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Review

The supraventricular tachycardias: Proposal of a diagnostic algorithm for the narrow complex tachycardias

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ABSTRACT

The narrow complex tachycardias (NCTs) are defined by the presence in a 12-lead electrocardiogram (ECG) of a QRS complex duration less than 120 ms and a heart rate greater than 100 beats per minute; those are typically of supraventricular origin, although rarely narrow complex ventricular tachycardias have been reported in the literature.

As some studies document, to diagnose correctly the NCTs is an arduous exercise because sometimes those have similar presentation on the ECG. In this paper, we have reviewed the physiopathological, clinical, and ECG findings of all known supraventricular tachycardias and, in order to reduce the possible diagnostic errors on the ECG, we have proposed a quick and accurate diagnostic algorithm for the differential diagnosis of NCTs.

Contents

Introduction

The narrow complex tachycardias (NCTs) are defined by the presence in a 12-lead electrocardiogram (ECG) of a QRS complex duration less than 120 ms and a heart rate greater than 100 beats per minute (bpm); those are typically of supraventricular origin, although rarely narrow complex ventricular tachycardias have been reported in the literature [1].

To diagnose correctly the NCTs is an arduous exercise because sometimes those have similar presentation on the ECG. Two studies suggested that medical house staff and attending physicians diagnose NCTs incorrectly in approximately 40% of cases [2,3]. A study by O'Rourke et al. showed that junior doctors operating within an accident and emergency department had difficulties distinguishing paroxysmal supraventricular tachycardia by fast atrial fibrillation and atrial flutter [4]. Other studies have confirmed these data showing the difficulties encountered in distinguishing paroxysmal supraventricular tachycardia from other NCTs [5–7]. In addition to the quota of incorrect diagnosis due to lack of specific knowledge of NCTs, physicians are often misled by excessive trusting of the frequently incorrect computer-generated interpretations of supraventricular tachycardias [8].

The aim of this article is to completely review the physiopathological, clinical, and ECG findings of supraventricular tachycardias and to propose a quick and accurate diagnostic algorithm in order to reduce the possible diagnostic errors in the presence of NCTs.

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The supraventricular tachycardias

The supraventricular tachycardias are usually benign and self-limited arrhythmias and the symptoms associated can be so slight that the some affected people do not seek medical attention. Nevertheless, sometimes they may cause palpitations, dyspnea, diaphoresis, angina, or vague chest discomfort, particularly in patients with underlying heart disease. Serious sequelae such as overt heart failure, myocardial infarction, and syncope are uncommon [9].

The supraventricular tachycardias may be classified into: (1) atrial tachycardias; (2) junctional tachycardia; (3) atrioventricular (AV) node reentrant tachycardia; and (4) AV reciprocating tachycardia utilizing an accessory AV connection.

(1) There are numerous types of *atrial tachycardias*.

Sinus tachycardia (Fig. 1) is defined as an increase in sinus rate to more than 100 bpm. Like in normal sinus rhythm, the P wave's axis in the frontal plane lies between 0 and +90°. The P waves have a normal contour, but a larger amplitude may develop and the wave may become peaked [10]. It is possible to discern:

- *Physiological sinus tachycardia*, that usually occurs as nonparoxysmal in response to an appropriate physiological stimulus (exercise and emotional stress), to pathological causes (hyperthyroidism, pyrexia, hypovolemia, and anemia), and to stimulant drugs (caffeine, alcohol, nicotine, salbutamol, aminophylline, atropine, catecholamines, etc.) [10].
- *Inappropriate sinus tachycardia*, that appears as a persistent nonparoxysmal tachycardia during the day, without secondary systemic causes, due to enhanced automaticity and/or to abnormal autonomic regulation of the sinus node (excess sympathetic and reduced parasympathetic tone). The mean age of presentation is 38 ± 12 years, a high proportion of patients usually are females (90%) and the degree of disability can vary from totally asymptomatic patients to individuals who are fully incapacitated because of palpitations, chest pain, dizziness, etc. [10].
- *Sinus node re-entry tachycardia*, often nonsustained that are due to a re-entry circuit that incorporates the sinoatrial node. They are symptomatic, paroxysmal, and terminate abruptly by an atrial premature beat. The rates of the tachycardia are rarely higher than 180 bpm. Contrary to popular belief, there is a high incidence of underlying organic heart disease [9,10].

Atrial fibrillation (AF) (Fig. 2) is a supraventricular tachyarrhythmia characterized by uncoordinated atrial activation with consequent deterioration of atrial mechanical function [11]. It is the most common arrhythmia in clinical practice, accounting for approximately one-third of hospitalizations for cardiac rhythm disturbances [11]. The most frequent pathoanatomic changes in AF are atrial fibrosis and loss of atrial muscle mass. The principal electrophysiological mechanisms of AF are "focal" mechanisms involving automaticity or multiple re-entrant wavelets; these mechanisms are not mutually exclusive and may at various times coexist in the same patient [11–13]. Common ECG findings of AF are the replacement of consistent P waves by rapid oscillations or fibrillatory waves that vary in amplitude, shape, and timing, associated with an irregular, frequently rapid ventricular response when AV conduction is intact [11,14]. The ventricular response to AF depends on electrophysiological properties of the AV node and other conducting tissues, the level of vagal and sympathetic tone, the presence or absence of accessory conduction pathways, and the action of drugs [11,15]. Although absolutely irregular cardiac cycles (R–R intervals) are common with this arrhythmia, regular cardiac cycles are possible in the presence of AV block or ventricular or junctional tachycardia. A rapid, irregular, sustained wide-QRS-complex tachycardia strongly suggests AF with conduction over an accessory pathway or AF with underlying bundle-branch block [11]. Reversible or secondary AF can occur in the setting of acute myocardial infarction, cardiac surgery, pericarditis, myocarditis, hyperthyroidism, pulmonary embolism, pneumonia, acute pulmonary disease, or other acute illness. In these settings, AF is not the primary problem, and treatment of the underlying disorder concurrently with management of the episode of AF usually results in termination of the arrhythmia without recurrence [11,16].

Atrial flutter is characterized by an organized atrial rhythm with a rate typically between 250 and 350 bpm. Electrophysiological studies have shown that this simple ECG definition includes tachycardias using a variety of re-entry circuits [17]. The re-entry

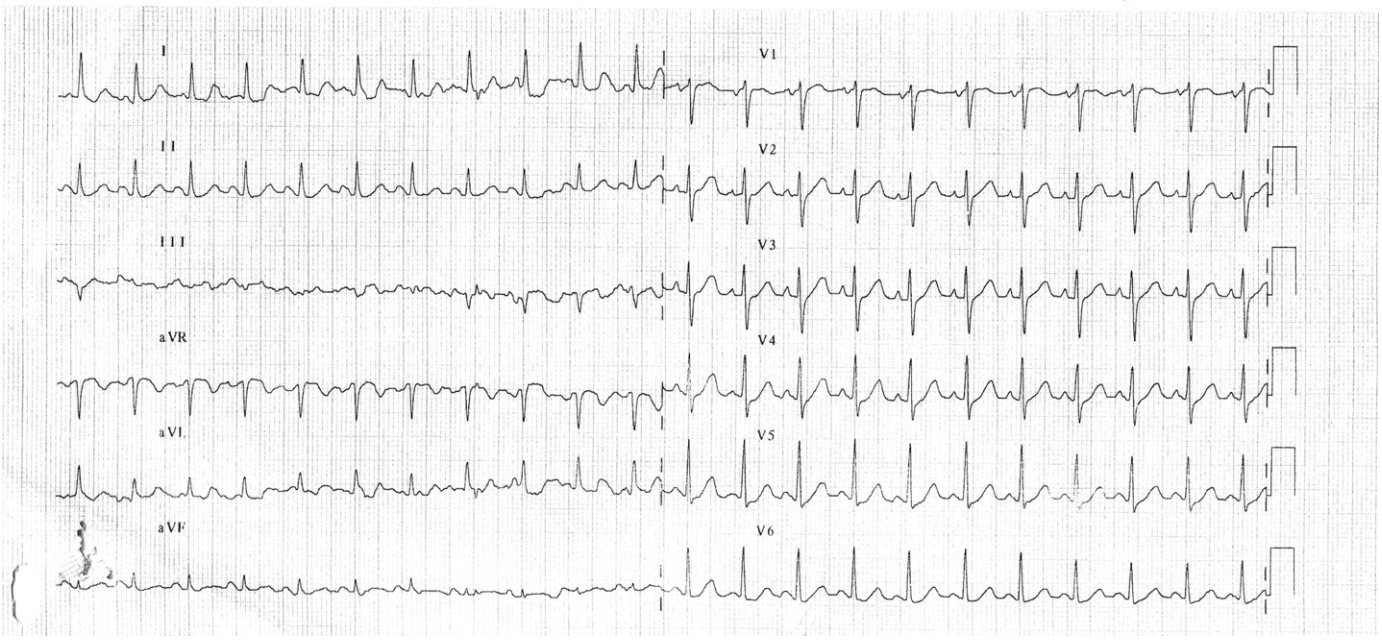

Fig. 1. Sinus tachycardia: a regular tachycardia with P waves visible and P waves' axis between 0 and +90° in the frontal plane.

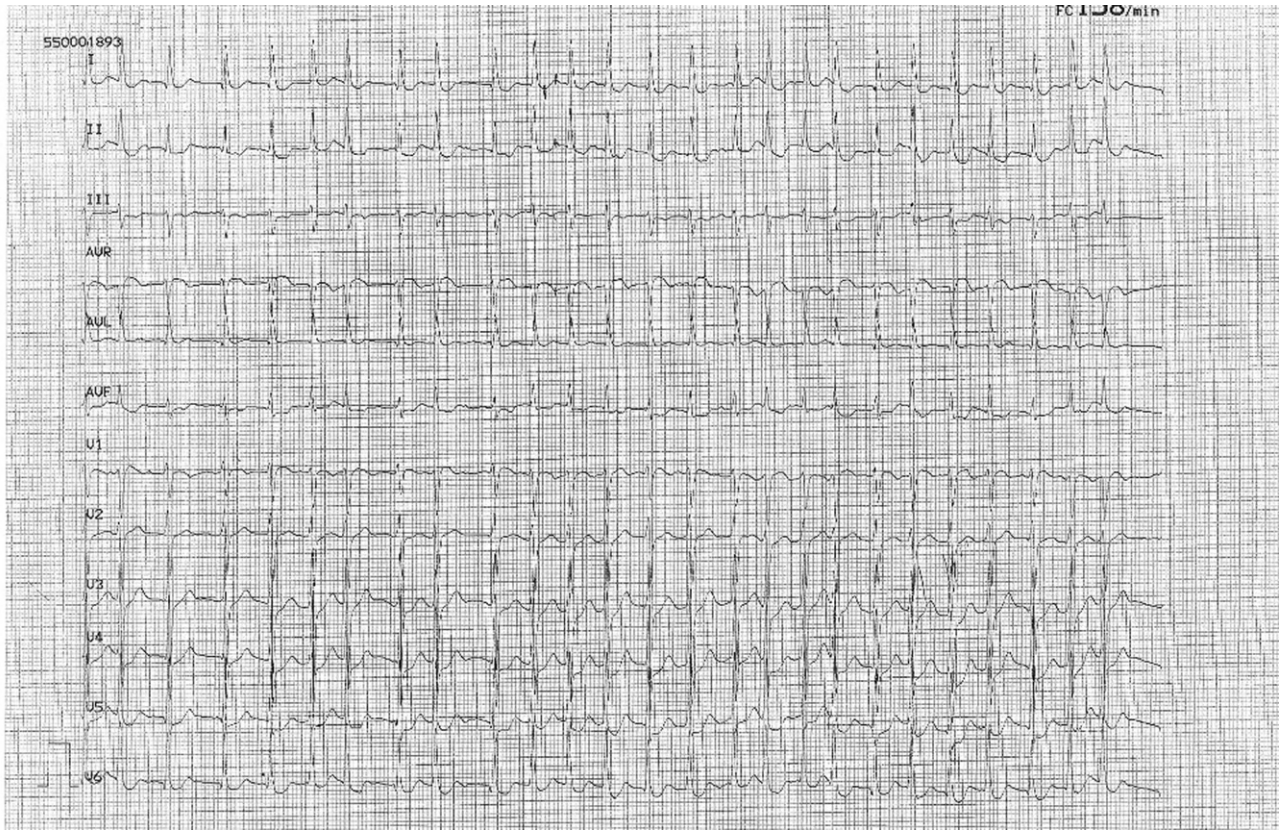

Fig. 2. Atrial fibrillation: an irregular tachycardia without P waves visible.

circuits often occupy large areas of the atrium and are referred to as "macro-re-entrant". The classic type of atrial flutter (i.e. typical flutter) is dependent on the cavotricuspid isthmus. The most common patterns include a tachycardia showing a counter clockwise rotation around the tricuspid valve; on the ECG are dominant negative flutter waves in the inferior leads and a positive flutter deflection in lead V1. A less common pattern involves clockwise rotation around the tricuspid annulus (reverse typical flutter); the ECG shows positive flutter waves in the inferior leads and wide, negative flutter waves in lead V1. Patients may at times show unusual ECG patterns (atypical flutter) [10].

Patients with atrial flutter commonly present with acute symptoms of palpitations, dyspnea, fatigue, or chest pain, but this arrhythmia may also have a clinical presentation with less typical symptoms or signs [10]. Regardless of this, atrial flutter is usually associated with more intense symptoms owing to more rapid ventricular rates. In most instances, patients with atrial flutter present with a two-to-one AV-conduction pattern; in that case, the flutter rate is approximately 300 per minute with a ventricular response of about 150 bpm. Flutter with varying AV block can result in a grossly irregular rhythm (Fig. 3) [10]. Atrial flutter may degenerate into AF and AF may convert to atrial flutter [11].

Focal atrial tachycardias are characterized by regular atrial activation from atrial areas with centrifugal spread. Neither the sinus node nor the AV node plays a role in the initiation or in the perpetuation of tachycardia [10].

Focal atrial tachycardias may be classified into two types:

- *Unifocal atrial tachycardia*, an arrhythmia due to enhanced automaticity, triggered activity, or micro-re-entry. They are characterized by a single P-wave morphologic pattern, the configuration of which depends entirely on the atrial site from which the tachycardia originates [9]. During unifocal atrial tachycardia, an isoelectric baseline is usually present between P waves, and it is used to distinguish atrial tachycardia from typical atrial flutter. The presence of AV block during tachycardia excludes the atrioventricular reciprocating tachycardia and makes the atrioventricular re-entrant nodal tachycardia unlikely [10]. The unifocal atrial tachycardia is usually benign, paroxysmal, and nonsustained; rarely there are incessant forms which may lead to tachycardia-induced cardiomyopathy. The atrial rate is generally between 150 and 250 bpm. An infrequent mechanism of unifocal atrial tachycardia is triggered activity due to digitalis intoxication; this drug-induced atrial tachycardia is usually characterized by development of atrial tachycardia with AV block [9,10].
- *Multifocal atrial tachycardia* (MAT), an arrhythmia characterized by the following electrocardiographic criteria: three or more different morphologic patterns of P waves and an irregular atrial rate averaging 100 bpm or more (generally lower than 250 bpm). Variations in the PR, PP, and RR intervals are common [18]. Isoelectric periods between adjacent P waves help to distinguish this arrhythmia from atrial fibrillation [9]. The putative mechanism is enhanced automaticity, although triggered activity is also a possibility [19]. Sixty percent of patients with this arrhythmia have pulmonary disease [19], but it may result from metabolic or electrolyte derangements. It is seldom caused by digitalis excess [10].

(2) The *junctional tachycardia* is an uncommon arrhythmia that may be due to either abnormal automaticity or triggered activity of the AV node or of the His bundle [9,10,18,20]. Electrocardiographically, this arrhythmia appears as a regular NCT with either AV dissociation or one-to-one retrograde conduction AV (Fig. 4) [9]; in that case, the P waves are negative in surface electrocardiographic leads II, III, and aVF and positive in

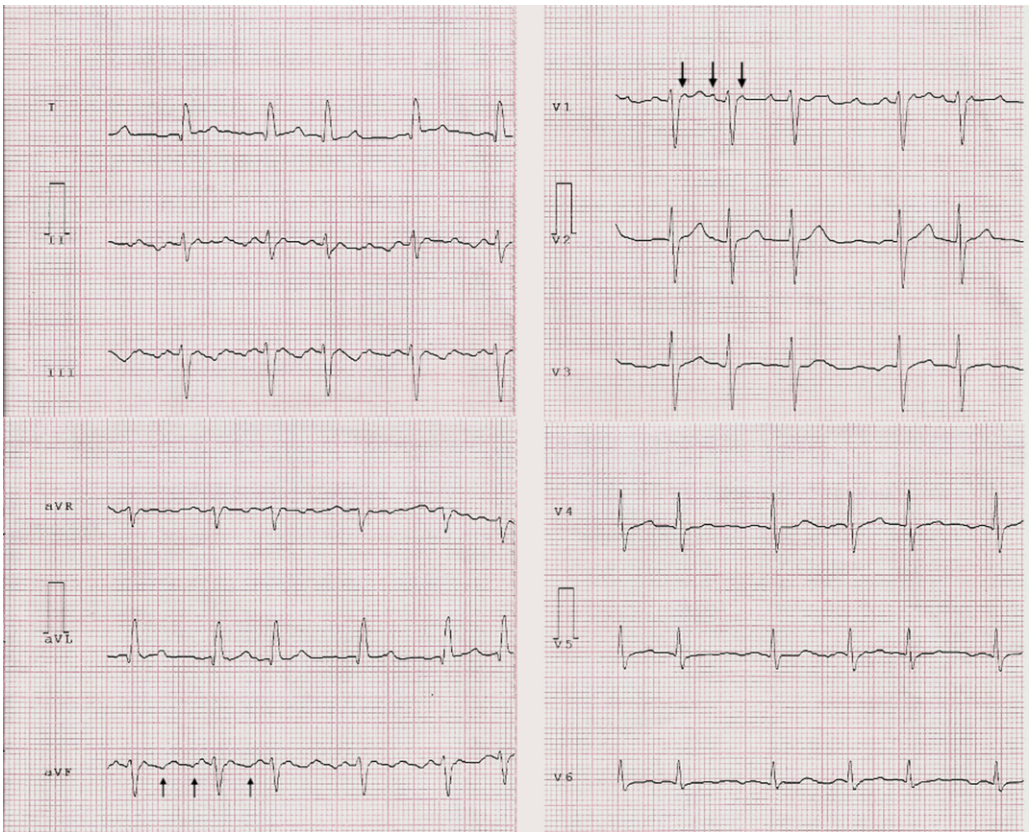

Fig. 3. Typical atrial flutter with variable atrioventricular conduction: an irregular tachycardia with F waves visible on the inferior leads and on the V1 lead with variable atrioventricular conduction.

lead V1 because the atria are activated in a caudocranial direction. The retrograde P wave is usually placed at once after the QRS complex, sometimes it is concealed by that and rarely it may be situated before the QRS complex [18].

There are two types of junctional tachycardia:

- *Automatic junctional tachycardia*, generally due to abnormal automaticity of the junctional focus. Rates of tachycardia are often between 110 and 250 bpm. AV dissociation is often present, although one-to-one retrograde conduction may be transiently observed. This arrhythmia usually presents in young adulthood.

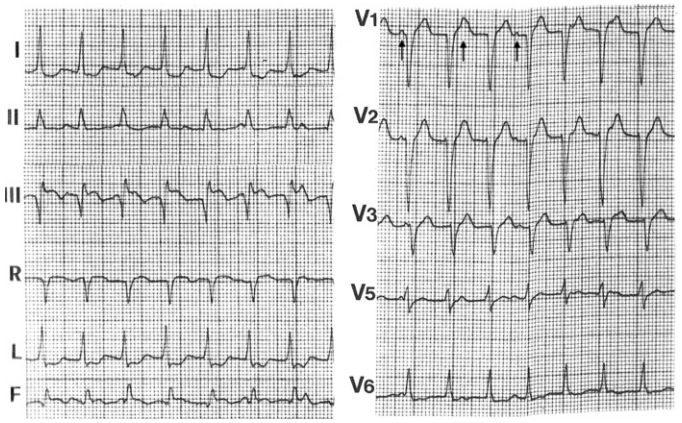

Fig. 4. Junctional tachycardia: a regular tachycardia with ventricular rate of 170 bpm. On the V1 lead, P waves with atrioventricular dissociation are visible. Modified from: Oreto [18].

It is generally exercise- or stress-related and may be found in patients with structurally normal hearts or in patients with congenital abnormalities such as atrial or ventricular septal defects. The patients are often quite symptomatic and, if untreated, may develop heart failure, particularly if their tachycardia is incessant [10].

- *Nonparoxysmal junctional tachycardia*, a benign arrhythmia with rates between 70 and 120 bpm. This arrhythmia shows usually a typical "warm-up" and "cool-down" pattern. Unlike the more rapid form of automatic junctional tachycardia, there is commonly one-to-one retrograde conduction AV. The arrhythmia may be a marker for a serious underlying condition such as digitalis toxicity, hypokalemia, postcardiac surgery, myocardial ischemia, inflammatory myocarditis, and chronic obstructive lung disease with hypoxia. In some cases, particularly in the setting of digitalis toxicity, anterograde AV-nodal Wenckebach conduction block may be observed [9,10].

(3) The *atrioventricular nodal reentrant tachycardia* (AVNRT) is the most common form of paroxysmal supraventricular tachycardia [9,10,21,22]. It is more prevalent in females [10,22,23], is not usually associated with structural heart disease [10] and, although it may appear at any age, most patients first seek medical attention during the fourth or fifth decade of life [9]. Rates of tachycardia are often between 140 and 250 bpm.

In patients with AVNRT at least two functionally distinct conduction pathways are demonstrable within the AV node during electrophysiologic study. One pathway, referred to as the fast pathway, is characterized by rapid conduction velocity and a relatively long refractory period. The second pathway, the so called slow pathway, typically has slow conduction velocity and a short refractory

period. On the ECG, these tachycardias are usually characterized by constant cardiac cycles and by an AV ratio of 1:1 [18].

Common AVNRT (slow–fast) (Fig. 5) is due to a reentrant circuit in which there is anterograde conduction over the slow AV nodal pathway and retrograde conduction over the fast pathway. It starts frequently with a supraventricular ectopic beat that, on finding the fast pathway in refractory period, travels in the slow pathway as to appear as a prolongation of the PR interval on the ECG. During tachycardia, the P waves are negative in surface electrocardiographic leads II, III, and aVF and positive in lead V1 because the atria are activated in a caudocranial direction, but they are rarely visible because they are superimposed on the QRS complex because of the simultaneous or nearly simultaneous activation of the atria and the ventricles. Generally, the tachycardia stops with a supraventricular ectopic beat, spontaneously, with vagal maneuvers (carotid-sinus massage, Valsalva maneuver) or with intravenous administration of AV nodal blocking drugs [18]. The reentry circuit stops usually within the slow pathway, so that the ECG shows a P wave unconcealed by a QRS complex [18].

In 10% of patients the reentry circuit is reversed, with anterograde conduction over the fast pathway and retrograde conduction over the slow pathway [9,22]. This tachycardia, known as uncommon AVNRT (fast-slow), is characterized by negative P waves on the inferior leads but positive on the V1 lead and it presents a RP interval longer than PR interval. Unlike the common, the uncommon AVNRT is usually initiated by a ventricular premature depolarization and is rarely sustained [9,24]. The reentry circuit stops usually within the slow pathway: consequently, the ECG shows a QRS complex without a following visible P wave [18].

Infrequently, both limbs of the tachycardia circuit are composed of slowly conducting tissue (slow–slow) and the P wave is inscribed after the QRS [10].

(4) The *atrioventricular reciprocating tachycardia* (AVRT) is the most common cause of supraventricular tachycardia after AV nodal reentry [21]. This type of arrhythmia involves the activity of extra nodal accessory pathways that are anomalous bands of conducting tissue that form a connection between the atrium and ventricle in addition to the normal AV conducting system. Accessory pathways can be classified on the basis of their location, type of conduction (decremental or nondecremental), and in relation to their ability of anterograde conduction, retrograde conduction, or both. When we have an anterograde accessory-pathway conduction during sinus rhythm, ventricular preexcitation occurs, because of early activation of a part of the ventricles over the accessory pathway. The diagnosis of Wolff–Parkinson–White (WPW) syndrome is reserved for patients who have both pre-excitation and tachyarrhythmias [9,10]. The ECG findings, in an AVRT, typically present AV conduction 1:1 and usually show a NCT at rates of 160–240 bpm [25].

AVRT is subclassified into orthodromic and antidromic. During orthodromic AVRT (Fig. 6), the reentrant impulse conducts over the AV node and the specialized conduction system from the atrium to the ventricle and utilizes the accessory pathway for conduction from the ventricle to the atrium; consequently delta waves are absent. This tachycardia is triggered by an atrial or ventricular ectopic beat and finishes spontaneously or with an atrial or ventricular ectopic beat too [26,27].

A variation of the orthodromic AVRT is the "permanent form of reciprocating junctional tachycardia", also known as "reciprocating tachycardia of Coumel" [28]. The circuit of this tachycardia includes an accessory pathway at low velocity of conduction (rate-dependent conduction) [29]. This arrhythmia presents a nonparoxysmal but incessant trend and starts whenever the anterograde conduction within the accessory pathway is stopped because of a slightly shorter cardiac cycle [18]. In the typical form, the localization of accessory pathway is posteroseptal and occurs consequently as negative P wave on the inferior leads but other localizations of accessory pathways are possible [10,30,31].

During antidromic AVRT, the reentrant impulse travels in the reverse direction, with anterograde conduction from the atrium to the ventricle occurring via the accessory pathway and retrograde

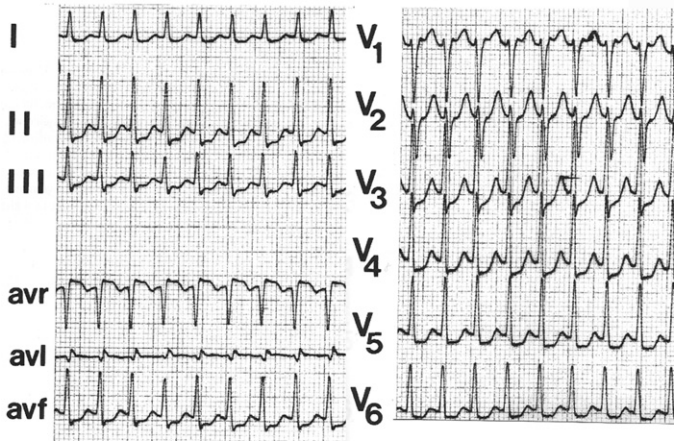

Fig. 5. Common atrioventricular nodal reentrant tachycardia (AVNRT): a regular tachycardia with ventricular rate of 230 bpm; the P waves are not visible but a pseudo *r'* wave is present on the V1 lead and a pseudo S wave on the inferior leads. Modified from: Oreto [18].

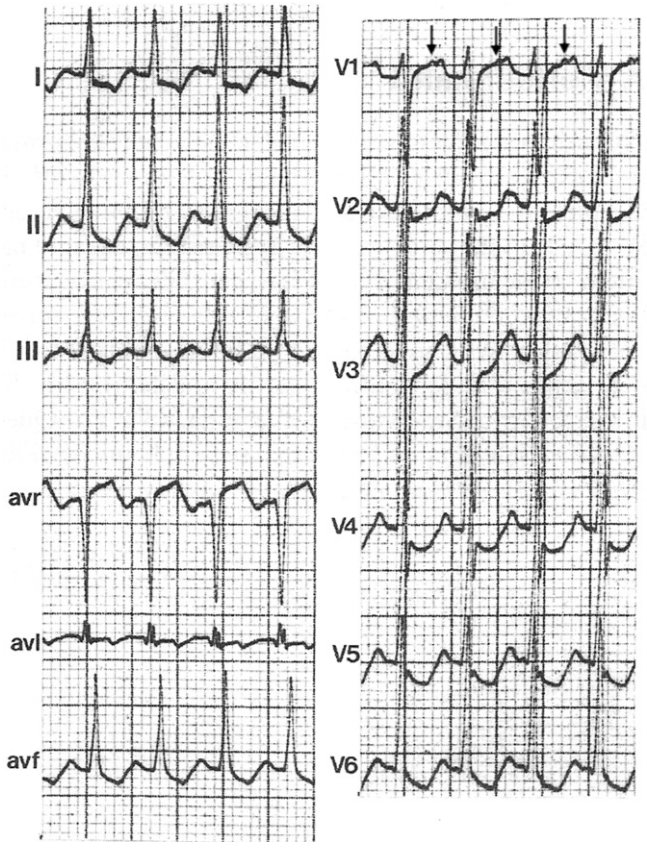

Fig. 6. Orthodromic atrioventricular reciprocating tachycardia (AVRT): a regular tachycardia with ventricular rate of 205 bpm; it is visible on V1 lead, a P wave inscribed in the T wave with RP > 70 ms and RP < PR. Modified from: Oreto [18].

conduction over the AV node or a second accessory pathway. This type of tachycardia is triggered by an atrial or ventricular beat and is characterized by a wide QRS configuration that is an exaggeration of the delta wave seen during sinus rhythm. Antidromic AVRT occurs in only 5–10% of patients with WPW syndrome and is more common among patients with multiple accessory pathways [9,10].

AF could be frequently found in patients with the WPW syndrome [32]; it has been estimated that one-third of patients with WPW syndrome also have AF [10]. AF and atrial flutter are particularly hazardous in patients with the WPW syndrome because most accessory pathways have rapid nondecremental conduction. These patients may achieve ventricular rates that approach or exceed 300 bpm during AF or atrial flutter; ventricular fibrillation (VF) and sudden death can occur under such circumstances [9].

The markers that usually are reported to identify patients at increased risk of sudden death include: (a) an anterograde effective refractory period of the accessory pathway below 250 ms (probably the most important factor) [33,34]; (b) a history of symptomatic tachycardia [10]; (c) multiple accessory pathways [33]; and (d) Ebstein's anomaly [10]. Conversely, the noninvasive markers that recognize a long refractory period of the accessory pathway and therefore identify patients at reduced risk of VF and sudden death are: (a) the evidence of intermittent preexcitation on the resting ECG [35]; (b) the sudden disappearance of the delta wave after the intravenous injection of a high dose of procainamide [36] or ajmaline [37]; and (c) the sudden disappearance of the delta wave at a relatively slow heart rate during exercise [38]. However, noninvasive tests are considered inferior to invasive electrophysiological assessment for risk of sudden cardiac death [10].

The narrow complex tachycardias: diagnostic algorithm

Since the differential diagnosis of NCTs is sometimes difficult because of similar presentation on the ECG, we propose a quick and detailed diagnostic algorithm (Fig. 7) to discern between all the possible types of existing NCTs. The algorithm does not include purposely some supraventricular tachycardias such as atrial flutter 1:1 because it is extremely rare and usually it seems to involve aberrant conduction for the high ventricular rate, the antidromic AVRT because it is characterized by a wide QRS configuration that is an exaggeration of the delta wave seen during sinus rhythm, and all wide QRS complex supraventricular tachycardias due to bundle

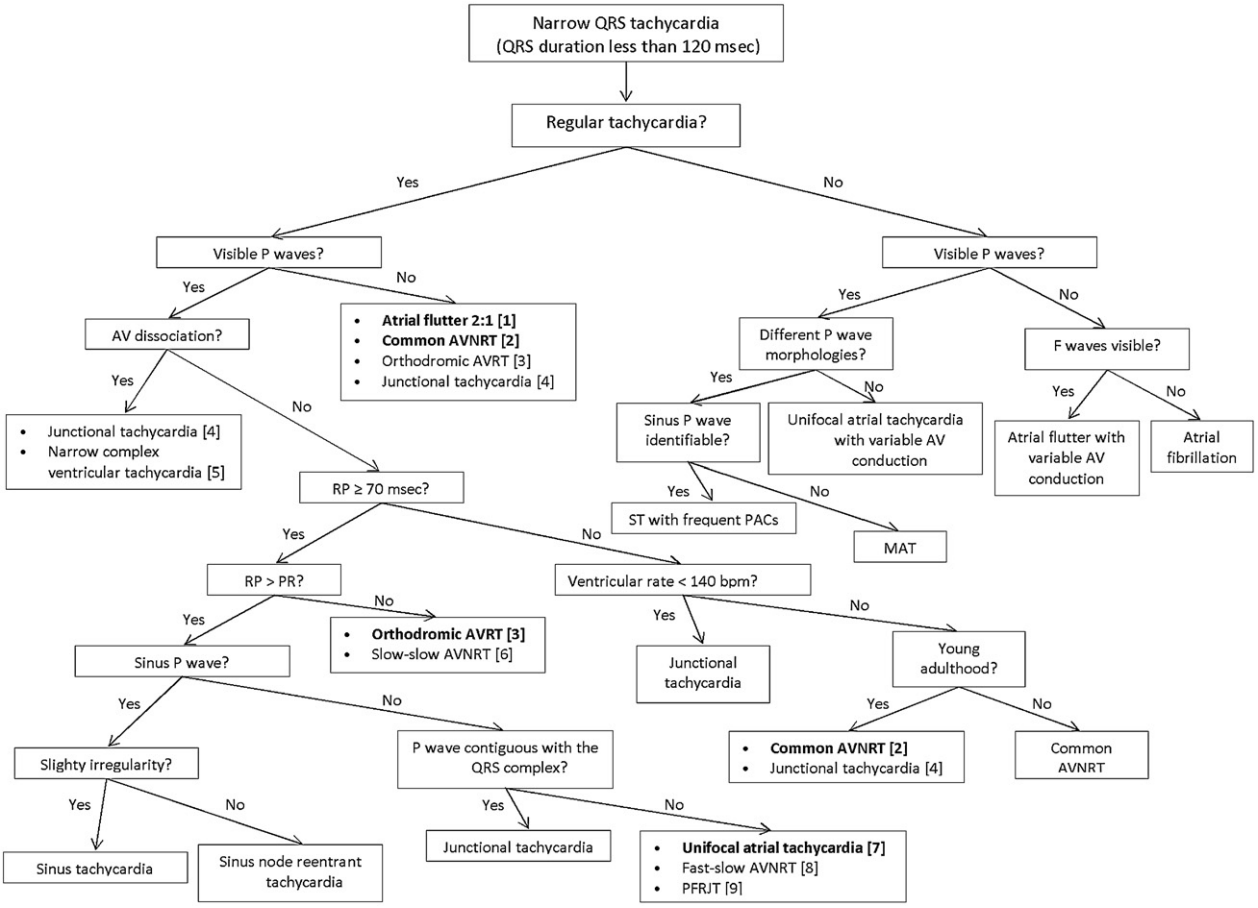

Fig. 7. Diagnostic algorithm. The figure shows a flow chart for a quick diagnostic reading of an electrocardiographic finding of narrow QRS tachycardia. Whenever the algorithm leads to two or more conclusions, the most frequent diagnostic option is shown in bold. Footnotes suggest additional diagnostic criteria that need to be considered to further confirm the suspected diagnosis. ST, sinus tachycardia; PACs, premature atrial contractions; AV, atrioventricular; AVNRT, atrioventricular nodal reentrant tachycardia; AVRT, atrioventricular reciprocating tachycardia; MAT, multifocal atrial tachycardia; PFRJT, permanent form of junctional reciprocating tachycardia; msec, milliseconds; bpm, beats per minute. Notes: Unifocal atrial tachycardia, compared with the atrial flutter, presents: atrial rate less than 250 bpm, the isoelectric line between the P waves and a greater variability of atrial cycles. *Notes*: (1) Ventricular rate between 125 and 175 bpm, traces of F waves on the inferior leads or on V1 lead. (2) Ventricular rate between 140 and 250 bpm, prominent jugular venous pulsations probable, pseudo *r'* in lead V1 probable, pseudo S in inferior leads possible, QRS alternans possible, ST depression ≥2 mm or TWI or both in ≥1 lead possible. (3) P wave usually visible (rarely concealed by T wave), QRS alternans probable, ST depression ≥2 mm or TWI or both in ≥1 lead probable, pre-excitation during sinus rhythm highly specific. (4) P waves negative in inferior leads and positive in lead V1 (whenever they are visible), usually in young adulthood. (5) Fusion and/or capture beats, concordant precordial pattern of the QRS complexes, QRS configuration different by the sinus rhythm. (6) P waves negative in inferior leads and positive in lead V1. (7) Atrial rate <250 bpm, any P wave axis, AV conduction variable. (8) Usually nonsustained, P waves negative in inferior leads and positive in lead V1. (9) Nonparoxysmal but incessant trend, usually P waves negative in inferior leads.

branch block pre-existing or to aberrant conduction tachycardia-dependence.

On the contrary, the algorithm includes the narrow complex ventricular tachycardia, an uncommon tachycardia that happens whenever the arrhythmia originates near the intraventricular conduction system; consequently, the ventricular activation ends in a relatively short time [1,39].

The algorithm provides a step by step approach and requires a single binary "yes" or "no" decision at each point; it does not always reach unambiguous conclusions because it is not always possible to diagnose exactly a NCT by a 12-lead ECG [18]. Whenever the algorithm leads to two or more conclusions, we put in bold type the most frequent diagnostic option and we suggest some additional criteria for obtaining the correct diagnosis with some notes.

The initial evaluation of a NCT provides the analysis of the regularity of the RR intervals and of the presence or absence of P waves. An irregular tachycardia without P waves is an AF but if F waves are visible and RR intervals are not all different then it will be an atrial flutter with variable atrioventricular conduction. An irregular tachycardia with the sinus P wave identifiable is a sinus tachycardia with frequent premature atrial contractions, if there are three or more distinct P wave morphologies it will be a MAT but if there is a single P wave morphology with variable RR intervals then it will be a unifocal atrial tachycardia with variable AV conduction.

The differential diagnosis among the unifocal atrial tachycardia and the atrial flutter is not always simple because sometimes it is difficult to distinguish the P waves of atrial tachycardia by the F waves of atrial flutter; in that case, an atrial rate less than 250 bpm, the presence of isoelectric line between the P waves and the greater variability of the atrial cycles suggest the diagnosis of unifocal atrial tachycardia.

A regular tachycardia without visible P waves is usually an atrial flutter 2:1 (Fig. 8) or a common AVNRT; alternative diagnosis may be the orthodromic AVRT or the junctional tachycardia, but the first diagnosis usually has visible P waves which rarely are concealed by the T waves and the second diagnosis is an uncommon tachycardia affecting usually young adults. Instead, the differential diagnosis between atrial flutter 2:1 and common AVNRT is not always simple. In the atrial flutter 2:1, indeed, the F waves are often concealed by QRS complex and T wave and in the common AVNRT the P waves are rarely visible because they are superimposed on the QRS complex because of the simultaneous or nearly simultaneous activation of the atria and the ventricles. Nevertheless, in the atrial flutter 2:1, a careful analysis of the ECG could show, on the inferior leads or on the V1 lead, some traces of F waves so that the diagnosis becomes possible. Moreover, in order to distinguish between atrial flutter 2:1 and common AVNRT, it is possible to utilize the vagal maneuvers; in the atrial flutter 2:1, those may cause a temporary block of some ventricular beats, so that the F waves become visible and the diagnosis is possible; in presence of common AVNRT, on the contrary, the vagal maneuvers may stop the tachycardia or result inefficient [18].

If an AV dissociation is observable, the arrhythmia may be a junctional tachycardia or a narrow complex ventricular tachycardia; a different QRS configuration and the presence of the other diagnostic criteria of ventricular tachycardia (fusion and/or capture beats, concordant precordial pattern, QRS axis, etc.) are helpful for the differential diagnosis [39–41].

In presence of sinus P wave, the sinus tachycardia presents a slight irregularity compared with the sinus node re-entrant tachycardia which, on the contrary, is extremely regular and usually paroxysmal and nonsustained [9,10]. In order to discern between the other tachycardias, in addition to the P wave morphologies, the RP and PR intervals are generally helpful.

Whenever it is not possible to reach a sure diagnosis using the algorithm, several additional clinical and ECG criteria may be considered.

The physical examination is rarely helpful, except in AVNRT, in which simultaneous activation of the atria and ventricles against closed AV valves causes prominent jugular venous pulsations [9]. The age is helpful in the junctional tachycardia that presents usually in young adults [10]. In the junctional tachycardia and in the

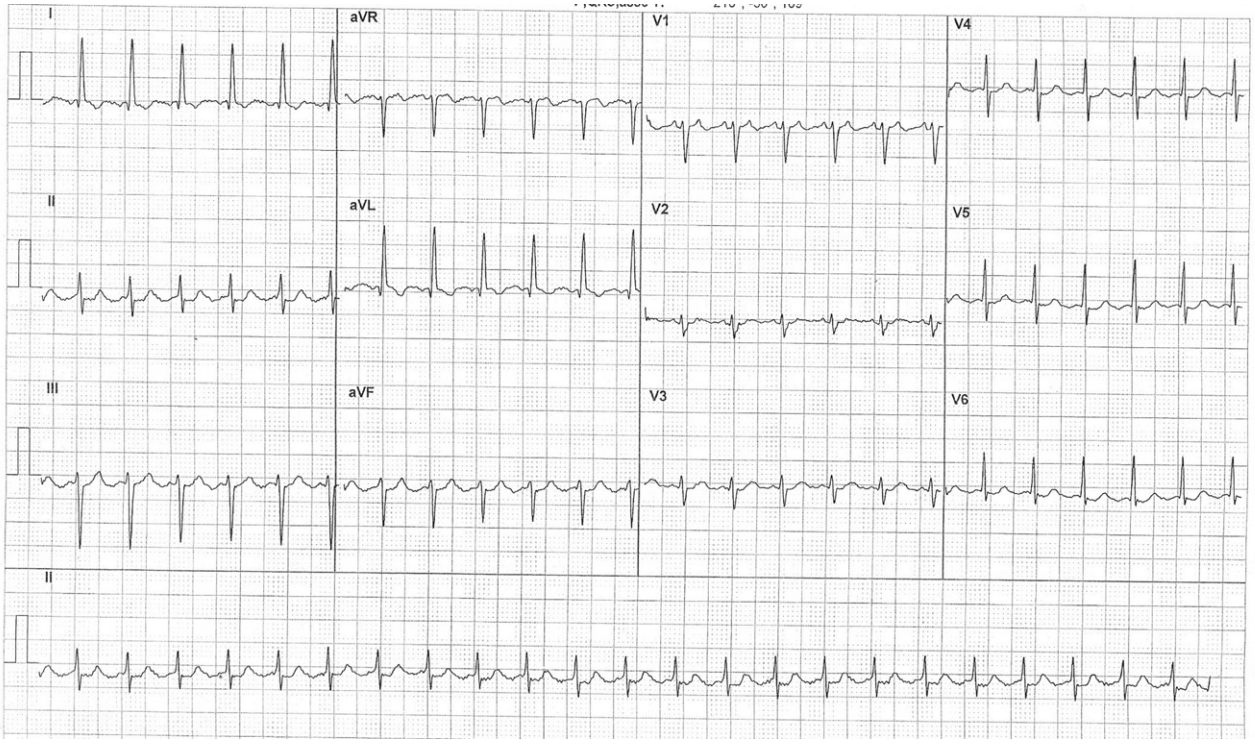

Fig. 8. Atypical atrial flutter 2:1: a regular tachycardia with ventricular rate of 140 bpm and F waves visible on the V1 lead with 2:1 atrioventricular block.

AVNRT, the P waves may be concealed by the QRS complex but, whenever they are visible, the P waves are negative in surface electrocardiographic leads II, III, and aVF and positive in lead V1 because the atria are activated in caudocranial direction; moreover, in the junctional tachycardia, the P waves may be rarely situated before the QRS complex if retrograde atrial activation is more quick than anterograde ventricular activation. The P wave axis in the AVRT depends on the accessory pathway localization and therefore there may be various P wave morphologies. The unifocal atrial tachycardia is characterized by a single P wave morphologic pattern, the configuration of which depends entirely on the atrial site from which the tachycardia originates; therefore, the P waves may be upright, biphasic or inverted; moreover, the unifocal atrial tachycardia may present variable atrioventricular conduction. Whenever the diagnosis of AVRT or of AVNRT is ambiguous, some additional criteria may be considered. The QRS alternans, defined as a beat to beat oscillation in QRS amplitude of $\geq$1 mm in at least one lead, is present significantly more often in AVRT than in AVNRT and it is present more often at the higher ventricular rates [42,43]. The presence of horizontal or downsloping ST segment depression of > 2 mm persisting 80 ms after the J point or T wave inversion, or both, in one or more leads, suggests that AVRT, or less likely AVNRT, is the mechanism of the tachycardia; nevertheless, the phenomenon may be the consequence of a distinct pattern of retrograde atrial activation [44]. If a baseline ECG is available, the presence of manifest preexcitation during sinus rhythm is a highly specific criterion of AVRT; alike, the finding of a pseudo r' wave in lead V1 and of a pseudo S wave in inferior leads is a criterion which has high specificity and high positive predictive value for common AVNRT (pseudo r' has higher sensitivity than pseudo S) [10,42,43]. The fast–slow and the slow–slow AVNRT are two rare arrhythmias; the first is usually nonsustained and presents an RP interval longer than PR interval, on the contrary the second presents an RP interval shorter than PR interval [9,10,24]. Finally, the "permanent form of reciprocating junctional tachycardia" is a syndrome characterized by a nonparoxysmal but incessant supraventricular tachycardia; usually, the localization of accessory pathway is posteroseptal and P waves are consequently negative in inferior leads [10].

Conclusions

The NCTs are a spectrum of tachycardias, typically of supraventricular origin, which, although have different physiopathological mechanisms, sometimes those may have a similar presentation on the ECG so that a correct diagnosis may be arduous.

We propose a quick and accurate algorithm to improve the diagnostic accuracy of the NCTs on the basis of the various ECG criteria and, whenever those are not sufficient, of some additional clinical criteria.

Conflict of interest

No relationships to declare.

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