

DIFFERENTIAL DIAGNOSIS OF TACHYARRHYTHMIAS AND THERAPY IN THE ABSENCE OF ESTABLISHED DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

A classification of narrow and wide-QRS tachycardias for differential diagnosis purposes is presented in Table 1.

Narrow-QRS tachycardias

Narrow QRS complexes are due to rapid activation of the ventricles via the His-Purkinje system, which suggests that the origin of the arrhythmia is above or within the His bundle. However, early activation of the His bundle can also occur in high septal VTs thus resulting in relatively narrow QRS complexes (110–140 ms)¹.

Electrocardiographic differential diagnosis

A 12-lead ECG taken during tachycardia is essential, although it may fail to lead to a specific diagnosis². A 12-lead ECG in sinus rhythm may also provide clues for the diagnosis of SVT. The presence of pre-excitation in a patient with a history of regular paroxysmal palpitations is generally suggestive of AVRT. The absence of apparent pre-excitation does not rule out the diagnosis of AVRT, since it may be due to a concealed AP that conducts only retrogradely.

Initiation and termination of tachycardia. Sudden prolongation of the PR interval occurs in typical AVJRT after an atrial ectopic beat. An AT may also be initiated by an atrial ectopic beat, but is not dependent on marked PR prolongation. Automatic, focal ATs are characterized by gradual acceleration (warm-up phenomenon), and then deceleration (cool-down phenomenon).³ Premature atrial or ventricular beats may trigger AVRT.

Regularity of tachycardia cycle length. Irregular tachycardias may represent focal or multifocal AT (MAT), focal AF and AFL with varying AV conduction (Figures 1 and 2). Irregular arrhythmias typically display variable P wave morphologies, and varying PP, RR, and PR intervals. AFL can have fixed AV conduction and present as a regular tachycardia, and even AF may appear almost regular when very fast. Incessant tachycardias may also be a permanent junctional reciprocating tachycardia (PJRT) and, rarely, atypical AVJRT. Re-entrant tachycardias, whether micro- or macro-re-entries, are usually regular. Cycle length (RR) alternans may be seen in AVJRT but these are periodic, constant changes, less than 15% of the tachycardia CL.⁴ If the irregularity exceeds 15% of the cycle length, a focal arrhythmia is much more likely⁵. QRS alternans is a rare phenomenon in slow SVTs, that may not be related to cycle length alternans, and has been initially described with AVRT^{6,7}. However, this may be seen in any fast SVT⁸. A change in ventricular CL preceded by a change in atrial CL is seen in

AT or atypical AVJRT. A change in ventricular CL preceding a change in subsequent atrial CL favours typical AVJRT or AVRT.^{4,9} A fixed ventriculoatrial (VA) interval in the presence of variable RR intervals excludes AT.²

P/QRS relationship. According to their P/QRS relationships, SVTs are classified as having short or long RP intervals. Short RP SVTs are those with RP intervals shorter than half the tachycardia RR interval, whereas long RP SVTs display $RP \geq PR$. Rarely, recording of U waves during typical AVJRT may simulate a long RP tachycardia¹⁰. At EPS very short VA interval (≤ 70 ms) usually indicates typical AVJRT, and, less commonly focal AT, although, rarely, it may also be seen in AVRT. A RP cut-off interval of 90 ms has been shown to be useful for surface ECG measurements, and can be used if P waves are visible¹¹, but data on actual RP measurement during various types of SVT are scarce. P waves similar to those in normal sinus rhythm suggest appropriate or inappropriate sinus nodal tachycardia, sinus nodal reentrant tachycardia, or focal AT arising close to the sinus node. P waves different from those in sinus rhythm and conducted with a PR interval equal to or longer than the PR in sinus rhythm are typically seen in focal AT. In AT, the conduction to the ventricles may be fast (1:1) or slow (3:1 or 4:1). The possibility of AFL with 2:1 conduction should also be considered if the ventricular rate during SVT is ~ 150 bpm, since the atrial activity is usually 250-330 bpm. In the presence of antiarrhythmic medication, lower atrial rates may also result in higher ventricular rates so should be administered together with drugs influencing AV-conduction.

In the case of relatively delayed retrograde conduction that allows the identification of retrograde P waves, a pseudo r deflection in lead V1 and a pseudo S wave in the inferior leads are more common in typical AVJRT than in AVRT or AT^{12,13}. These criteria are specific (91–100%) but modestly sensitive (58% and 14%, respectively)¹². A difference of RP intervals in leads V1 and III > 20 ms is also indicative of AVJRT rather than AVRT due to a posteroseptal pathway¹³. The presence of a QRS notch in lead aVL has also been found as a reliable criterion suggesting AVJRT¹⁴, while a pseudo r in aVR was shown to have higher sensitivity and specificity than a pseudo r in V1 for the diagnosis of typical AVJRT¹⁵. However, in all of these studies, cases of AT or atypical AVJRT were limited or entirely absent.

AV block or dissociation during narrow QRS tachycardia is not often seen, but it rules out AVRT, as both atria and ventricles are parts of the circuit. The development of bundle branch block (BBB) during SVT may also be helpful in the diagnosis of AVRT. BBB ipsilateral to the AP can result in CL prolongation due to VA prolongation, as the ventricular arm of the circuit is prolonged by conduction through the interventricular septum from the conducting bundle branch¹⁶. It should be noted, however, that lengthening in VA interval may not necessarily result in CL prolongation, due to switch of antegrade conduction from slow to fast AV nodal pathway.

Vagal maneuvers and adenosine. Vagal maneuvers (such as carotid sinus massage) and adenosine injection may help in clinical diagnosis, particularly in situations in which the ECG during tachycardia is unclear. Possible responses to vagal maneuvers and adenosine are shown in Figure 3. Termination of the arrhythmia with a P wave after the last QRS complex is very unlikely in AT, and most common in AVRT and typical AVJRT. Termination with a QRS complex is often seen in AT, and possibly in atypical AVJRT. Adenosine does not interrupt macro-reentrant atrial tachycardias¹⁷. Fascicular VTs, in particular, are verapamil but not adenosine-sensitive. Most VTs, as opposed to SVTs, do not respond to carotid sinus massage, but a narrow QRS VT originating at the left bundle branch and terminated with carotid sinus massage has been reported¹⁸.

Electrophysiology study

On certain occasions, an electrophysiology study may be necessary to establish the diagnosis. Several electrophysiologic techniques and maneuvers can be employed for differential diagnosis of regular narrow QRS tachycardia.^{19,20}

Wide-QRS tachycardias

Wide QRS tachycardias can be VT, SVT conducting with bundle branch block aberration, or over an AP, with reported proportions of 80, 15 and 5%, respectively²¹. The correct diagnosis of VT is critical to management as misdiagnosis and administration of drugs usually utilized for SVT can be harmful for patients in VT²². **Therefore, the default diagnosis should be VT until proven otherwise.** The differential diagnosis includes²³:

1. SVT with BBB. This may arise due to pre-existing BBB or the development of aberrancy during tachycardia (so-called phase 3 block) which is more commonly a right bundle branch block (RBBB) pattern, due to the longer RP of the right bundle branch.
2. SVT with antegrade conduction over an AP, “pre-excited SVT”, that participates in the circuit (antidromic AVRT) or is a bystander during AF, focal AT/AFL, AVJRT.
3. SVT with widening of QRS interval induced by drugs or electrolyte disturbances. Class IC and IA drugs cause use-dependent slowing of conduction, and class III drugs prolong refractoriness at His-Purkinje tissue more than in the ventricular myocardium. They can both result in atypical BBB morphologies during SVT that mimics VT.
4. Pacemaker-related endless loop tachycardia and artefacts can also mimic VT.

Electrocardiographic differential diagnosis

If the QRS morphology is identical during sinus rhythm and tachycardia then this is most likely not a VT. However, bundle branch re-entrant VTs and high septal VTs exiting close to the conduction system can have similar morphologies to sinus rhythm. The presence of a contralateral BBB pattern in sinus rhythm is more indicative of VT.

AV dissociation. The presence of either AV dissociation or capture/fusion beats in the 12-lead ECG during tachycardia are key diagnostic features of VT (Table 2). AV dissociation may be difficult to recognize because P waves are often hidden by wide QRS and T waves during a wide QRS tachycardia. P waves are usually more prominent in inferior leads, and modified chest lead placement (Lewis lead).²¹ The relation between atrial and ventricular events is 1:1 or greater (more atrial than ventricular beats) in most SVTs. AVJRT can be associated with 2:1 conduction,²⁴ but this is rare. Although VA conduction can be found in up to 50% of patients with VT and a 1:1 relation is possible, most VTs have a relation <1:1 (more QRS complexes than P waves).

QRS duration. A QRS duration >140 ms with RBBB or >160 ms with LBBB pattern suggests VT. These criteria are not helpful for differentiating VT from SVT in specific settings, such as pre-excited SVT or when class IC or class IA antiarrhythmic drugs are administered²⁵.

QRS axis. Since VT circuits (especially post MI or in cardiomyopathies) frequently lie outside the normal His-Purkinje network, significant axis shifts are likely to occur, enabling diagnosis. Therefore, in SVT patients with aberrancy the QRS axis is confined between -60° and +120°. Extreme axis deviation (axis from -90° to ± 180°), in particular, is strongly indicative of VT both in the presence of RBBB and LBBB²³.

Chest lead concordance. The presence of negative chest lead concordance (all QRS complexes negative V1–V6) is almost diagnostic of VT with a specificity of >90% but only present in 20% of VTs (Figure 3). Positive concordance can be indicative of VT or an antidromic tachycardia utilizing a left posterior or left lateral AP²⁶.

RBBB morphology

Lead V1: Typical RBBB aberrancy has a small initial r', since in RBBB the high septum is activated primarily from the left septal bundle. Therefore, the following patterns are evident: rSR', rSr', or rR', in lead V1. However, in VT the activation wavefront progresses from the LV to the right precordial lead V1, in a way that a prominent R wave (monophasic R, Rsr', biphasic qR complex, or broad R >40 ms) will be more commonly seen in lead V1.²⁷ Additionally, a double-peaked R wave (M pattern) in lead V1 favours VT if the left peak is taller than the right peak (the so-called "rabbit ear" sign). A taller right rabbit ear characterizes the RBBB aberrancy but does not exclude VT.

Lead V6: A small amount of normal RV voltage is directed away from V6. Since this is a small vector in RBBB aberrancy, the R:S ratio is >1. In VT, all of the RV voltage, and some of the left, is directed away from V6, leading to an R:S ratio <1 (rS, QS patterns). An RBBB morphology with a R:S ratio in V6 of <1 is seen rarely in SVT with aberrancy, mainly when the patient has a left axis deviation during sinus rhythm.

Differentiating fascicular VT from SVT with bifascicular block (RBBB and left anterior hemiblock) is very challenging. Features that indicate SVT in this context include QRS > 140 ms, r' in V1, overall negative QRS in aVR, and R/S ratio >1 in V6.

LBBB morphology

Lead V1: As stated above for RBBB, for the same reasons, the presence of broad R wave, slurred or notched down stroke of the S wave, and delayed nadir of S wave are strong predictors of VT.²⁷

Lead V6: In true LBBB, no Q wave is present in the lateral precordial leads. Therefore, the presence of any Q or QS wave in lead V6 favours VT indicating the activation wavefront is moving away from the LV apical site.

A number of algorithms have been developed to differentiate VT from SVT.²⁸⁻³⁰ The most established are the Brugada algorithm,²⁸ and the Vereckei algorithm²⁹ which utilizes a single lead aVR.³¹

RS interval in precordial leads.

The absence of RS complex in precordial leads (only R and S complexes are seen on ECG) is only found in VTs (Figure 4). An RS complex is found in all SVTs and in 74% of VTs. The longest interval from the onset of the R wave to the deepest part of the S wave longer than 100 ms, irrespective of the morphology of the tachycardia, is not observed in any SVT with aberrant conduction. About half of the VTs have an RS interval of 100 ms or less and the other half have an RS interval of more than 100 ms. The Brugada et al algorithm has a sensitivity and specificity of 98,7% and 96,5%, respectively.²⁸

QRS complex in aVR lead.

During sinus rhythm and SVT, the wavefront of depolarization proceeds in a direction away from lead aVR, yielding a negative QRS complex in lead aVR with few exceptions (eg inferior MI). On the contrary, the presence of an initial R wave (Rs complex) in aVR suggests VT (Figure 5). The Vereckei et al algorithm has a 91.5% overall accuracy in the diagnosis of VTs.²⁹

R-wave peak time (RWPT) at lead II ≥ 50 ms.

This criterion has the potential advantage that lead II is a lead easy to obtain and it almost always is present on ECG rhythm strips recorded in different settings (e.g. ECG monitoring in emergency rooms and intensive care units). The R-wave peak time (RWPT) at lead II ≥ 50 ms, independent of whether the complex is positive or negative, has been reported to have a sensitivity of 93%, and a specificity of 99%, for identifying VT (Figure 6),²⁹ but these results were not verified in the first large external application of this criterion.³²

All these criteria have limitations. Conditions like bundle branch reentrant tachycardia, fascicular VT, VT with exit site close to the His–Purkinje system, and wide-QRS tachycardia occurring during antiarrhythmic drug treatment are difficult to diagnose by using

mentioned morphological criteria. This is most pronounced in VT originating from septal sites, particularly Purkinje sites and the septal outflow tract regions.³³ Left posterior fascicular VT, the most common form of idiopathic left ventricular VT is frequently misdiagnosed as SVT with RBBB and LAH aberration (Figure 7).³⁴

Differentiation between VT and antidromic AVRT is extremely difficult for the very fact that the QRS morphology in antidromic AVRT is similar to that of a VT with its origin at the insertion of the accessory pathway in the ventricular myocardium. An algorithm has been derived for differential diagnosis, based on the analysis of 267 wide-QRS tachycardias, consisting of VT and antidromic AVRT. The derived criteria were found to offer sensitivity of 75% and specificity of 100%³⁵, and the algorithm was also validated in another study,³⁶ but experience is still limited.

In fact, several independent studies have found that various ECG-based methods have specificities of 40–80% and accuracies of ~75%^{1,26,37–40}. Indeed, a similar diagnostic accuracy of ~75% would be achieved effortlessly by considering every wide QRS tachycardia to be a VT, because only 25–30% are SVTs. Therefore, emerging approaches to integrate these algorithms to provide more accurate scoring systems are being evaluated.⁴¹

Electrophysiology study

On certain occasions, an EPS is necessary for diagnosis.

Irregular Tachycardias

An irregular ventricular rhythm most commonly indicates AF, MAT, or focal AT/AFL with variable AV conduction, and may occur in the context of both narrow and broad QRS complexes. When AF is associated with rapid ventricular rates, the irregularity of this ventricular response is less easily detected and can be misdiagnosed as a regular SVT⁴². If the atrial rate exceeds the ventricular rate, then AFL or AT (focal or multifocal) is usually present. Polymorphic VT and, rarely, monomorphic VT, may also be irregular. Occasionally, a junctional, non-reentrant tachycardia may have a variable rate.

The differential diagnosis of an *irregular wide QRS* tachycardia is either preexcited AF or polymorphic VT or atrial arrhythmias with variable block in the context of aberrancy. Pre-excited AF is manifest itself by irregularity, a varying QRS morphology, and a rapid ventricular rate (owing to the short RP of the AP). The changing QRS morphology results from varying degrees of fusion due to activation over both the AP and the AVN (or over two APs) which also results in variation in the width of the delta wave. The ventricular rate tends to be higher than in those with non-pre-excited AF.⁴³

ACUTE THERAPY IN THE ABSENCE OF DIAGNOSIS

Narrow-QRS Tachycardias

A narrow-QRS tachycardia usually (although not invariably) represents an SVT and acute therapy is discussed in Epidemiology and Principles of Therapy of SVT.

Wide-QRS Tachycardias

In a patient with wide QRS tachycardia who is haemodynamically stable, the response to **vagal manoeuvres** may provide insight to the mechanism responsible for the arrhythmia. SVT with aberrancy, if definitively identified, may be treated in the same manner as narrow complex SVT, with vagal manoeuvres or drugs (adenosine and other AV node blocking agents such as beta-blockers or calcium-channel blockers) (Figures 8 and 9, and Tables 3 and 4).⁴⁴

Some drugs used for the diagnosis or treatment of SVT (e.g. verapamil) can cause severe haemodynamic deterioration in patients with a previously stable VT.^{22,45,46} Thus, they should only be used for the treatment of patients in whom the diagnosis of SVT is fully established and secure. **Adenosine** may be helpful by means of allowing a diagnosis or interrupting an adenosine-sensitive VT, but it must be avoided if pre-excitation on resting ECG suggests a pre-excited tachycardia. There is a risk that in antidromic re-entry it may precipitate cardiac arrest if there is induction of AF by adenosine, as may occasionally occur.⁴⁷

For pharmacologic termination of a haemodynamically stable wide QRS complex tachycardia of unknown aetiology, i.v. **procainamide or amiodarone** can be used in hospital. In the PROCAMIO trial in patients with well-tolerated wide QRS tachycardia and with or without reduced LVEF, procainamide was associated with less major cardiac adverse events and a higher proportion of tachycardia termination within 40 min, compared to amiodarone.⁴⁸ In case the mechanism of the arrhythmia is not fully understood, the arrhythmia should be treated as VT.

Irregular Tachycardias

A **wide QRS complex irregular tachycardia** is usually a manifestation of AF. Rarely polymorphic VT and, very rarely, monomorphic VT may also present as irregular tachycardias.⁴⁹ Electrical cardioversion is the acute treatment of choice in irregular pre-

excited tachycardias associated with haemodynamic instability. Specific therapy of preexcited AF is discussed in the Chapter on AVRT.

If the rhythm is well tolerated with a **narrow QRS complex irregular tachycardia**, this should be considered to be likely AF, and rate control with beta-blockers or calcium-channel blockers and elective chemical or electrical cardioversion once thromboprophylaxis is in place, may be appropriate.

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