpitations and pre-syncope crises since childhood was referred for a prolonged episode. Electrocardiogram findings were suggestive of orthodromic atrioventricular reentrant tachycardia. Observe the negative retrograde P wave in the inferior leads inscribed within the ST segment.

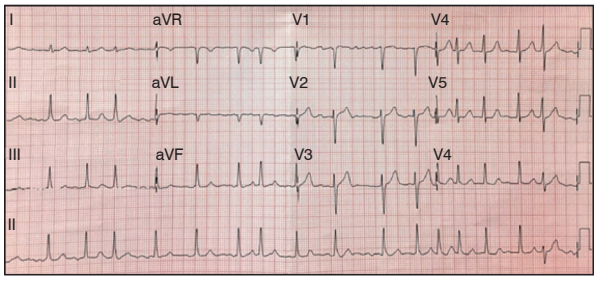

Figure 10. A 73-year-old patient with coronary disease and recurrent episodes of palpitations for 1 week. Electrocardiogram findings were suggestive of atrial fibrillation. Note the irregularity of the RR intervals, the absence of the P wave, and the presence of F waves.

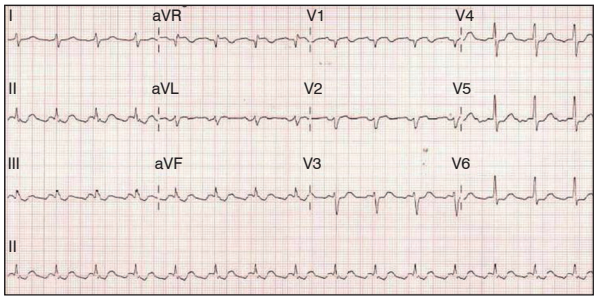

Figure 11. A 66-year-old patient with chronic obstructive pulmonary disease referred for a 3-day history of worsening dyspnea. Electrocardiogram findings were suggestive of typical counter-clockwise atrial flutter. Observe the negative F waves in DII, DIII, and aVF and positive waves in V1.

INITIAL APPROACH IN EMERGENCY SITUATIONS

The main symptoms that patients with SVT present are palpitations, chest pain, dizziness, fatigue, and dyspnea.[1,2] Signs of low cardiac output and syncope are rarer in this group of arrhythmias, and their presence demands the assessment of structural cardiopathy.[1-3] In the emergency room, bedside transthoracic echocardiography can be useful in these cases.

Some symptoms indicate more specific presentations such as the sensation of palpitations in the anterior cervical region (frog sign) denoting simultaneous atrial and ventricular activation with the atria contracting against the closed atrioventricular valves, which occurs by AVNRT.[10] Erratically irregular pulses are suggestive of AF or AFL/AT with variable AV conduction.[3-5]

Nevertheless, tachycardia frequently terminates spontaneously before the electrocardiographic recording in the emergency room. In these cases or when the symptoms are nonspecific, the external event monitor (loop recorder) can be an interesting alternative for elucidation of palpitations as observed by Epifanio et al. in a study of 112 patients.[11] This is because the correlation between symptoms and significant cardiac arrhythmias (SVT, AF, ventricular tachycardia) is poor.

On arriving at the emergency service, patients with such symptoms or changes in the assessment of vital signs (such as tachycardia, hypotension, tachypnea, and decreased oxygen saturation) should be forwarded to the emergency room for immediate cardiac monitoring, oxygen therapy (if hypoxemic), and venous access catheters for drug administration.[1,2] A physical examination directed to the clinical condition should be conducted, including vital signs and cardiopulmonary

evaluation, level of consciousness, and peripheral perfusion in an active search for signs of hemodynamic instability as described below.[1,2]

Tachyarrhythmia potentially causing hemodynamic instability are those in which the heart rate is >150 bpm. However, in patients with structural cardiopathy, especially in patients with valvulopathy (significant aortic and mitral stenosis) or significant ventricular dysfunction FE ≤ 35% (ejection fraction), lower heart rates may be poorly tolerated and progress with signs of instability.[1,2]

The main guidelines, including the latest revision of *Advanced Cardiac Life Support* of 2015, highlights the following markers of hemodynamic instability: hypotension, angina pectoris, cardiogenic shock, acute changes in mental status, and signs of congestive heart failure.[2,3] Patients with a narrow QRS complex tachycardias who present with any of the above criteria should undergo electrical cardioversion (ECV) on an emergency basis.[3] Such patients may be monitored only by the cardiac monitor, with a recording performed by the electrocardiograph on the cardioverter/defibrillator, dispensing with the 12-lead electrocardiogram.

However, those patients who present with hemodynamic stability must undergo electrocardiography in the emergency room in up to 10 minutes, as it has a fundamental role in the diagnosis and management of the presentation in this scenario. The algorithm for the initial approach and conduction of SVT in emergency rooms is shown in Figure 12.

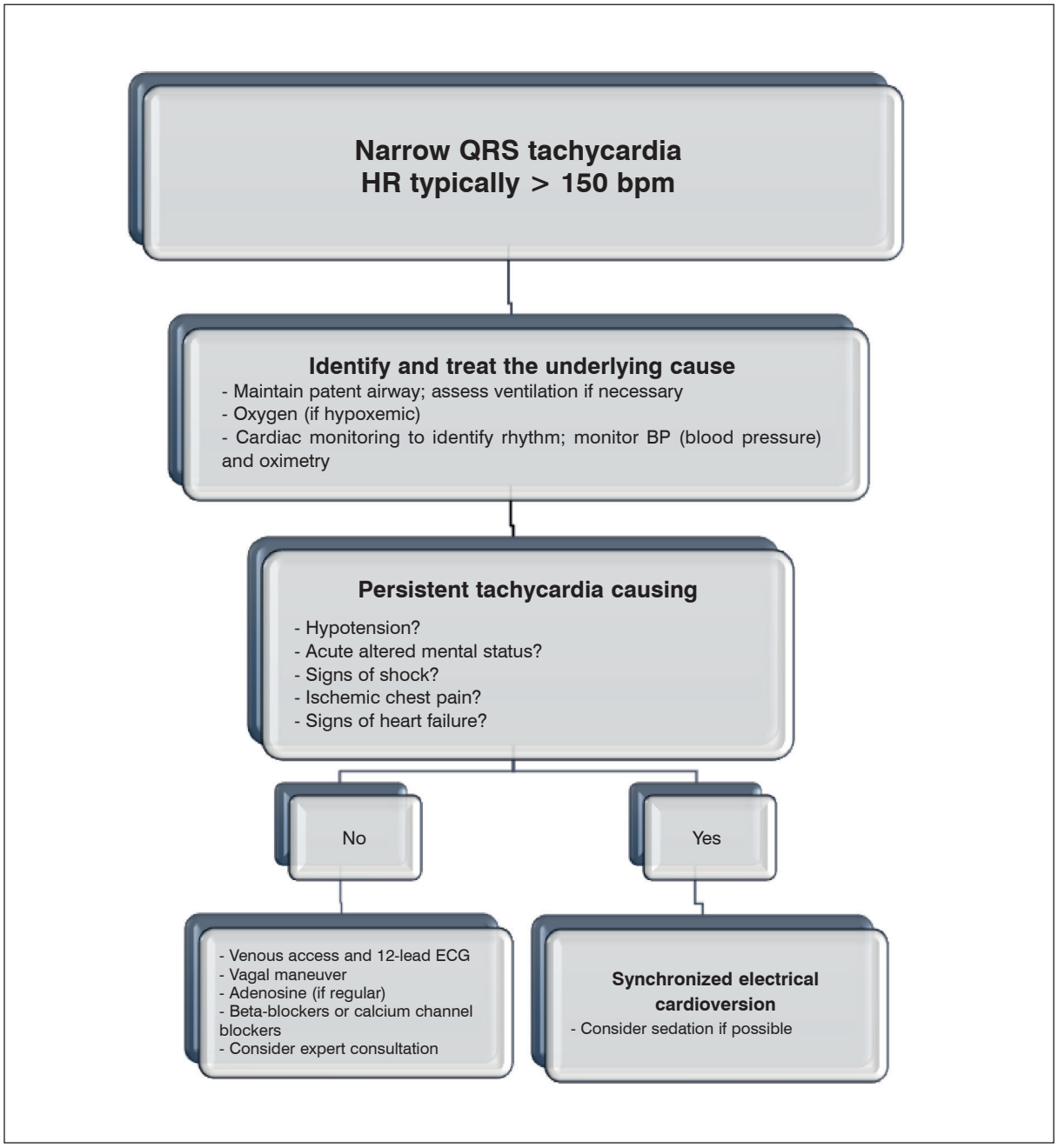

Figure 12. Algorithm for the treatment of unstable and stable SVT.[2]

TREATMENT OF HEMODYNAMICALLY UNSTABLE SVT

Patients who show signs of clinical instability, as already noted, should be immediately subjected to synchronized ECV (electrical cardioversion) with a biphasic shock of 100 J (can be increased to 150 J in cases of failure of the first shock).

Sedation should always be performed whenever possible. The drug of choice in the presence of instability is etomidate considering its lower potential to exacerbate the patient's already deteriorating hemodynamic condition. However, it can be waived in patients with important signs of shock or significantly decreased consciousness.[1,2] The absence of adequate fasting (6-8 hours) should not be contraindicated for the ECV given the possibility of hemodynamic deterioration and imminent risk of progression to cardiorespiratory arrest. In these cases, the team should be prepared and take the necessary measures to mitigate aspiration and its consequences.

Patients with AF and AFL and hemodynamic instability should not have the ECV postponed to take into account the potential of thromboembolism and should subsequently receive the appropriate anticoagulation.[7-9]

TREATMENT OF HEMODYNAMICALLY STABLE SVT

Regular SVT

The first measure for the treatment of stable PSVT with regular RR aims to revert the rhythm or slow the atrioventricular conduction. For both, vagal maneuvers are used (carotid sinus massage [CSM], Valsalva maneuver) with efficacy of 19.4–54%.[12-14] CSM is contraindicated in patients with a history of transient ischemic attack (TIA) or ischemic stroke (IS) as well as those with audible murmur on auscultation of the carotid arteries. The risk of atheroembolism during CSM in elderly patients should be stressed, even in the absence of an audible murmur.

A recent study detailed the performance of a modified Valsalva maneuver with elevation of the limbs at the end demonstrating approximately 43% efficacy in the reversion of PSVT.[15] Whatever the vagal maneuvers performed, the duration is 10–15 seconds and two attempts are reasonable, with failure to revert an indication of intravenous drug administration in hemodynamically stable patients. In the absence of reversion with the vagal maneuvers, drug treatment must be administered.[16]

The first line of treatment for regular tachyarrhythmias with narrow QRS complex in stable patients is adenosine (6 mg IV bolus followed by 20 mL of 0.9% SF and limb elevation). A second dose (12 mg) may be required in some patients. Adenosine is contraindicated in patients with unstable conditions, asthmatics, and those with angina pectoris.[16,17] One should inform the patient about the effects (malaise, dyspnea, temporary AV block) before the drug administration. It should be remembered that adenosine shortens the atrial refractory period, which may lead to AF, and is contraindicated in patients who have manifest pre-excitation on the ECG due to the risk of AF driven by the accessory pathway leading to ventricular fibrillation and cardiac arrest.[16,18] In cases of failure of this medication to reverse tachyarrhythmia, non-dihydropyridine calcium channel blockers (verapamil or diltiazem) or short-acting beta-blockers (metoprolol or esmolol) may be administered.[16,19]

Non-dihydropyridine calcium channel blockers act on slow calcium channels, being negative inotropic and chronotropic, causing blockage of the sinus node and atrioventricular node. Attention should be given to the use of calcium channel blockers in the emergency setting due to the possibility of significant arterial hypotension. These drugs should not be used in cases of signs of congestive heart failure or in patients with tachycardia and a wide QRS even though the presumptive diagnosis is SVT, especially in those cases where there is a suspicion of the presence of accessory pathway with signs of ventricular preexcitation.[16,18]

Beta-adrenergic receptor blockers are chronotropic, dromotropic, and negative inotropes that reduce the automatism of the sinus node, block the atrioventricular node, and are indicated in PSVT non-responsive to vagal maneuver or adenosine. It should be remembered that, besides the potential to cause hypotension, beta-blockers are contraindicated in asthmatic patients.[16,19]

Moreover, in all attempts of reversion, pharmacologic or not, a new ECG must be recorded, even if there is no reversal of arrhythmia, because the slowing down of the frequency of tachyarrhythmia can provide important data for the diagnosis and treatment of the condition, such as the visualization of P-waves in the case of a TA or F waves in the case of AFL.[16]

In the absence of the reversion of stable conditions, small non-randomized studies showed the efficacy of amiodarone in the reversal of PSVT, although evidence for its use as a first choice agent is lacking.[20] Upon failure of pharmacological reversion or in the presence of signs of hemodynamic impairment, as already noted, immediate synchronized ECV is indicated.[16,21]

Focal atrial tachycardias are poorly responsive to adenosine. In these cases, beta-blockers or non-dihydropyridine calcium channel blockers are considered first-line drugs, with 30–50% efficacy, being especially effective in cases of atrial tachycardia with automatic mechanism. However, in some cases, it may be necessary to use amiodarone to reverse tachyarrhythmia, especially in those patients who present ventricular dysfunction.[22]

A summary of the doses and side effects of the main antiarrhythmic drugs used in emergency situations is presented in Table 1.

After reversal of the condition, the main precipitating causes of the presentation should be identified and treated (hydroelectrolytic disorders, changes in thyroid function, infectious processes, etc.). In their absence, after a brief 2–4-h monitoring period, patients can be discharged from the hospital with referral to a specialist for the management of the condition and evaluation of continuous treatment, either through drug control or an electrophysiological study and ablation, which is particularly effective in nodal reentrant tachycardia and in WPWS-related tachycardias.[16] Inefficacy in reversion of the condition and/or the presence of untreated clinically significant precipitating factors indicates hospital admission.

IRREGULAR SVT - AF AND AFL

In the emergency setting, the AF and the AFL of non-valvular etiology are considered acute with a duration < 48 hours and chronic with a duration > 48 hours. The importance

Table 1. Antiarrhythmic agent doses and side effects.

Agent	Doses	Side Effects
Adenosine	6 mg IV bolus; may repeat 12 mg bolus after 1-2 minutes	Bronchospasm, atrial fibrillation, brief asystole
Amiodarone	Loading: 300 mg IV in 30-40 min Additional: 150 mg in 20-30 min Maintenance: 1 mg/min in 6 h + 0.5 mg/min at 18 h	Phlebitis, hypotension, and bradycardia (rapid infusion)
Metoprolol	5 mg IV in 1-2 min; may repeat every 5 min (maximum dose 15 mg)	Bradycardia, atrioventricular conduction disturbances, bronchospasm, hypotension
Propafenone	450 mg (<70 kg) or 600 mg (>70 kg) (maximum dose: 900 mg/day)	High atrial flutter with 1:1 conduction, bradycardia, gastrointestinal symptoms, sleep disorders, headaches
Verapamil	5-10 mg IV in 1-2min; may repeat every 15-30 min (maximum dose: 20-30 mg)	Severe hypotension, especially in patients with ventricular dysfunction, atrioventricular block, skin rash, nausea, dyspnea, dizziness, paresthesia, psychosis, urticaria, Stevens-Johnson syndrome and, rarely, PCR

of this classification lies in the possibility of forming intracardiac thrombi (Figure 13).[23] In patients with valvular etiology (severe mitral stenosis and valvular prosthesis in the mitral position), this criterion of arrythmia duration is not valid for defining patients at low or high risk of events because these cases should always be treated as high-risk cardioembolic events.

Episodes of AF/AFL of non-valvular etiology with a duration < 48 hours can be managed with an initial attempt to control the pace with or without the need for anticoagulation as long as there is a precise determination of symptom onset.[7] The use of antiarrhythmic agents promotes chemical cardioversion in 50% of cases[24]; however, in the short term, electrical cardioversion restores sinus rhythm more quickly and effectively with a shorter hospitalization.[25] The anteroposterior positioning of the pads promotes passage of higher energy through the left atrium and more effectively achieves sinus rhythm.[26]

If opting for chemical cardioversion, the drug of intravenous choice is amiodarone.[8] Amiodarone mainly blocks potassium channels, increasing cardiac fiber refractoriness to depolarization. It mainly acts on the atrial and ventricular myocardium. Due to the vehicle, it can cause hypotension. It is contraindicated in patients with hyperthyroidism and in those who have already had significant side effects with it. During attempted rhythm reversal, electrolytes should be dosed and any hydroelectrolytic disorders corrected, especially those related to potassium and magnesium.[8,9]

Oral propafenone is a good alternative for those patients with structurally normal heart. If there is a successful reversal of sinus rhythm and without side effects, propafenone can be used in the strategy known as *Pill-in-the-Pocket,* which consists of treating future crises with drugs outside the hospital environment safely and effectively, abolishing the need for a trip to the emergency service.[27]

In cases of valvular AF/AFL and those of non-valvular etiology with an episode duration > 48 hours or indeterminate, effective anticoagulation with warfarin is recommended (target international normalized ratio, 2.0–3.0) or with one of the new oral anticoagulants (NOACs) for a period of 3 weeks before cardioversion should be maintained for a period of 4 weeks or indefinitely in patients at high risk of thrombo-embolism phenomena (CHADS2VASC score ≥ 2).[8,9] There is also the possibility of immediate cardioversion if transesophageal

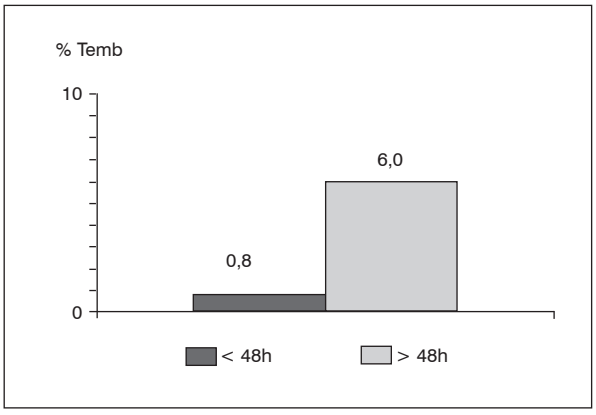

Figure 13. Incidence of thromboembolism (Temb) with atrial fibrillation duration <48 or >48 h.[21]

echocardiography is performed, excluding the presence of thrombus in the left atrium while maintaining the anticoagulation status after the procedure as already cited.[8,9]

The use of NOACs as an alternative to warfarin in the cardioversion scenario was evaluated in several studies such as XVERT for rivaroxaban, EMANATE for apixaban, ENSURE-AF for edoxaban, and others for dabigatran; all of these drugs were safe alternatives to warfarin in the scenario of electrical cardioversion to restore sinus rhythm in the case of AF.[28-31]

The administration of heparin (preferably low molecular weight) should be performed prior to cardioversion in all patients, even those whose AF duration was <48 hours, provided that there is no contraindication to their use, such as recent intracranial bleeding.[9] In this scenario, the same studies that evaluated the NOACs as an alternative to warfarin also evaluated faster protocols of electrical cardioversion with shorter duration of use of such medications before cardioversion. In the XVERT study, for example, there was no difference regarding the strategy of early (1–5 days) or late (3–8 weeks) cardioversion with rivaroxaban compared with warfarin, demonstrating a significant reduction in the time to cardioversion in this scenario with the use of rivaroxaban.[28] The newly published EMANATE study reported similar results for apixaban.[29]

Symptom management in this scenario, with episodes lasting > 48 hours or an uncertain duration, is usually

accomplished through the control of cardiac frequency using beta blockers or calcium channel blockers (diltiazem, verapamil) to control the ventricular frequency. If the patient presents clinically with signs of congestive heart failure, digitalis such as intravenous lanatoside C can be used.[8,9] This drug presents positive inotropic properties but chronotropic and negative bathmotropic properties, reducing the ventricular rate in cases of high ventricular response.[8,9]

After cardioversion (chemical or electrical), the main precipitating causes should be identified and treated (hydroelectrolytic disorders, changes in thyroid function, infectious processes, etc.). In the absence of these conditions, after a brief 24-hour monitoring period, patients may be discharged from the hospital. They should be referred for outpatient follow-up with pacing control and propafenone (in those patients without coronary artery disease, structural heart disease or conduction disorders on ECG), sotalol or amiodarone in other cases, and full oral anticoagulation for 4 weeks if non-valvar AF/AFL or a low thromboembolism risk (CHADS2VASC score < 2), or indefinitely in patients with AF/AFL with valvar etiology or high thromboembolism risk (CHADS2VASC score ≥ 2).[9] Such cases should be referred to the specialist for management of the condition regarding the continuity of anticoagulation and evaluation of treatment strategies, whether consisting of drugs or catheter ablation.[9]

In cases of ineffective treatment in which the rhythm cannot be reversed, ventricular response cannot be adequately controlled, or there is a baseline condition without treatment or adequate compensation, such as an uncompensated cardiac insufficiency, hospitalization is indicated.

CONCLUSIONS

Narrow QRS tachyarrhythmias are medical emergencies. It is essential that the professional who provides the first emergency room care leads the case in order to avoid hemodynamic deterioration and the adverse outcome of the condition. For such, there is well-documented and easily accessible scientific evidence in the literature, directing the treatment appropriately, with rare cases in the emergency scenario where there is a need for expert assessment.

Cases presenting clinical signs of instability should be subjected to synchronized electrical cardioversion with brevity. The remaining cases can be treated medically. Stable PSVT cases can be treated with vagal maneuvers prior to drug treatment.

Patients presenting with AFL and AF should be evaluated for the prevention of thromboembolic events based on etiology (valvular vs. non-valvular), timing of symptom onset, and risk of thromboembolic phenomena using CHADS2VASC scoring.

CONFLICTS OF INTEREST

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

CONTRIBUIÇÕES DOS AUTORES: Cada autor contribuiu individual e significativamente para o desenvolvimento do manuscrito. FS, THV e LCP foram os principais contribuintes na redação do manuscrito. GF e MJOFCI realizaram a revisão crítica do manuscrito e desenvolveram o conceito intelectual do trabalho.

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