Chapter

Electrocardiographic Differential Diagnosis of Narrow QRS and Wide QRS Complex Tachycardias

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Abstract

Narrow QRS complex tachycardias or Wide QRS complex tachycardias are common problems encountered in clinical practices. Although such tachycardias often occur in patients with a normal anatomy and/or function of heart and rarely represent life-threatening conditions, they are common sources of morbidity and/or mortality. Narrow QRS complex tachycardias are fast cardiac rhythms with QRS duration of 120 ms or less while wide QRS complex tachycardias are fast cardiac rhythms with QRS duration of 120 ms or more. Origins of narrow QRS complex tachycardias are above or within the His bundle. Wide QRS complex tachycardias can be ventricular tachycardias, supra-ventricular tachycardias with bundle branch block or accessory pathway. The purpose of this chapter is to present the differential diagnosis of narrow and wide QRS complex tachycardias.

Keywords: narrow QRS tachycardia, wide QRS tachycardia, tachycardia, electrocardiograms

1. Introduction

Differential diagnosis and treatment of tachycardias is a common dilemma encountered by physicians or cardiologists. Although such tachycardias often occur in patients with a normal heart, they may cause bothersome symptoms and rarely represent life-threatening conditions. Among these tachycardias with a heart rate greater than 100 beats per minute (bpm), the narrow QRS complex tachycardias (NCTs) are defined by the presence in a 12-lead electrocardiogram (ECG) of a QRS complex duration less than 120 ms and the wide QRS complex tachycardias (WCTs) are defined by the presence in a 12-lead ECG of a QRS complex duration more than 120 ms (**Figure 1**) [1–10]. The NCTs are typically of supraventricular origin above or within the His bundle, although rarely narrow complex ventricular tachycardias (VT) have been reported in the literature in which early activation of the His bundle can also occur in high septal VT, resulting in relatively narrow QRS complexes of 110–140 ms (**Table 1**, [1–5]]. The WCTs can be VT or supraventricular tachycardia (SVT) with right or left bundle branch block (BBB) or right or left accessory pathway (**Table 1**, [6–10]]. Because administration of medications based on misdiagnosis of these tachycardias can be harmful and sometimes fatal, diagnosis

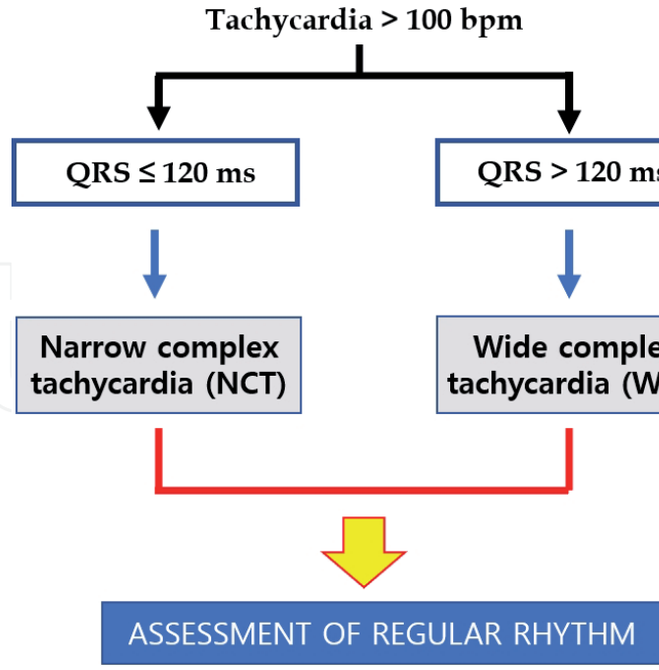

Figure 1.
Differential diagnostic algorithm of NCTs and WCTs.

The NCTs with QRS duration less than 120 ms	The WCTs with QRS duration more than 120 ms
Regular rhythm	*Regular rhythm*
• Sinus tcahycardia	• VT/Ventricular flutter
• Sinus node reentrant tachycardia	• Antidromic AVRT
• Atrial tachycardia	• SVT with aberrant conduction / BBB
• Atrial flutter	• Atrial tachycardia with accessory pathway
• Junctional tachycardia	• Junctional tachycardia with accessory pathway
• Paroxysmal SVT (AVNRT / Orthodromic AVRT)	
• Permanent junctional reciprocating tachycardia (PJRT)	
Irregular rhythm	*Irregular rhythm*
• Atrial tachycardia with variable AV conduction	• Polymorphic VT / Ventricular fibrillation
• Multifocal atrial tachycardia (MAT)	• Antidromic AVRT with variable VA conduction
• Atrial flutter with variable AV conduction	• Pre-excited AF
• Atrial fibrillation	• Torsades de pointes
	• AF or atrial flutter or focal atrial tachcyardia with varying block conducted with aberration

SVT; supra-ventricular tachycardia, AVNRT; atrio-ventricular nodal re-entrant tachycardia, AVRT; atrio-ventricular reciprocating tachycardia, AV; atrio-ventricular, VT; ventricular tachycardia, BBB; bundle branch block, AF; atrial fibrillation, VA; ventriculo-atrial.

Table 1.
Differential diagnosis of NCTs and WCTs.

of these tachycardias is critical [11–13]. The accurate, rapid diagnosis in patients with these tachycardias still remains a significant clinical dilemma, because the published numerous ECG algorithms and criteria are complicated and difficult to recall in urgent clinical situations [11–13]. We have reviewed ECG findings of the NCTs and WCTs in order to reduce the possible diagnostic errors on the ECGs.

2. NCTs

The NCTs are common problems encountered in clinical situations [1–5, 14–21]. The key to approaching the diagnosis of these arrhythmias is identifying atrial activity (P waves) on the ECG and classifying these tachycardias according to the presence of AV dissociation (**Figure 2**) and then re-classifying according to long RP or short RP (**Table 2**) [1–5, 14–21]. On the basis of these algorithm, a differential diagnosis can

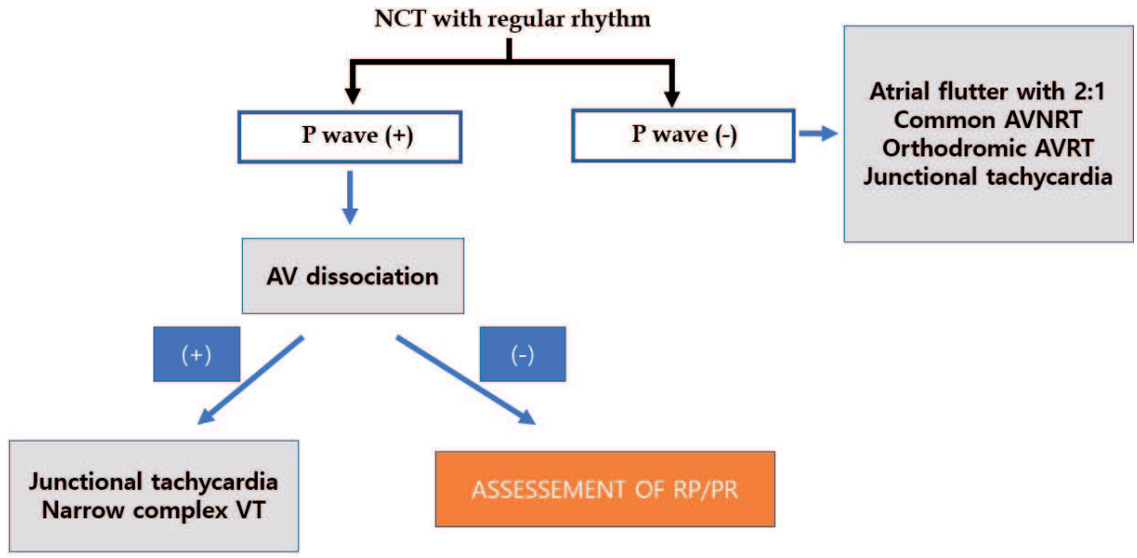

Figure 2.
Differential diagnostic algorithm of NCTs with regular rhythm.

Short RP (RP < PR)	Long RP (RP > PR)
• AVNRT	• Sinus tachycardia
• AVRT	• Sinus nodal reentrant tachycardia
• Junctional tachycardia	• Atrial tachycardia
	• Junctional tachycardia
	• PJRT
	• AVNRT (unusual type, fast-slow)
	• AVRT (atypical type)

AVNRT; atrio-ventricular nodal re-entrant tachycardia, AVRT; atrio-ventricular reciprocating tachycardia, PJRT; Permanent junctional reciprocating tachycardia.

Table 2.
Differential diagnosis of NCTs according to RP interval.

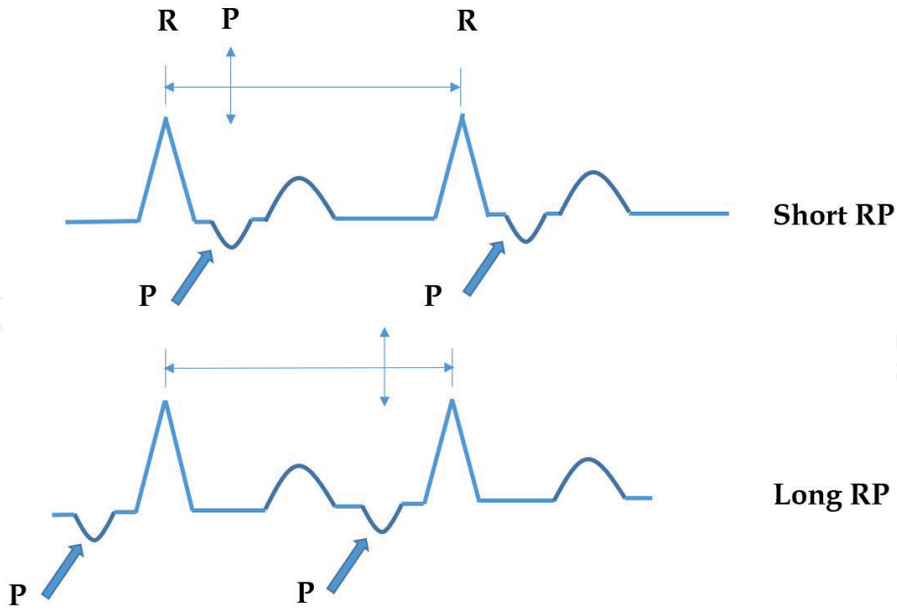

Figure 3.
Schematic demonstration of short RP and long RP.

be generated, logical therapy can be delivered for termination of the tachycardia, and a plan can be developed to prevent recurrence.

Short RP tachycardias are defined as regular tachycardias in which interval from QRS complex to P wave (upper arrows, **Figure 3**) much less than interval from P wave to subsequent QRS complex, whereas long RP tachycardias are defined as regular tachycardias in which interval from QRS complex to P wave much more than interval from P wave to subsequent QRS complex (lower arrows, **Figure 3**) [1–5, 14–21].

2.1 NCTs with regular rhythm

2.1.1 Sinus tachycardia

Sinus tachycardia is defined as an increase in sinus rate to more than 100 bpm with regular rhythm. The rate increases gradually and may show beat to beat variation. Although generally identifiable by a P wave of normal morphologic features that precedes each QRS complex, sinus tachycardia can be difficult to recognize when the P wave begins to fuse with the T wave of the preceding QRS complex. Sinus tachycardia is usually a physiological response such as fever, anxiety, pain, hyperthyroidism but may be precipitated by sympathomimetic drugs or endocrine disturbances [5, 14–21].

2.1.2 Sinus nodal reentrant tachycardia

The morphologic appearance of sinus nodal reentrant tachycardia is identical to that of sinus tachycardia. In contrast to sinus tachycardia, the rate is very regular and initiation and termination are abrupt without an underlying physiological stimulus. Vagal maneuver may be successful in stopping the arrhythmia [5, 14–21].

2.1.3 Atrial tachycardia

Atrial tachycardia (AT) is usually a NCTs accounting for 5–15% of SVT. Other than sinus tachycardia, AT is the most common long RP tachycardia. In AT, an atrial source

outside the sinoatrial node due to focal automatic activity or re-entry circuit activates the atria. Accordingly, P-wave morphologic characteristics vary depending on the site of this source. Digitalis toxicity should be suspected in patients with paroxysmal AT with AV block [5, 14–22].

2.1.4 Atrial flutter

Atrial flutter is a reentrant rhythm of the right atrium typically with an atrial rate of 250 to 350 beats/min. The flutter may circulate in a counterclockwise direction around the tricuspid annulus in the frontal plane (typical, counterclockwise flutter) or in a clockwise direction (atypical, clockwise flutter). P waves have a characteristic "sawtooth" appearance, and 2:1 AV block is common. Because one flutter wave occurs in the ST-T segment and another flutter wave occurs before each QRS complex in atrial flutter with 2:1 AV conduction, atrial flutter is neither a short RP nor a long RP tachycardia [5, 14–22].

2.1.5 Junctional tachycardia

Non-paroxysmal junctional tachycardia (NPJT) is a tachycardia that arises in the AV junction. Although often described as a short RP tachycardia, because NPJT causes ventricular activation almost concurrently with atrial activation, a substantial portion (25%), which is described as a long RP tachycardia, actually show P waves that slightly precede the QRS complex. and in some cases, AV dissociation may be present. Unlike AVNRT and AVRT, initiation and termination are gradual. NPJT is often associated with digitalis intoxication, inferior myocardial infarction, myocarditis, and mitral valve surgical procedures [5, 14–23].

2.1.6 Paroxysmal SVT (AVNRT/Orthodromic AVRT)

2.1.6.1 AVNRT

AV nodal reentrant tachycardia (AVNRT) is characterized by a tachycardia with supraventricular origin, with sudden onset and termination generally at rates between 150 and 250 beats/min and is the most common cause of SVT except atrial fibrillation, atrial flutter, and sinus tachycardia. In majority of patients (noted as the "typical" or "slow-fast" AVNRT), anterograde conduction to the ventricle occurs over the "slow" pathway and retrograde conduction to the atrium occurs over the "fast" pathway and the atria are activated either simultaneously with or just after activations of the ventricles and this common type is classified as a short RP tachycardia. Rarely, in "atypical" or "fast-slow" AVNRT, the reentry occurs in the opposite direction in which anterograde conduction occurs over the "fast" pathway, while retrograde conduction occurs over "slow" pathway, and this rare type is classified as a long RP tachycardia [24–31].

2.1.6.2 AVRT

AV reentrant tachycardia (AVRT) involves reentry between the atria and ventricles with use of the AV node-His bundle conduction as the anterograde and slow pathway and an accessory conduction as the retrograde and fast pathway. This pattern is also known as orthodromic reciprocating tachycardia (ORT). This type is not apparent by analysis of the ECG during sinus rhythm because the ventricle is not pre-excited and

the accessory pathway is said to be "concealed". In tachycardia, retrograde conduction over the accessory pathway is fast and yields a short RP tachycardia [24–31].

In contradistinction to ORT resulting in NCTs, antidromic AVRT has anterograde conduction over the accessory pathway and retrograde conduction over the AV node-His bundle resulting in WCTs [24–31].

The following factors are important differences between AVNRT and AVRT [24–31]:

In contradistinction to AVNRT, an 1:1 relationship is necessary for AVRT because both the atria and the ventricles are part of the reentry circuit. Therefore, if AV block occurs during tachycardia, AVRT is excluded.

If bundle branch block occurs during ORT and the length of the tachycardia cycle increases, AVNRT is excluded because the His-Purkinje system is not part of the tachycardia reentry circuit in AVNRT. The converse is not necessarily true because the absence of cycle length change with the occurrence of bundle branch block does not exclude AVRT.

2.1.7 Permanent junctional reciprocating tachycardia (PJRT)

As discussed with AVRT, certain types of reentrant circuits exist in which the accessory AV connection has AV nodal properties such as slow conduction. In PJRT, excitation over the postero-septal accessory pathway conducts very slowly, because of a long and tortuous route of pathway. Tachycardia is maintained by anterograde AV nodal conduction and retrograde conduction over slow accessory pathway. Because of slow conduction property of accessory pathway, retrograde atrial activation is delayed, and a long RP tachycardia results. Patients with this type of accessory pathway almost never have preexcitation (a delta wave) on ECGs during sinus rhythm [5, 14–23].

2.2 NCTs with irregular rhythm

2.2.1 Atrial tachycardia with variable AV conduction

Atrial tachycardia with atrioventricular block is typically seen with digoxin toxicity. The ventricular rhythm is usually regular but may be irregular if atrioventricular block is variable [5, 14–21].

2.2.2 Multifocal atrial tachycardia (MAT)

MAT is characterized by P waves with variable morphologies and variable PR intervals. Differential diagnosis between MAT and atrial fibrillation can be possible by the presence of isoelectric baselines between the P waves in MAT. MAT is seen typically in patients with chronic obstructive pulmonary disease or digoxin toxicity [5, 14–21, 32].

2.2.3 Atrial flutter with variable AV conduction

Atrial flutter is due to a re-entry circuit in the right atrium with secondary activation of the left atrium. This produces atrial contractions at a rate of about 300 beats/min as flutter (F) waves. F waves show broad and saw-tooth appearances and are best seen in lead V1 and the inferior leads [5, 14–21].

2.2.4 Atrial fibrillation

This is the most common sustained arrhythmia with overall prevalence is 1% to 1.5%. Atrial fibrillation is caused by multiple re-entrant circuits or "wavelets" of activation sweeping around the atrial myocardium without effective atrial contraction. Atrial fibrillation is seen on the ECG as irregular baseline undulations of variable amplitude and morphology (called f waves) discharging at a frequency of 350 to 600 beats/min.

With normal conduction, ventricular rate shows frequency between 100 and 150 beats/min. Atrial fibrillation with slow ventricular responses or AV block is seen typically in patients with digoxin toxicity [5, 14–21, 33–36].

3. WCTs

3.1 WCTs with regular rhythm

3.1.1 VT/ventricular flutter

Monomorphic ventricular tachycardia is common in patients with a history of previous myocardial infarction. Other rare causes of monomorphic VT include right or left ventricular outflow tract ventricular tachycardia and right ventricular dysplasia.

Ventricular flutter appears as a sine wave pattern with regular, large oscillations on the ECG and can progress to ventricular fibrillation [37–44].

3.1.2 Antidromic AVRT

Antidromic AVRT includes a reentrant circuit with accessory pathway as the anterograde pathway, and AV node–His bundle as the retrograde pathway. Some patients (3 to 8%) with WPW syndrome show mechanisms of antidromic AVRT [24–31].

- SVT with aberrant conduction/BBB
- Atrial tachycardia with accessory pathway
- Junctional tachycardia with accessory pathway

3.2 WCTs with irregular rhythm

3.2.1 Polymorphic VT/ventricular fibrillation

Polymorphic VT is most commonly caused by abnormalities of ventricular muscle repolarization. The predisposition to this problem usually manifests on the ECG as a prolongation of the QT interval. Congenital problems include long QT syndrome and catecholaminergic polymorphic ventricular tachycardia. Acquired problems are usually related to drug toxicity or electrolyte abnormalities, myocardial ischemia. Class III anti-arrhythmic drugs such as sotalol and amiodarone prolong the QT interval and may in some circumstances be pro-arrhythmic. Other relatively common drugs include some antibiotics and antihistamines [37–44].

Ventricular fibrillation is a terminal arrhythmia in which ventricular contractions are uncoordinated and too weak to eject blood. The ECG shows irregular, chaotic deflections of varying amplitude and shape [37–44].

- Antidromic AVRT with variable VA conduction
- Pre-excited AF (AF with ventricular pre-excitation)
- Torsades de pointes

The ECG demonstrates a polymorphic VT characterized by the QRS complexes of changing amplitude that appear to twist around the isoelectric line and occur at the rates of 200 to 250 beats/min. Most data suggest that early afterdepolarizations are responsible for both the QT prolongation and the torsades de pointes. The most common causes are congenital severe bradycardia, potassium depletion and use of class IA and IC drugs. Clinical features depend on whether torsades de pointes is due to acquired or congenital long QT syndrome. Some episodes may persist and progress to ventricular fibrillation, leading to sudden death. In congenital long QT syndrome, long QT intervals predispose the patient to an R-on-T phenomenon, wherein the R-wave, representing ventricular depolarization, occurs during the relative refractory period at the end of repolarization [37–44].

- AF or atrial flutter or focal atrial tachcyardia with varying block conducted with aberration

4. ECG criteria favoring ventricular rather than supra-ventricular tachycardia in WCTs

There are several algorithms that are currently used to help distinguish Supraventricular Tachycardia (SVT) with aberrancy and Ventricular Tachycardia (VT) (**Table 3**). Many of these algorithms and criteria have limitations [44–49, 57–71].

4.1 Sandler and Marriott criteria (1965)

[RBBB morphology] Identical activation vector = SVT.

If the initial 20 ms of the QRS are the same in WCT as in sinus rhythm, SVT is favored with a positive predictive value (PPV) of 92%. The sinus rhythm ECG must be available for this analysis.

[RBBB morphology] An rSR' where S crosses baseline = SVT with a PPV of 91%.

[RBBB morphology] Triphasic QRS in V1 = SVT with a PPV of 92%.

[RBBB morphology, LBBB morphology] Precordial concordance = VT. A QRS, which is predominantly positive or predominantly negative in every precordial lead, overwhelmingly favors VT with specificity of 95–100% and a PPV of 89–100% [44–50, 57–71].

4.2 Wellens criteria of right bundle branch block

AV dissociation = VT. Of all criteria, this is the most secure with specificity of 100% and PPV of 100%. It holds true regardless of bundle branch pattern or other morphology criteria.

QRS duration	
• > 160 ms with LBBB pattern or > 140 ms with RBBB pattern	
• QRS duration during tachycardia is narrower than in sinus rhythm	
QRS axis	
• Right superior (northwest) axis	
• RBBB pattern with left axis deviation	
• RBBB pattern with normal axis	
• LBBB pattern with right axis deviation	
• QRS axis shift >40 degrees between sinus rhythm and tachycardia	
Precordial QRS concordance	
Positive or negative concordance in all precordial leads	
AV dissociation	
• AV ratio < 1	
• VA ratio > 1 (VA block)	
• Fusion beats	
• Capture beats	
RBBB morphology	
Lead V1	Lead V6
• mono or biphasic QRS	• R/S < 1, QS, QR, monophasic R
• R, qR, Rs, broad R	
• Triphasic QRS (Rsr' ['Rabbit ears'])	
LBBB morphology	
Lead V1–2	Lead V6
• Initial r wave ≥40 ms	• Any Q wave (QR, QS)
• Onset of QRS to S nadir interval ≥ 70 ms	(Absence of Q wave favors SVT)
• Notching on the downstroke of S wave	
Initial R wave (+) in lead aVR	
R wave peak time in lead II ≥ 50 ms	
Vi/Vt ≤ 1	

RBBB; right bundle branch block, LBBB; left bundle branch block, SVT; supra-ventricular tachycardia.

Table 3.
ECG criteria favoring ventricular rather than supra-ventricular tachycardia in WCTs.

[RBBB morphology] QRS duration >140 ms = VT with specificity of 57–75% and PPV of 89%.

[RBBB morphology] Left axis deviation = VT with PPVs of 88–94%. With extreme left axis (more negative than −90°), the PPV is 98%.

[RBBB morphology] Mono- or biphasic QRS morphologies in V1 favors VT with PPV of 82–83%.

If the V1 QRS is triphasic, an R:S ratio < 1 in V6 (that is, R wave smaller than S wave) favors VT with PPV of 90%.

[RBBB morphology] Rsr' ('Rabbit ears') = VT. In an unusual triphasic V1, with the left R wave taller than the right, and the S wave not crossing the baseline, favors VT with PPV of 100% [44–49, 57–71].

4.3 Griffith criteria

A history of myocardial infarction, QRS morphology in leads aVF and V1 ([1] predominant negative deflection in aVF in tachycardia with RBBB pattern and Q wave, [2] a monophasic or biphasic waveform in V1 in tachycardia with RBBB pattern, [3] QS or qR waveform in tachycardia with LBBB pattern favored a diagnosis of VT) and frontal plane axis > 40° when compared with baseline the ECG favored a diagnosis of VT. The presence of AV dissociation and/or the presence of premature ventricular beats during sinus rhythm that show morphologies same to that observed in tachycardia favored a diagnosis of VT [51–53, 57–71].

4.4 Kindwall criteria of left bundle branch block (LBBB)

[LBBB morphology] V1 or V2 with initial R > 30 ms = VT.
[LBBB morphology] V1 or V2 QRS onset to nadir of S wave >60 ms = VT.
[LBBB morphology] V1 or V2 with notching on the S wave downstroke = VT.
[LBBB morphology] Any Q in V6 = VT [54, 57–71].

4.5 Pava criteria using the measurement of the R-wave peak time (RWPT) in lead II

An R-wave peak time, with the interval from QRS onset to first change in polarity (R or S peak) in lead II ≥ 50 ms, independent of whether the complex is positive or

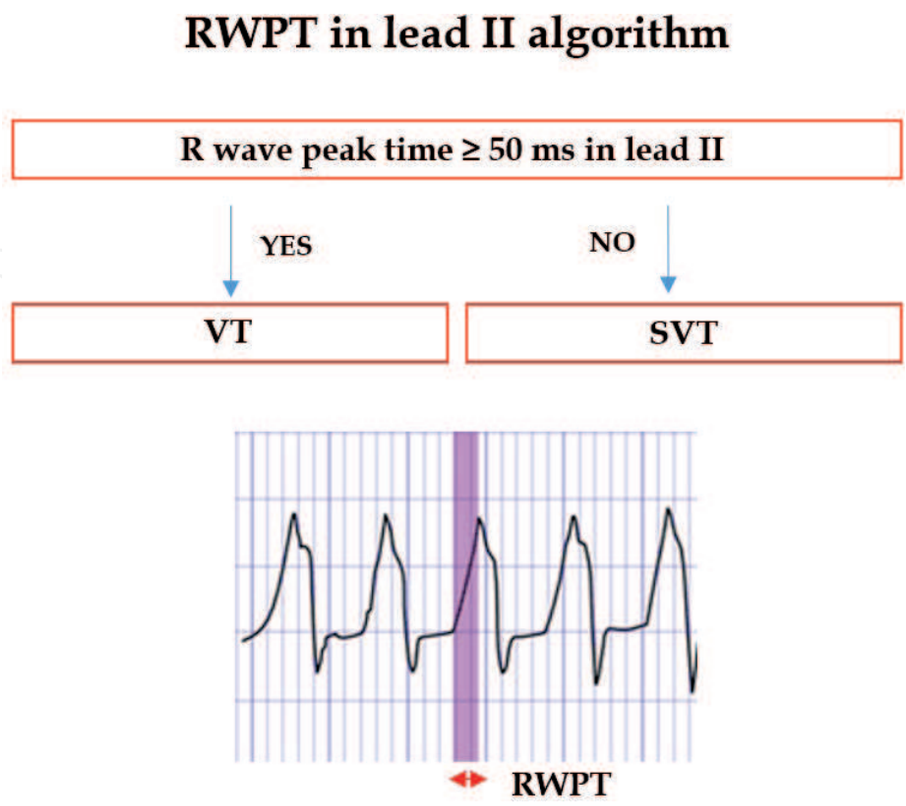

Figure 4.
The R-wave peak time (RWPT) in lead II.

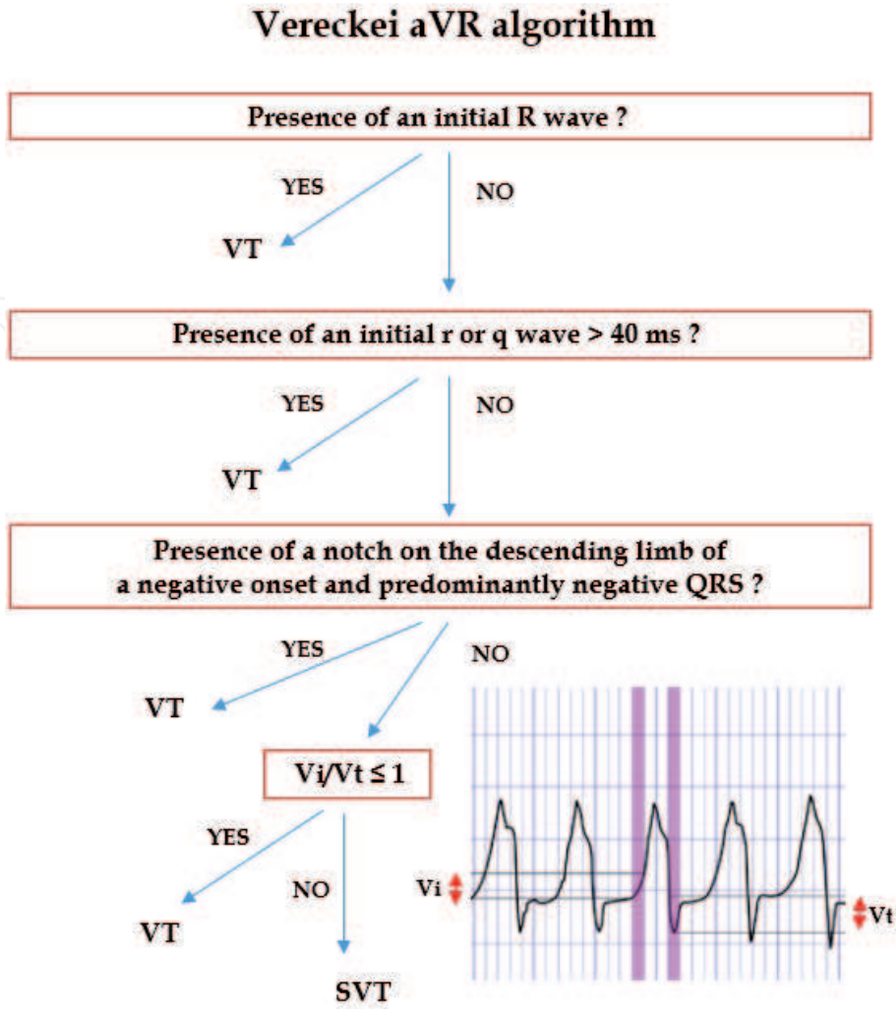

Figure 5.
Vereckei aVR algorithm.

negative, has been reported to have a sensitivity of 93% and specificity of 99% for identifying VT (**Figure 4**) [55, 57–71].

4.6 Vereckei aVR algorithm

Vereckei et al. published four-step algorithms with the incorporation of new criteria of V_i/V_t (**Figure 5**) [56–71].

The four steps were used in the following sequence:

1. If an initial R wave was present in lead aVR, VT was diagnosed.

2. If an initial, non-dominant q or r in aVR > 40 ms, VT was diagnosed.

3. If the morphology of WCT did not correspond to BBB or fascicular block, VT was diagnosed.

4. In the last step when the V_i/V_t ratio, obtained by measuring the voltage of the initial 40 ms (V_i) and the terminal 40 ms of a QRS (V_t) in any ECG lead, was ≤1 the diagnosis of VT, if the V_i/V_t was >1 the diagnosis of SVT was made (**Figure 5**).

During WCT due to SVT, after the initial rapid septal activation over the normal His-Purkinje system, the slow intraventricular activation occurs in the mid to terminal portion of the QRS, thus the $V_i/V_t > 1$.

4.7 Brugada algorithm

Brugada algorithm is the most widely known and commonly used algorithm (**Figure 6**) [57–71].

Brugada algorithm is as follows:

1. Is there concordance present in the precordial leads (leads V1-V6)?

"Are all of the QRS complexes completely upright, or downward in the precordial leads?"

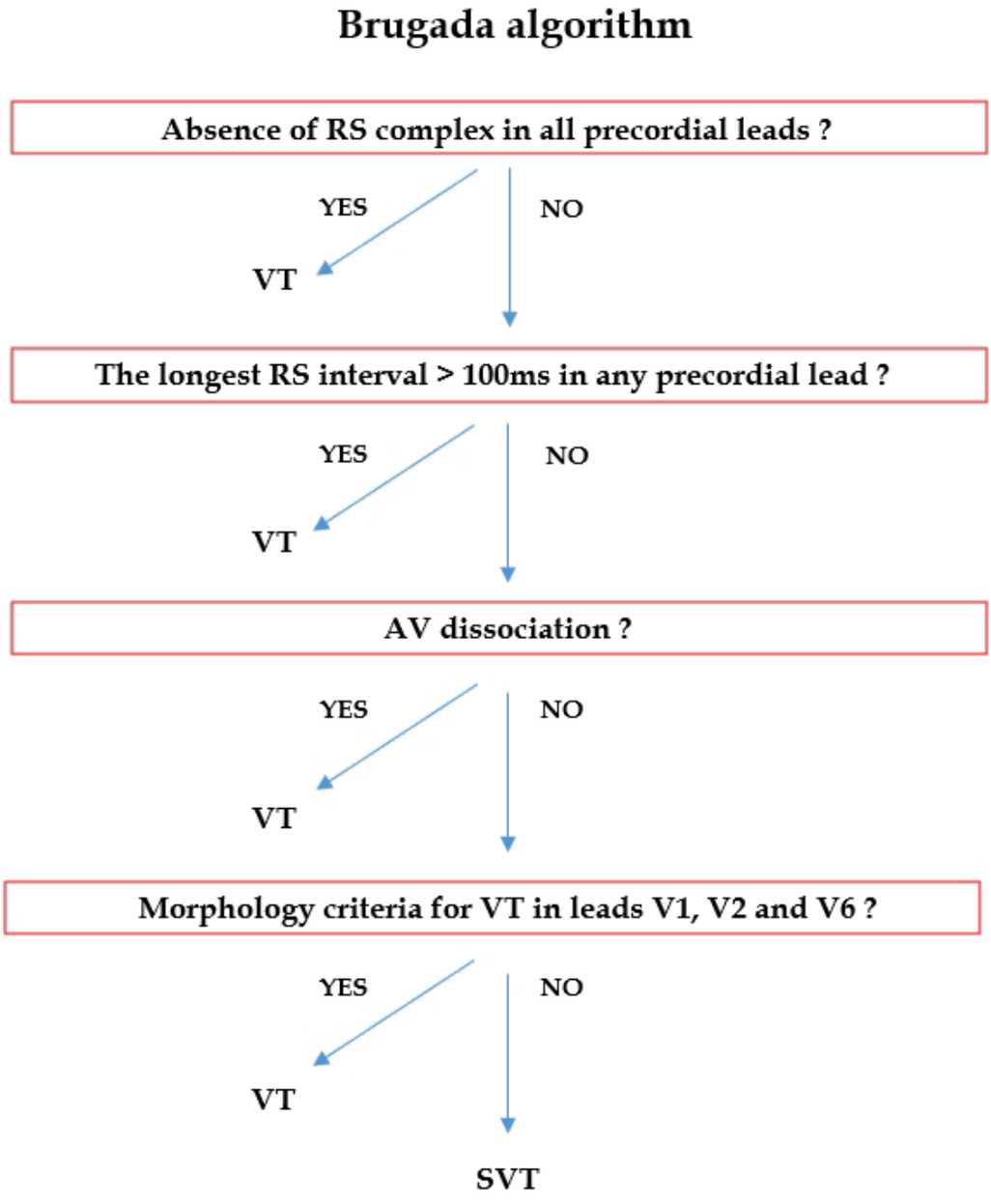

Figure 6.
Brugada algorithm.

Right bundle branch block morphology	**Lead V1: Monophasic R, biphasic qR, broad R (>40 ms), Rsr' (the so-called 'rabbit ears' sign)** **Lead V6: R:S ratio < 1**
Left bundle branch block morphology	Lead V1–2: Broad R wave, slurred or notched downstroke of S wave, delayed nadir of S wave Lead V6: Q or QR or QS wave

Table 4.
Morphology criteria for VT in leads V1, V2 and V6.

If the answer is yes, then VT is the diagnosis

2. Is the R to S interval (between the onset of the R wave and the nadir of the S wave) > 100 ms in any one precordial lead?

If the answer is present, then VT is the diagnosis

3. Is AV dissociation present?

"AV dissociation occurs when P waves are seen at different rates than the QRS complexes."

If the answer is present, then VT is the diagnosis

4. Examine the morphology of the QRS complex to see if it meets the specific criteria for VT, as **Table 4**.

5. Conclusions

The ECG criteria or algorithms for the diagnosis of NCTs and WCTs has undergone evolution and development in concert with the field of cardiology itself, but the necessity of a correct diagnosis remains unchanged [57–71]. The world has not yet seen the 'one criterion to end all criteria' or 'simplest criterion' with high sensitivity and specificity, and it seems unlikely to appear in our near future. Therefore, physicians or cardiologists should be cautioned against overreliance in these ECG criteria or algorithms for the interpretation of the ECGs.

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