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REVIEW

Diagnostic criteria of broad QRS complex tachycardia: decades of evolution

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Broad QRS complex tachycardia still presents a diagnostic challenge when confronted with a 12-lead electrocardiogram (ECG). The ECG differential diagnosis includes ventricular tachycardia vs. supraventricular tachycardia with functional aberration, pre-existing bundle branch block, intraventricular conduction disturbances, or pre-excitation. Despite all available criteria, broad complex tachycardias are still misdiagnosed or remain undiagnosed. This paper will briefly review the most recognized criteria.

Keywords Broad QRS complex tachycardia • Wide QRS complex tachycardia • Ventricular tachycardia

Introduction

Broad QRS complex tachycardia (BCT) still presents a diagnostic challenge when confronted with a 12-lead electrocardiogram (ECG). The ECG differential diagnosis includes ventricular tachycardia (VT) vs. supraventricular tachycardia (SVT) with aberrant conduction, pre-existing bundle branch block (BBB), intraventricular conduction disturbances, or pre-excitation. Ventricular tachycardia is the most important differential diagnosis because of its least favourable prognostic value. An accurate diagnosis with an immediate treatment is usually required. Delay or misdiagnosing VT with an inappropriate intravenous administration of drugs used for the treatment of SVT, such as verapamil and adenosine, can cause severe haemodynamic deterioration and may provoke ventricular fibrillation (VF) and cardiac arrest.[1–6] Several criteria have been described for the differentiation between VT and SVT with a wide QRS complex. This paper will briefly review the most recognized criteria.

History and physical examination

Ventricular tachycardia is the most common cause of regular BCT, accounting for up to 80% of all cases. The history of prior myocardial infarction (MI), congestive heart failure, and recent angina pectoris has positive predictive values for VT of 98, 100, and 100%, respectively. Age >35 years has a sensitivity of 92%.[7] Cardiovascular signs and symptoms during tachycardia such as palpitation, syncope or angina, blood pressure, and patient's clinical status are important in indicating the haemodynamic state of the patients but they are not helpful in determining the mechanism of the tachycardia.[8–10] Mostly, VT does not respond to carotid sinus massage (CSM). Reentrant SVT usually slows down and may stop with carotid pressure. Cyclic-AMP-related VT that terminates with CSM has also been reported.[11]

Atrioventricular dissociation

The finding of atrioventricular (AV) dissociation is one of the most useful practical criteria for the diagnosis of VT. However, retrograde ventriculo-atrial (VA) conduction, whether 1 to 1 or 2 to 1, or Wenckebach VA conduction occurs in up to 50% of all VT.[12,13] In these cases, intravenous adenosine—although not completely safe—may assist in the diagnosis by establishing complete VA block and revealing AV dissociation. Atrioventricular dissociation can also be seen in rare cases of AV nodal reentry tachycardia (AVNRT) and uncommon types of Mahaim tachycardia (nodo-fascicular/nodo-ventricular), in which the retrograde conduction is absent.[14–17]

Clinical detection of atrioventricular dissociation

Stroke volume and blood pressure fluctuations with variable intensity of the first heart sound can be observed during AV dissociation.[18] The presence of canon A waves as a reflection of simultaneous atrial and ventricular contraction in the presence of AV dissociation strongly suggests VT and should be distinguished from the frog sign which occurs during every beat and is usually

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seen in AVNRT. Interestingly, the frog sign is usually visible only in cases of AVNRT and not in cases of VT with retrograde VA conduction. A possible explanation is the change in the position of the AV ring during the systolic cycle. During AVNRT, the retrograde P occurs early in systole when the AV ring is still positioned backward towards the atria, whereas during VT with retrograde conduction, the retrograde P occurs later in systole when the AV ring has moved towards the apex of the heart which enlarges the atria and minimizes the venous backflow. Through the same mechanism, the frog sign is absent in orthodromic circus movement tachycardias.[19]

Electrocardiographic detection of atrioventricular dissociation

In our experience, AV dissociation is generally detectable in a lead where the P-wave is most prominent whether anterograde or retrograde. This lead is usually one of the inferior leads considering the lead in which the QRS complex is the most modest, i.e. least interfering with the relatively prominent P-wave. QRS variability can indicate AV dissociation. Obviously, care should be taken to record technically excellent ECGs without disturbances of the baseline. The mechanism of changes in QRS amplitude is not completely clear. The most probable explanation is that the QRS voltage is determined by the variability of ventricular filling and volume. Additionally, super-imposition of the P-wave on the R-wave or mechanical factors such as changes in the ventricular position within the thorax following different atrial contractions may play a role. Electrical alternans is usually seen in (narrow complex) circus movement tachycardia and beat-to-beat variations can occur when the atrial rate is half the ventricular rate, or in the case of alternating discrete initial conduction disturbance with beat-to-beat variation in depth of initial q-wave in lead V6. The use of the Lewis lead can be helpful in detecting the P-waves on the ECG. The Lewis lead can be obtained from registration of lead I by placing the right arm electrode to the right, second intercostal space adjacent to the sternum, and the left arm electrode to the right, fourth intercostal space adjacent to the sternum. Voltage should be calibrated at 1 mV = 20 mm (*Figure 1*).[20]

Echocardiographic detection of atrioventricular dissociation

Although very valuable and currently widely feasible, echocardiography has infrequently been used in the detection of AV dissociation. Atrioventricular dissociation can be easily detected using different echocardiographic modalities like M-mode, mitral valve movement, flow Doppler, or tissue Doppler (*Figure 2*).

The atrial fibrillation toolbox

The atrial fibrillation (AF) toolbox is a new medical tool designed to detect, display, and enlarge atrial activities with separation/suppression of the QRS complexes.[21] The atrial activity can therefore be analysed isolated from the ventricular activity. This device is originally developed to detect AF. The AF toolbox might play an important role in the detection of AV dissociation, but its value in BCT still needs further evaluation.

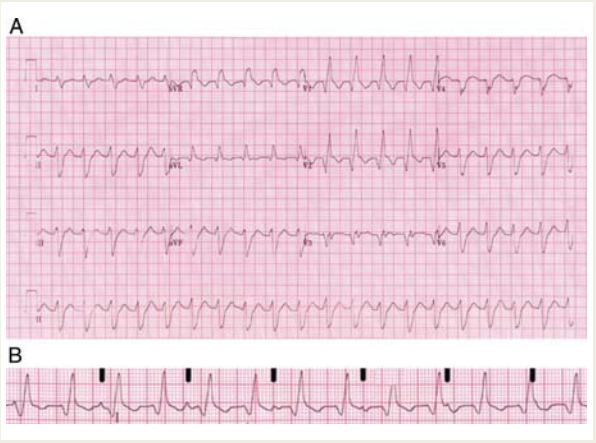

Figure 1 Lewis lead (*B*): during BCT (*A*), the presence of atrioventricular dissociation is indicated by vertical black bars. Reproduced with permission from the publisher.[20]

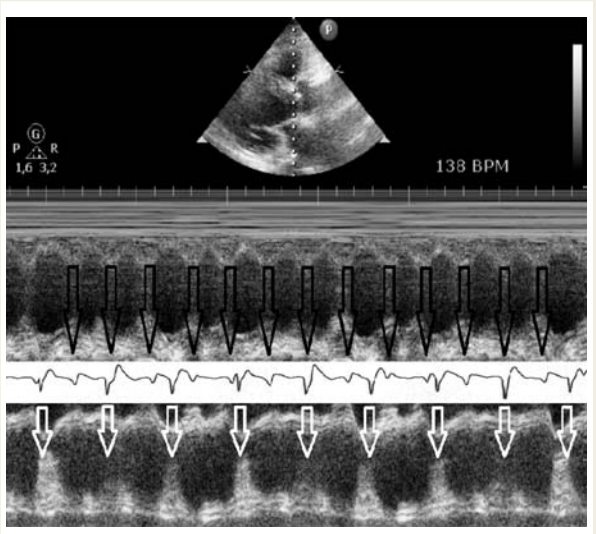

Figure 2 Echocardiographic detection of AV dissociation. Black arrows represent right ventricular contraction. White arrows represent left atrial contraction. A rhythm strip made from an external pacemaker lead positioned in the right atrium showing a clear correlation with the echocardiographic M-mode image.

Electrocardiographic criteria

The classical criteria

In 1965, Sandler and Marriott[22] attempt to differentiate between ventricular ectopic beats (VEBs) with right BBB (RBBB)-like morphology and unselected examples of RBBB. They found that 92% of VEBs with a RBBB have a monophasic or a biphasic pattern in lead V1, whereas a triphasic pattern (rsR′, rSR′, RsR′) was only found in 6% of VEBs. The generally accepted criteria to recognize ventricular extrasystoles at their time were those beats that have no visible evidence of premature atrial activity preceding them

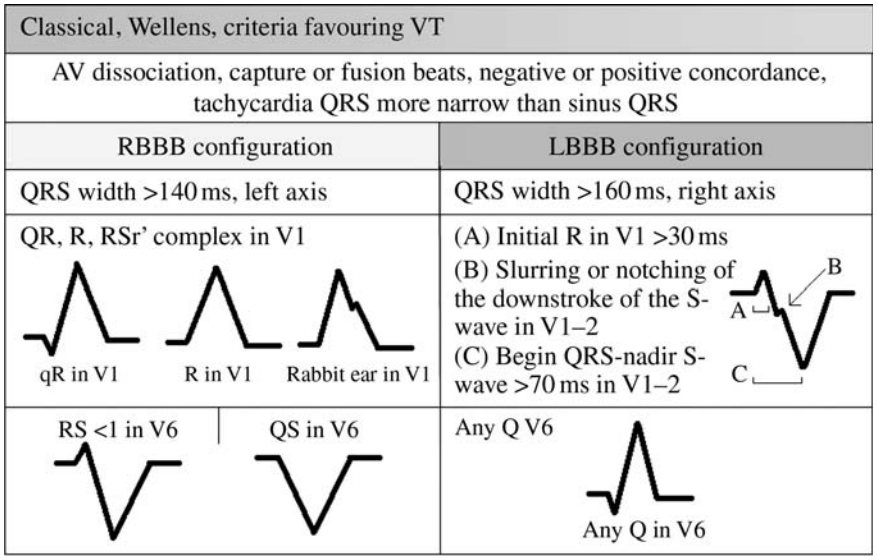

Figure 3 Classical 'Wellens' criteria favouring VT in patients without AAD.[1,12] One hundred VTs (68 ischaemic, 18 idiopathic, and 14 miscellaneous) and 100 SVTs with aberrant conduction were included in the comparison.

and are followed by a fully compensatory pause; the authors were well aware that such beats are indistinguishable from AV nodal extrasystoles with aberration and no retrograde conduction. Marriott[15] and Marriott and Sandler[18] also noticed that 'concordant' precordial patterns, whether entirely upright or entirely inverted, were almost diagnostic for ventricular origin. Positive concordance may also occur during antidromic tachycardia using a left posterior or left lateral accessory pathway. Negative concordance is nearly always VT. However, Volders *et al.*[23] published a case report of a 17-year-old male with pectus excavatum and SVT with left BBB (LBBB) resulting in a BCT with negative concordance. Marriott also introduced in 1971 the term 'Rabbit ears' for a double-peaked R-wave in lead V1. Whereas a 'good rabbit' with a taller right peak being typical for RBBB aberrancy, a 'bad rabbit' with a taller left peak suggests ventricular origin. In 1972, Swanick *et al.*[24] continued the differentiation between right VEB and supraventricular beats showing LBBB and found that (i) the presence of S-wave in V4 greater in depth than the S in lead V1, (ii) a wide r-wave $\geq$0.03 s in lead V1, and (iii) negative QRS polarity in lead I, all favour ventricular origin. In 1978, Wellens *et al.*[12,13] used His-bundle recording for the first time to determine the site of origin of tachycardia with broad QRS complex and create the so-called 'classical criteria' (*Figure 3*).[25] Later, Coumel *et al.*[26] showed that a QR pattern in leads other than aVR or a QS pattern in V5–6 during VT was present in 89% of the patients with old MI, while constantly absent in patients with idiopathic VT.

The Brugada algorithm

In 1991, Brugada *et al.*[27] analysed 554 BCTs and produced simple criteria in a stepwise approach irrespective of the QRS complex morphology, whether RBBB- or LBBB-like, with a sensitivity and specificity of 0.987 and 0.965, respectively. The algorithm (*Figure 4*) begins with the identification of an RS complex in any precordial lead, if failed the diagnosis of VT is made. If an RS complex is present in one or more precordial leads, the next step is to measure the longest RS interval. If an RS interval is longer than 100 ms, the diagnosis of VT is made. If not, the next step of the algorithm is to consider whether AV dissociation is present. If so, the diagnosis of VT is made. If absent, the classical morphology criteria for VT are used (*Figure 3*). If both lead V1 and V6 fulfil the criteria for VT, the diagnosis of VT is made. If not, the diagnosis of SVT with aberrant conduction is made by exclusion of VT.

The aVR 'Vereckei' algorithm

In 2007, a new algorithm analysed in 287 patients with a high accuracy[27] has been proposed by Vereckei *et al.*[28] They found that the following criteria were suggestive of VT: (i) the presence of AV dissociation; (ii) the presence of an initial R-wave in lead aVR, and (3) measuring the voltage during the initial 40 ms (V_i), the terminal 40 ms (V_t), their ratio (V_i/V_t), and that $V_i/V_t \leq 1$ was suggestive of VT (*Figure 5*). In 2008, the same group presented a simplified algorithm using only lead aVR, analysed in 313 patients with the same accuracy[29] as their first algorithm. Criteria for VT in lead aVR were (i) the presence of an initial R-wave, (ii) width of an initial r- or q-wave >40 ms, (iii) notching on the initial downstroke of a predominantly negative QRS complex, and (4) $V_i/V_t \leq 1$ (*Figure 6*).

Other criteria

Griffith *et al.*[30,31] performed in 1991 a multivariate analysis in 102 patients to identify which of 15 clinical or 11 ECG variables are independent predictors of VT. They found that (i) the history of previous MI, (ii) in lead aVF, a predominant negative deflection was suggestive of VT especially when Q-wave was present in RBBB pattern tachycardia. In LBBB pattern tachycardia, a QS or qR waveform in lead aVF is highly suggestive for VT, whereas an Rs complex was specific for SVT, (iii) in RBBB pattern tachycardia, a monophasic or biphasic

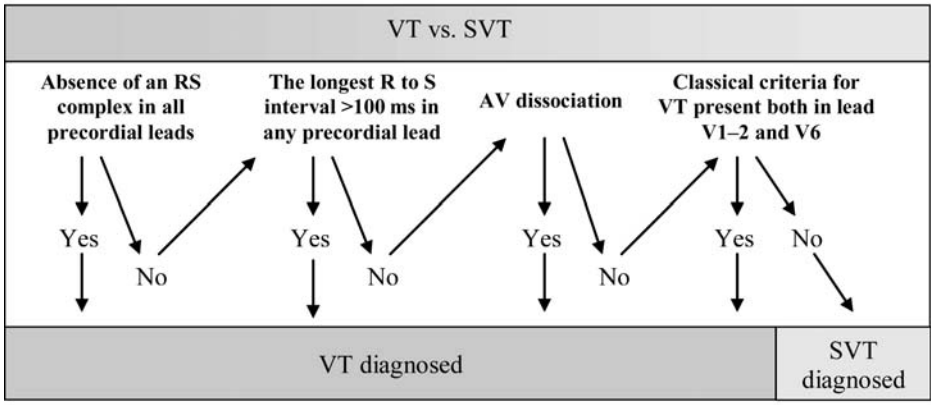

Figure 4 Three hundred and forty-eight VTs and 170 SVTs with aberrant conduction were included in the comparison. None of the patients were on antiarrhythmic drugs (the Brugada algorithm[27]).

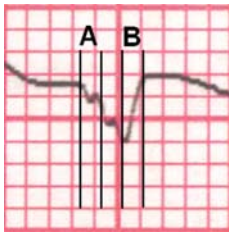

Figure 5 Velocity ratio between initial and terminal 40 ms of the QRS complex. Example of the use of V_i/V_t in lead aVR: (*A*) in the initial 40 ms, the voltage of the QRS complex is difficult to measure because of the notch, but it is <0.2 mV. (*B*) in the terminal 40 ms, the voltage is 0.3 mV. V_i (0.2)/V_t (0.3) <1, suggesting slower initial forces and thus ventricular tachycardia. A ratio >1 suggests SVT (Vereckei *et al.*[28]).

waveform in lead V1 suggested VT, a triphasic RSR, rSR configuration suggested SVT, and (iv) change in axis of more than 40° between sinus rhythm (SR) and tachycardia were independent predictors of VT. If none of the above variables was found, the diagnosis was almost certainly SVT. If one criterion was found, the diagnosis was probably SVT. If two criteria were found, then the diagnosis was probably VT. If three or four criteria were found, the diagnosis was almost certainly VT. The predictive accuracy of this method was 93%, increased to 95% by including two other criteria: independent P-wave activity and VEB during sinus rhythm (SR) with the same QRS morphology as that in tachycardia. In another series, Griffith *et al.*[31] studied 53 patients with LBBB pattern tachycardia and found that the classical VT criteria have a sensitivity of 100% in patients with previous MI, but only 50% in patients with structurally normal hearts or non-ischaemic cardiomyopathy. Griffith *et al.*[32] proposed later that unless typical BBB morphology was found in the setting of BCT, VT should be diagnosed by default. Grimm *et al.*[33] compared in 240 BCTs the value of the classical criteria published by Wellens in 1978 and those published by Brugada in 1991 and found that both criteria have a sensitivity of more than 90% and a specificity of about 70% for tachycardias with RBBB morphology and 87% for tachycardias with LBBB morphology. They also found that the combination of these two criteria did not increase sensitivity or specificity. Alberca *et al.*[34] analysed 12 previously described morphological criteria[9,12,27,35,36] suggestive of VT on 232 patients with SR and fixed intraventricular conduction delay and found that only 5 of the 12 criteria had a specificity of 90%: (i) an Rsr′ or Rr′ QRS complex in V1 in the presence of an RBBB morphology[12] with a specificity of 98%, (ii) a QS, QR or R pattern in V6 in the presence of an RBBB morphology[12] with a specificity of 98%, (iii) any Q in V6 in the presence of an LBBB morphology[35] with a specificity of 92%, (iv) a concordant pattern in all precordial leads[15,18] with a specificity of 100%, and (v) the absence of an RS complex in all precordial leads which was particularly useful for LBBB morphology[27] with a specificity of 91%. The following criteria: QRS duration more than 140 ms,[12] a left axis with RBBB morphology,[12] right superior axis with RBBB morphology,[9,36] monophasic or biphasic R-wave in V1 with RBBB morphology,[12,36] a relation R/S <1 in V6 with RBBB morphology, an R >30 ms in lead V1 or V2 or >60 ms from QRS onset to nadir S with LBBB morphology, a notched downstroke S-wave with LBBB morphology, and an R-to-S interval >100 ms in one precordial lead, had a specificity of 0.43, 0.54, 0.87, 0.80, 0.85, 0.78, 0.66, 0.69, and 0.63, respectively. Finally, artefact that mimics VT, particularly when observed on a rhythm strip, can lead to misdiagnosis. Knight *et al.*[37] surveyed 55 internists, 221 cardiologists, and 490 electrophysiologists with a case simulation that included a two-lead ECG monitor tracing of an artefact simulating a BCT and found that the rhythm strip was misdiagnosed as VT by 94% of the internists, 58% of the cardiologists, and 38% of the electrophysiologists (*Figure 7*).

Limitations of morphological criteria

Conditions like bundle branch or interfascicular reentry tachycardia, fascicular VT, VT exit site close to the His–Purkinje system, pre-excited SVT, and BCT occurring during antiarrhythmic drug

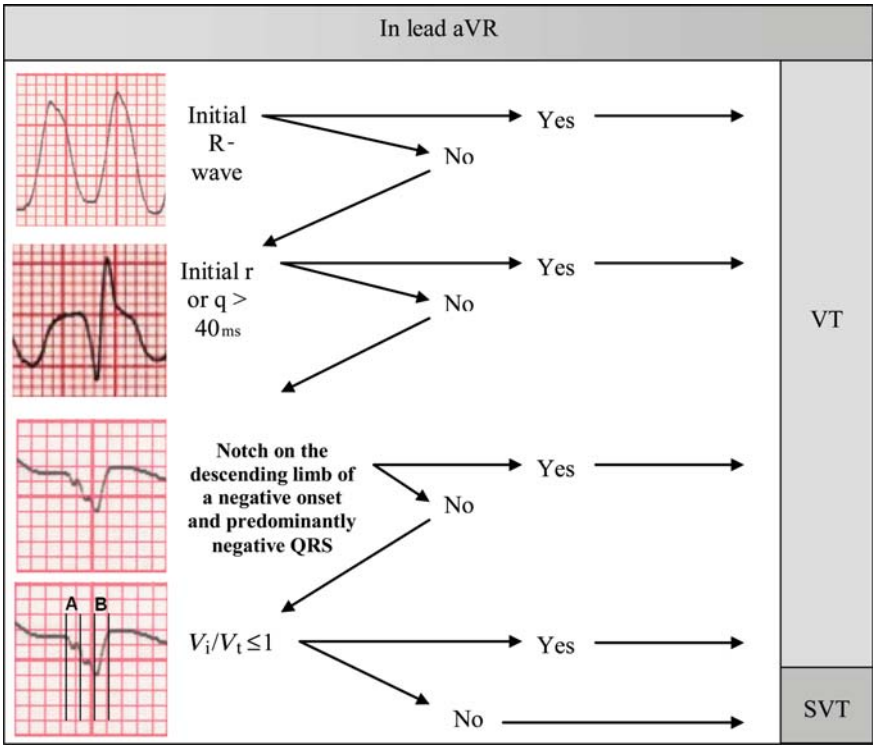

Figure 6 Three hundred and fifty-one VTs and 132 SVTs were included in the comparison. Next to ischaemic VTs and SVT with aberration, pre-existing BBB (144), pre-excited tachycardia (20), idiopathic fascicular VT (11), idiopathic right ventricular outflow tract VT (13), other types of idiopathic VT (13), and patients on class I or III antiarrhythmic drugs (158) were also included (AVR algorithm[29]).

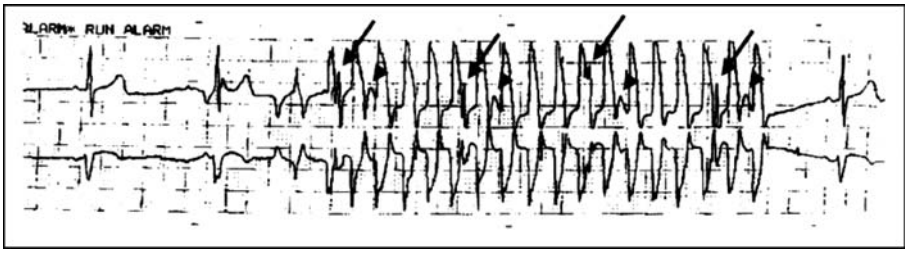

Figure 7 Two-lead ECG monitor tracing of an artefact simulating a BCT. The rhythm strip was misdiagnosed as VT by 58% of the cardiologists surveyed. From Knight *et al.*[37] Reproduced with permission from the publisher.

(AAD) treatment are difficult to diagnose by using the morphological criteria, as most of the studies did not include these patients in the differentiation. In this section, we will discuss some of these conditions.

Broad QRS complex tachycardia occurring during anti-arrhythmic drug treatment

Antiarrhythmic drugs especially those that slow conduction like class Ic drugs may cause proarrhythmia represented by monomorphic VT. On the other hand, class Ic AADs may cause bizarre aberrant conduction during SVT which may mimic VT[38] (*Figure 8*). The QRS complex during aberration is characterized by a QRS duration between 180 and 240 ms and bizarre RBBB with a right or northwest axis, or atypical LBBB with left axis, mimicking VT. The explanation of the occurrence of bizarrely shaped BBB during treatment with class Ic AADs, particularly flecainide, is yet uncertain. A possible explanation is the occurrence of use-dependent conduction delay especially in the myocardium beyond the block.[39] In contrast, conduction is relatively unhampered in areas activated by way of the His–Purkinje system, preserving the initial part of the QRS complex unchanged. That conduction delay due to flecainide is more pronounced in the ventricular myocardium than in the His–Purkinje system has been suggested by animal experiments. In contrast to the situation

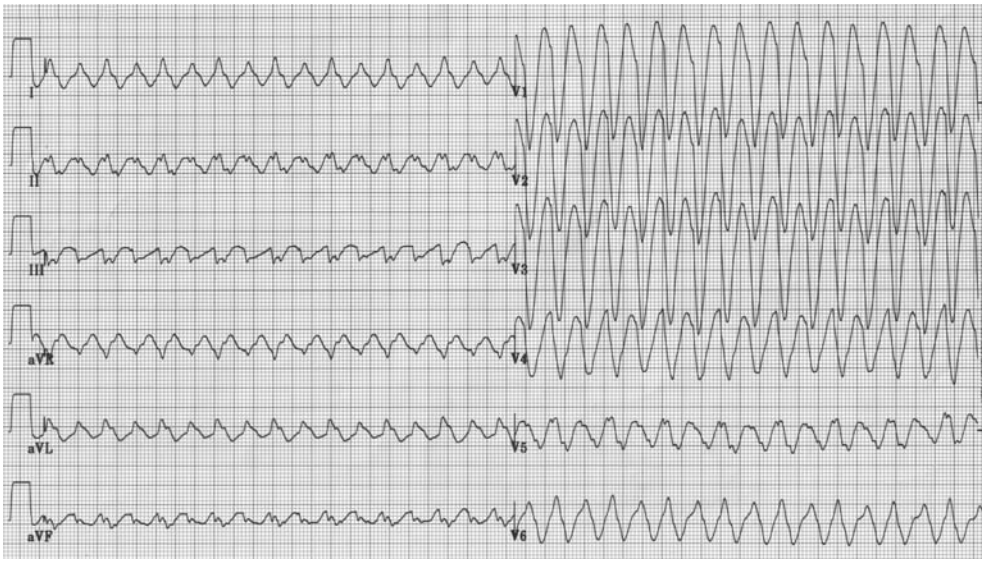

Figure 8 Atrial flutter with one-to-one conduction in a patient on oral flecainide. Note the extreme broad and bizarre QRS complexes mimicking VT. An additional alternating intraventricular conduction delay is also present, most apparent in lead 3, making the correct diagnosis of SVT more complicated.

during a straightforward BBB, this may result in an exaggerated asynchrony of activation of the various parts of the heart, giving rise to a bizarre type of BBB.[38]

Class III AADs, especially pure IKr blockers like dofetilide may also cause atypical aberrant conduction and sequential bilateral BBB which may be easily misdiagnosed as multiple monomorphic VTs.[40,41] This relates to the fact that class III AADs prolong the refractory period in the Purkinje system much more than in the ventricular myocardium. In addition, differential effects within the Purkinje system (left vs. right, distal vs. proximal, left posterior vs. left anterior fascicular region) may cause bizarre QRS complexes, whereas QRS duration is not terribly prolonged. The sequential bilateral block patterns mimicking multiple monomorphic VTs are due to cycle length-dependent changes in refractory periods among bundle branches. Misdiagnosis is enhanced not only by atypical BBB morphologies and the repetitive multimorphology BCT, but also by an atypically long coupling interval before the onset of these BCTs as well as the relatively long cycle length during BCTs (*Figure 9*).

Bundle branch and interfascicular reentry tachycardia

These are uncommon forms of VT usually seen in patients with an acquired heart disease and significant conduction system impairment. The surface ECG in SR characteristically shows intraventricular conduction defects with or without PR interval prolongation. A relatively narrow baseline QRS complex suggests a role of functional conduction delay in the genesis of bundle branch reentry. The QRS morphology during VT is a typical BBB pattern, usually LBBB, and may be identical to that in SR.[42–45] In contrast to VT of myocardial origin, bundle branch reentry with a LBBB pattern characteristically shows rapid intrinsicoid deflection in the right precordial leads, indicating that initial ventricular activation occurs through the specific conduction system.

Interfascicular tachycardia has been less commonly reported. This tachycardia usually has an RBBB morphology. The orientation of the frontal plain axis is variable and may depend on the direction of the reentrant circuit. Anterograde activation over the left anterior fascicle and retrograde through the posterior fascicle would be associated with right-axis deviation and the reversed activation sequence with left-axis deviation.[46]

Fascicular ventricular tachycardia

This relatively narrow QRS complex VT presents usually in young patients. It can be classified into three subtypes: the common originating from the left posterior fascicle with an RBBB configuration and superior axis; the uncommon type originating from the left anterior fascicle with RBBB configuration and right-axis deviation; and the rare type originating in the upper septal fascicle with a narrow QRS complex and a normal axis.[11] The diagnosis of fascicular tachycardia is difficult due to the relatively narrow complexes and the young age of the patients with no evidence of structural heart disease. Capture beats and fusion beats may be present, suggesting the diagnosis of VT rather than SVT. The QRS duration in fascicular tachycardia can vary from 100 to 140 ms. The RS duration in the precordial leads is $<$80 ms, which is in contrast with VT associated with structural heart disease where the RS interval is generally $>$100 ms.[47] Fascicular and bifascicular VT with an alternating focus between the anterior and the posterior fascicle can be seen in patients with digitalis intoxication or Andersen–Tawil syndrome.

Pre-excited supraventricular tachycardia

No morphological differentiation is theoretically possible between VT and SVT with AV conduction over an accessory pathway.[12,35] In

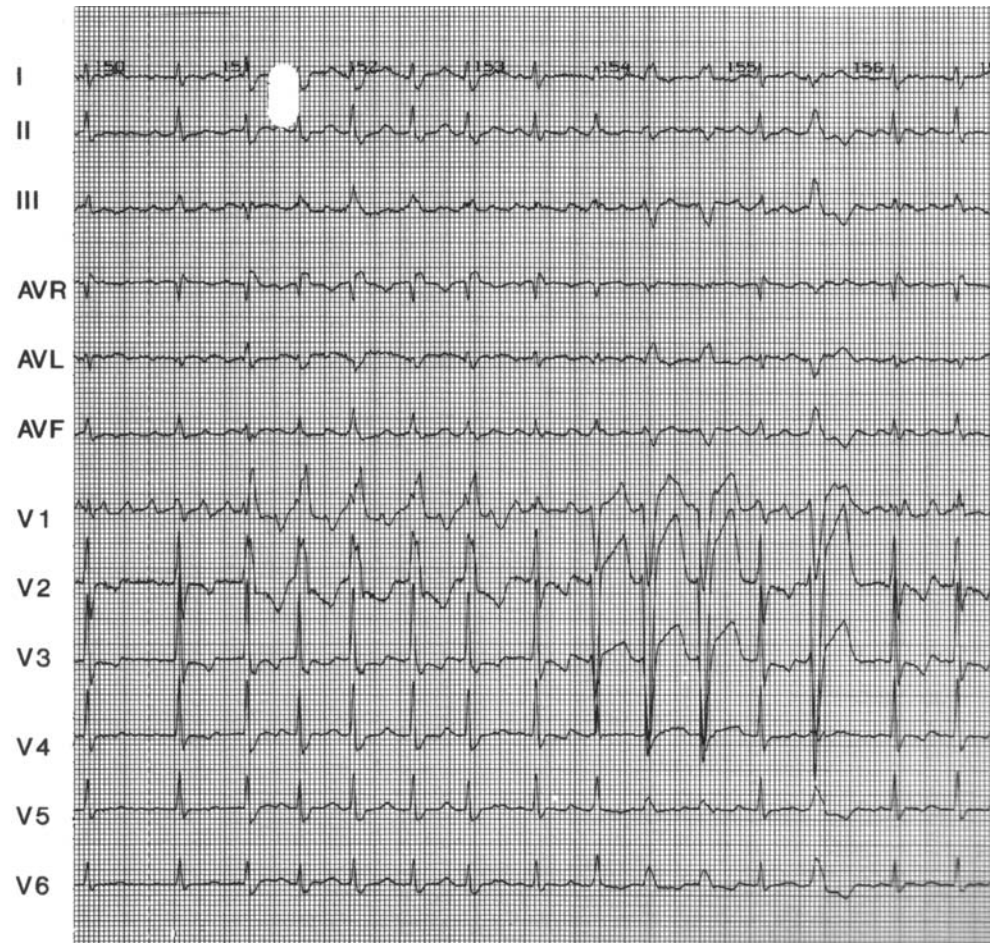

Figure 9 Change from RBBB to LBBB aberrant conduction during AF in a patient receiving dofetilide for attempted chemical cardioversion.

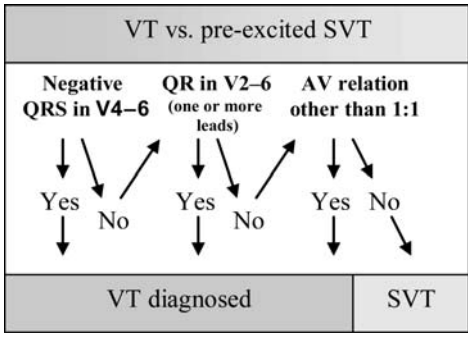

Figure 10 Ventricular tachycardia vs. pre-excited SVT; adapted from Steurer *et al.*[48]

1994, Steurer *et al.*[48] studied 149 VT, 113 with previous MI, and 118 pre-excited SVT to differentiate between them. They designed a stepwise approach with three ECG criteria (*Figure 10*). The criteria favouring VT were: (i) the presence of predominantly negative QRS complex in leads V4–6. (ii) The presence of a QR complex in one or more of the leads V2–6. (iii) Atrioventricular relation different from 1:1 (more QRS complexes than P-waves). The final sensitivity and specificity of these three steps were 75 and 100%, respectively.

Conclusion

Broad complex tachycardia often exhibits, especially at higher frequencies, an indistinct morphology to make a certain diagnosis. Despite all available morphological criteria, BCTs are still misdiagnosed or remain undiagnosed. To achieve a high positive predictive value of more than 95% to identify VT, we prefer combining three clinical criteria; any of the morphological criteria, AV dissociation detected clinically through the ECG or echocardiography, and the past history of myocardial disease (MI, cardiomyopathy, congenital heart disease, and previous surgery). If the diagnosis is still uncertain and typical BBB morphology is missing, VT should be diagnosed by default. Procainamide prolongs the refractory period of the myocardium, the accessory pathway, and the retrograde conduction of the fast AV nodal pathway and therefore, when in doubt, may be given, thus avoiding drugs with potentially harmful effects.[49]

Conflict of interest: all authors have read and approved the manuscript, and no part of this manuscript is being published or under consideration for publication elsewhere. There are no conflicts of interest for any of the authors.

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