6 Wide QRS Tachycardias: Aberrant Conduction or Ventricular Origin?

Mirko Beltrame, Silvia Cesini, Alessandro Maolo, and Cristina Pierandrei

6.1 Case 1

6.1.1 Clinical Contest

A 76-year-old man was referred to the emergency room (ER) complaining of palpitation and fatigue. The patient reported a history of hypertension, dyslipidemia, type 2 diabetes mellitus, and chronic renal failure. The past history included also persistent atrial fibrillation episodes, a previous myocardial infarction 3 years earlier with severe left ventricular dysfunction. A biventricular ICD was implanted as primary indication because of 28% EF.

6.1.2 ECG Analysis

A 12-lead standard ECG was recorded at ER arrival.

There is a wide QRS tachycardia (Fig. 6.1).

Heart rate is 140 bpm. RR intervals are slightly irregular with RR intervals between 420 and 440 ms. Atrial fibrillation is therefore unlikely.

QRS is 160 ms length. To distinguish between supraventricular tachy with aberrant conduction and ventricular tachycardia, we have firstly to check for P waves and possible atrioventricular dissociation.

However P waves are not clearly evident in the trace, and therefore it is hard to recognize a possible atrioventricular dissociation. Fusion beats and capture beats would have been very useful when present in favor of a ventricular tachycardia, but they are not. In the precordial leads, there is no concordance among the QRS complexes that in case would have been a clue possibly suggestive of a ventricular origin of the arrhythmia. We may notice that the first part of the QRS measured from its beginning to the nadir of S wave is superior to 100 ms (VT criterion) in the precordial leads. Also the intrinsicoid deflection (Fig. 6.2), measured from the beginning of the QRS complex to the peak of the R wave, is slow being 60 ms length.

QRS axis is right deviated (+120°) and has a right bundle branch block morphology. In lead V1, a wide R wave pattern is present and in V6 an rS morphology with an R/S ratio inferior to 1. In VR, we can also notice an initial Q wave longer than 40 ms.

The ST segment is markedly downsloped.

M. Beltrame · S. Cesini · A. Maolo (✉)
C. Pierandrei
Clinica di Cardiologia e Aritmologia,
Ospedali Riuniti di Ancona, Università Politecnica delle Marche, Ancona, Italy

A. Capucci (ed.), *New Concepts in ECG Interpretation*,
https://doi.org/10.1007/978-3-319-91677-4_6

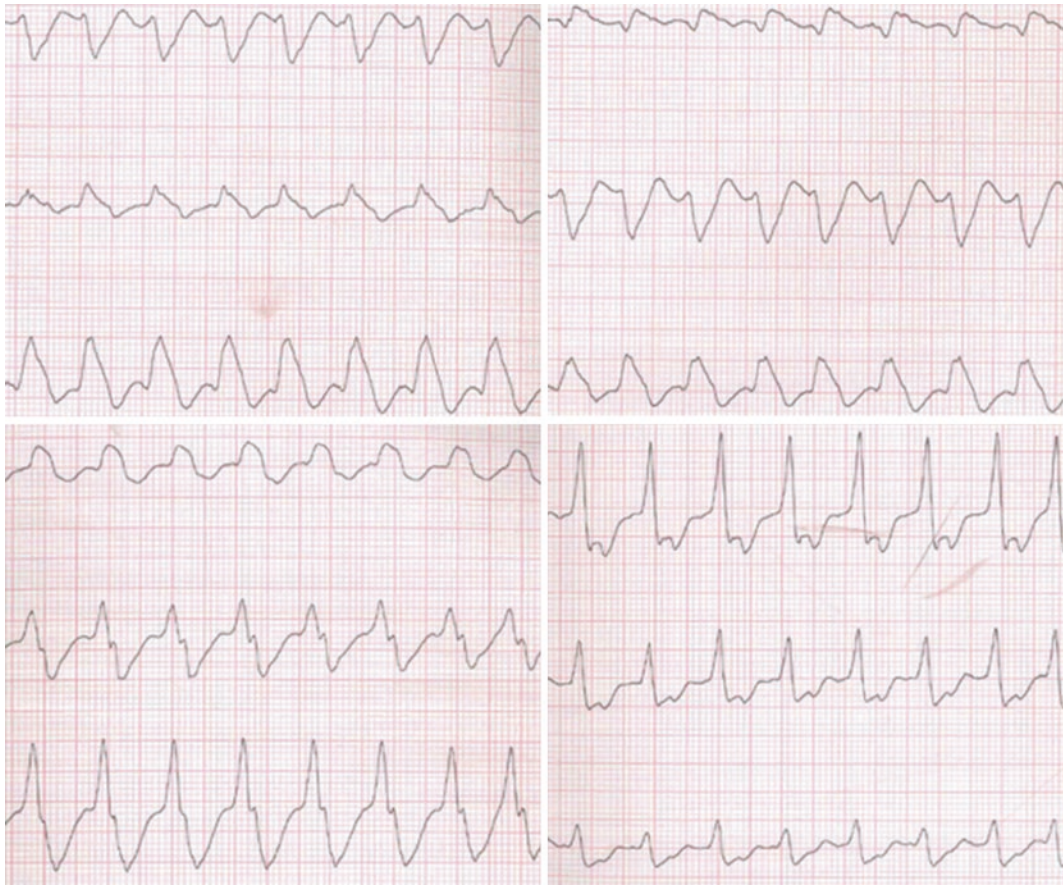

Fig. 6.1 Standard 12-lead electrocardiogram of case 1

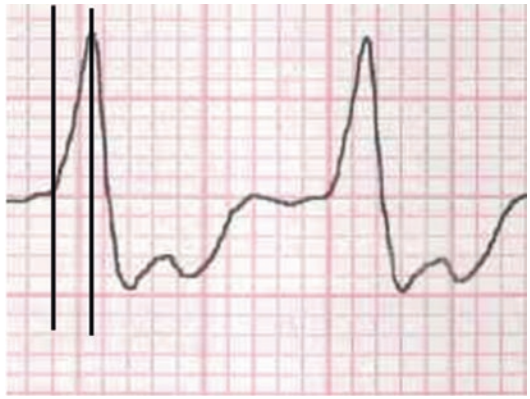

Fig. 6.2 Intrinsicoid deflection in lead V5 on standard 12-lead electrocardiogram of case 1

6.1.3 Diagnostic Summary

This ECG analysis only on the basis of morphologic-wide QRS complex criteria strongly suggests a ventricular tachycardia origin even in absence of a P/QRS clear dissociation.

The endocavitary-recorded ICD tracings (Fig. 6.3) did instead show a dissociation between atrial and ventricular activities. Atrial endocavitary signals are regular with an atrial rate of 115 bpm (RR 520 ms) that is lower than the ventricular rate. The endocavitary recording analysis therefore confirms the ventricular origin of the tachycardia.

This arrhythmia was interrupted by an ICD shock.

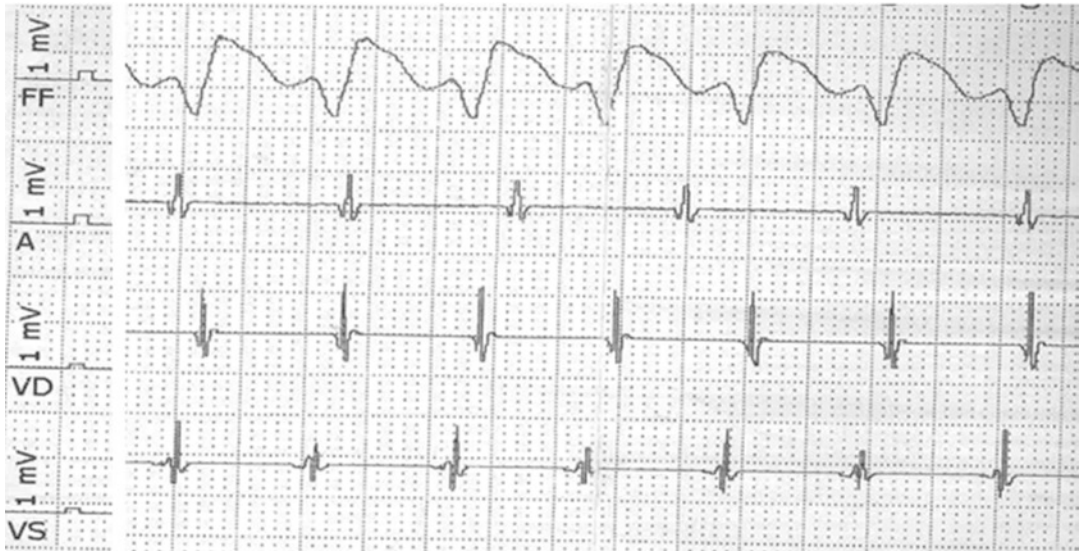

Fig. 6.3 Endocavitary tracing recorded by the ICD in case 1

6.2 Case 2

6.2.1 Clinical Contest

A 73-year-old male was admitted to ER complaining of palpitations and chest pain. The past medical history included arterial hypertension and dyslipidemia.

6.2.2 ECG Analysis

There is (Fig. 6.4) a wide QRS tachycardia.

Heart rate is 165 bpm. RR intervals look very regular (360 ms).

QRS complexes measure 140 ms. The presence of a regular rhythm certainly excludes an atrial fibrillation as a possible tachycardia mechanism.

Atrial activities (P or F waves) are not evident; thus a possible atrioventricular dissociation is not here a useful diagnostic criterion. Fusion beats and capture beats are not present either.

There is a positive concordance of the QRS complexes in the precordial leads, and the intrinsicoid deflection in V5 and V6 leads measures 60 ms. v_i/v_t is <1 (Vereckei criterion).

The QRS axis is right deviated (+110°). It has a right bundle branch block morphology with a wide R wave pattern in both V1 and V6 leads. There is an initial Q wave lasting 60 ms in VR

The ST segment is markedly downsloped.

6.2.3 Diagnosis

The morphologic criteria of QRS suggest therefore the diagnosis of ventricular tachycardia.

The patient was successfully treated with a DC shock of 200 J.

The following sinus rhythm ECG did show an ST segment elevation in the inferior leads.

A coronary arteriography was performed that showed a significant stenosis of the right coronary artery which was successfully treated with PCI and drug-eluting stent implantation.

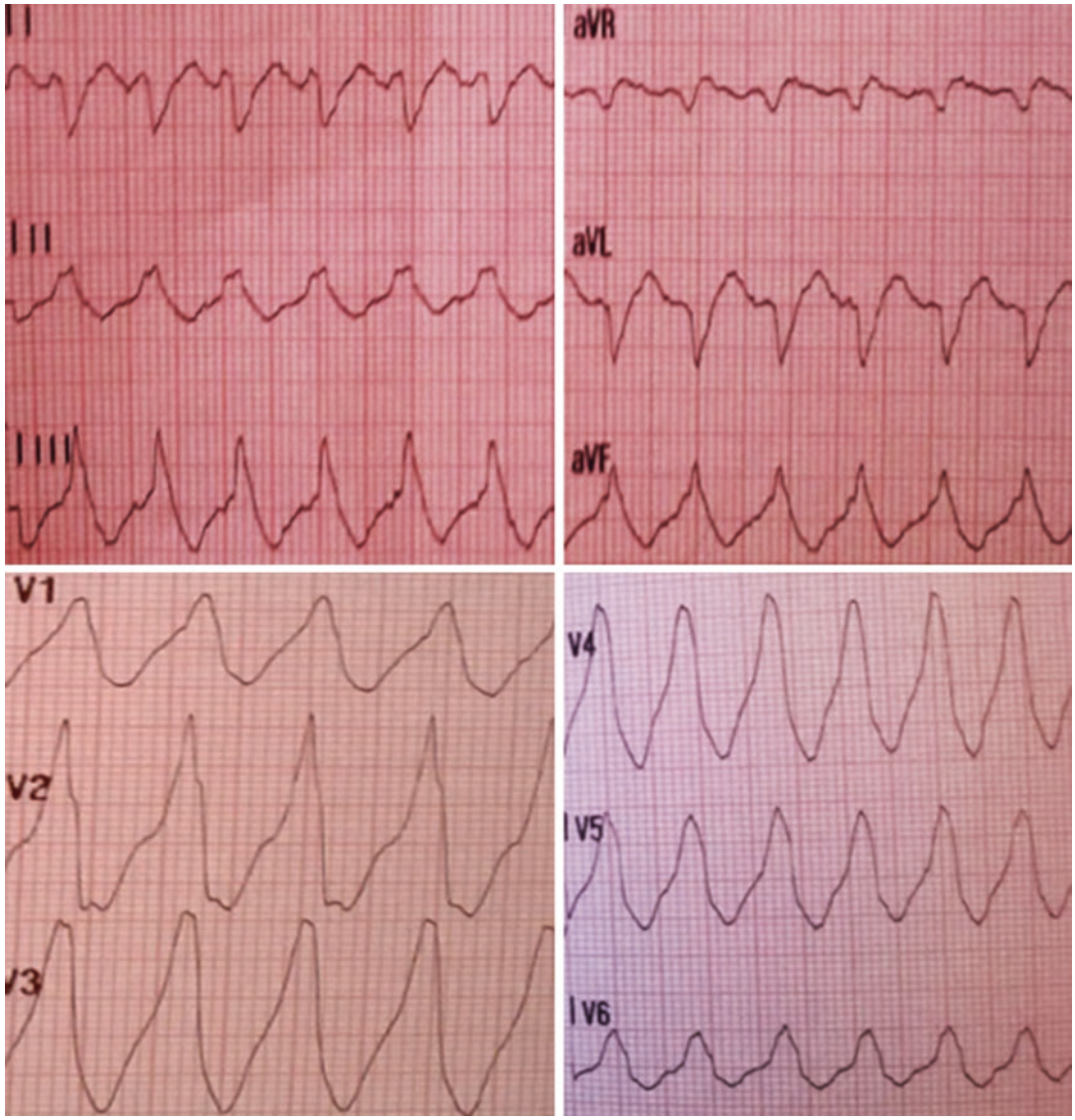

Fig. 6.4 Standard 12-lead electrocardiogram of case 2

6.3 Case 3

6.3.1 Clinical Contest

A 42-year-old male came to ER for persistent palpitations.

The past medical history included the presence of asymptomatic bicuspid aortic valve and ascending aortic dilatation for which a yearly follow-up was scheduled.

A 12-lead ECG was recorded.

6.3.2 ECG Analysis

There is (Fig. 6.5) a wide QRS tachycardia. Heart rate is 210 bpm, RR intervals are regular (280 ms), and QRS duration is 160 ms. The presence of a regular rhythm excludes the hypothesis of atrial fibrillation, and the heart rate is faster than in a classical 2:1 atrial flutter.

Not any atrial activity is evident, and we cannot ascertain any possible atrial ventricular strict relation. There are no fusion and capture beats.

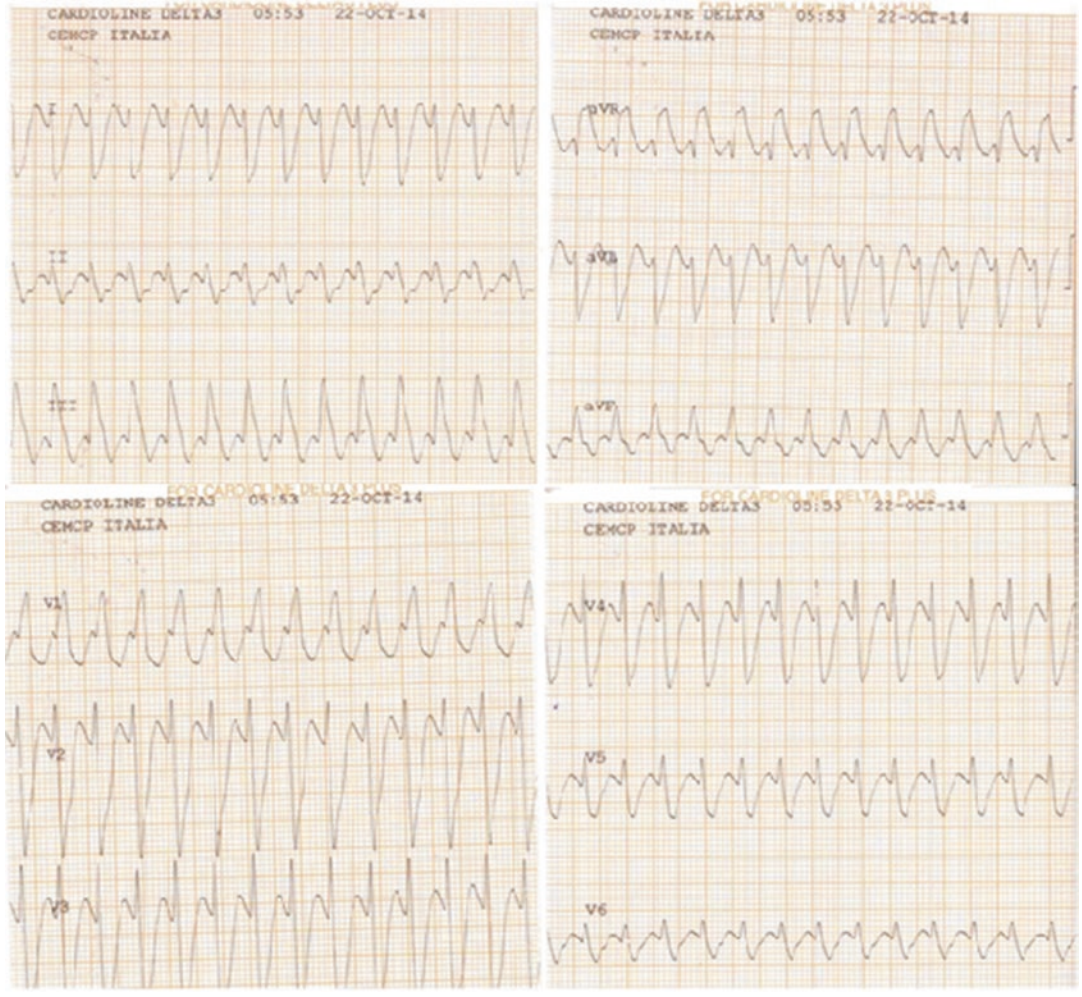

Fig. 6.5 Standard 12-lead electrocardiogram of case 3

Morphological criteria: no QRS concordance in the precordial leads. Time from QRS onset to S nadir in precordial leads is 80 ms. The intrinsicoid deflection in leads II, V4, V5, and V6 measures 35 ms. v_i/v_t is >1 (Vereckei criterion).

The QRS axis is right deviated (+160°). A right bundle branch block morphology is evident with an rR′ wave pattern in V1 and an rS morphology with an R/S ratio inferior to 1 in V6.

6.3.3 Diagnostic Conclusion

By considering the morphologic features, intrinsicoid deflection's characteristics, and Brugada and Vereckei algorithms, this is a supraventricular tachycardia with aberrant atrioventricular conduction.

The arrhythmia ended up spontaneously, and the sinus rhythm ECG did not show any conduction abnormality.

The patient underwent an electrophysiological study that wasn't able to induce any type of tachycardia, neither supraventricular nor ventricular. Nevertheless, during the incremental atrial stimulation up to 220/min, an aberrant conduction with right bundle brunch morphology and right axis deviation appeared with a QRS superimposable to the tachycardia's one.

Supraventricular tachycardia with aberrant atrioventricular conduction remains the most likely diagnosis.

6.4 Case 4

6.4.1 Clinical Context

A 68-year-old woman with past medical history of paroxysmal atrial fibrillation and arterial hypertension underwent cardiac surgery for ascending aorta replacement. At the 5th day after surgery, the patient complained of palpitations and dyspnea.

6.4.2 ECG Analysis

A 12-lead standard ECG was recorded.

There is (Fig. 6.6) a wide QRS tachycardia. Heart rate is 185 bpm, RR intervals are regular (320 ms), and QRS length is 120 ms. The rhythm is regular, so the hypothesis of atrial fibrillation is unlikely. Is a VT or a SVT with aberrant conduction?

There are visible notches inside the ascending branch of T waves in the inferior leads, possibly referable to P waves (or F wave). These P waves could be the result of a retrograde conduction or coming from atria (atrial tachycardia/flutter 1:1 or 2:1). P-wave axis is not clearly definable; however an atrioventricular dissociation can be excluded.

Fusion and capture beats are not present.

QRS complexes in the precordial leads are not concordant. Time from QRS onset to the nadir of S wave in V5–V6 is 80 ms. The R-peak time (intrinsicoid deflection) in leads II and V6 is 30 ms. In VR, there is an initial Q wave lasting about 40 ms. v_i/v_t is >1 (Vereckei criteria).

The QRS axis is extremely left deviated (−135°). A right bundle branch block morphology is evident with a wide R wave pattern in V1 and an rS morphology with an R/S ratio inferior to 1 in V6, both peculiar of ventricular tachycardia.

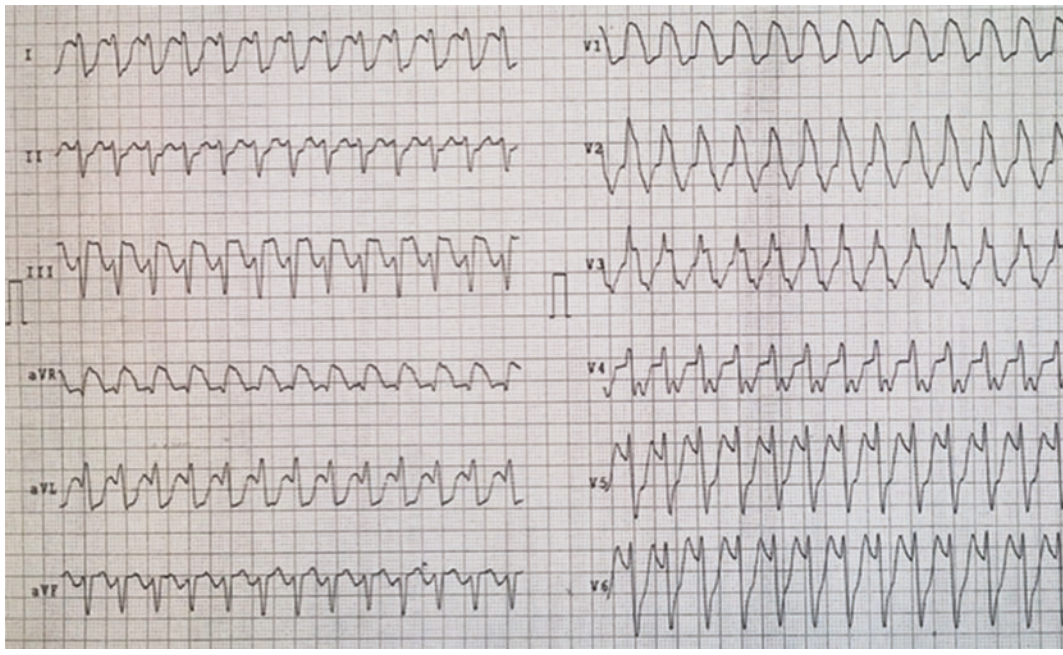

Fig. 6.6 First standard 12-lead electrocardiogram recorded for case 4

6.4.3 Diagnosis

The diagnosis is challenging because the classical criteria are discordant.

The tachycardia ended after a 5 mg metoprolol intravenous injection.

Some days later, the patients moved to our clinic where he had recurrence of the same symptoms. The following ECG was recorded (Fig. 6.7).

The ECG shows a narrow QRS complexes tachycardia. The heart rate is 150–130 beats/min. RR interval is not regular. F waves with an atrial rate of 280 bpm are visible. The F axis is oriented upward (−90°). QRS length is 80 ms, and no intraventricular conduction abnormality is present. T waves are negative in left precordial leads V4-V5-V6.

RR irregularities and F waves with a rate of 280/min suggest a common atrial flutter with a variable AV conduction (2:1; 3:1).

By going back to the previous medical records, it comes out that the patient was assuming flecainide 200 mg/day at the time of the initial tachycardia. The drug was then discontinued.

A posteriori, the first ECG was interpreted as atrial flutter with 1:1 AV conduction and aberrant QRS complexes. In the second ECG, there is a faster atrial rate because of lacking of the slowing atrial conduction influence of flecainide.

The patient underwent an electrophysiological study in which no ventricular arrhythmias were induced. However an atypical atrial flutter/atrial tachycardia with an atrial rate of 280/min was easily induced.

The class 1C drug flecainide is a sodium channel blocker which slows phase 0 of action potential, delaying conduction, with minimal effect on QT and with a mainly use-dependent effect.

Flecainide also prolongs the intra-atrial conduction and consequently reduce atrial rate. The decrease in atrial flutter rate to range of 180–200 beats/min can favor a 1:1 ventricular response.

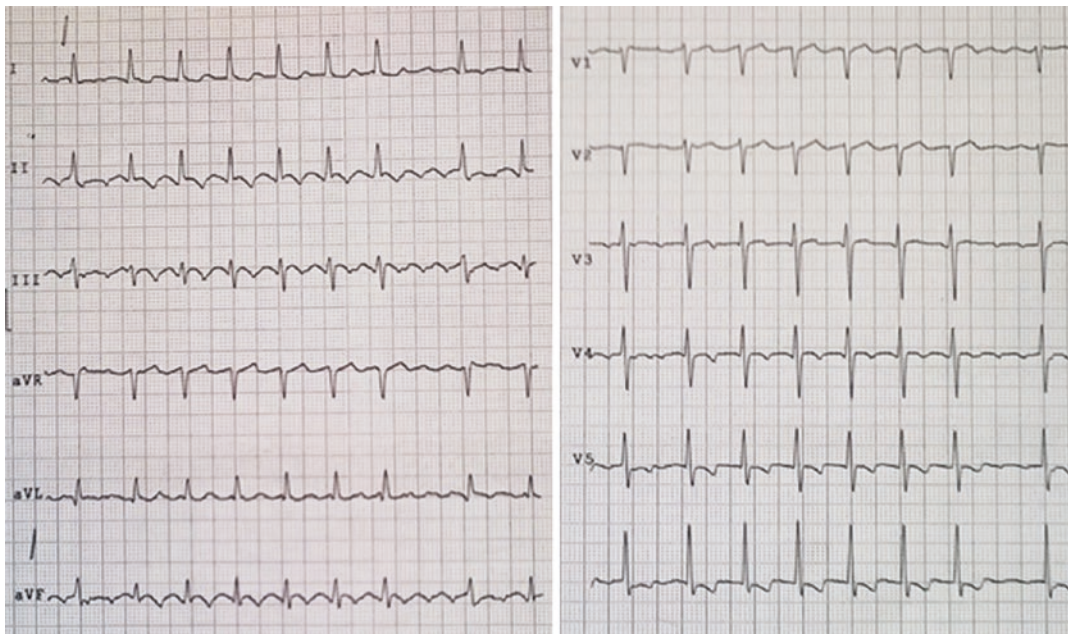

Fig. 6.7 Second standard 12-lead electrocardiogram recorded for case 4

This tachycardia effect happens in 0.5% of the treated patients during a chronic therapy.

6.4.4 From ECG to Pathology

Wide QRS complex tachycardias (WTC) are usually characterized by HR >100 bpm and QRS ≥120 ms duration.

Ventricular tachycardia (VT) is the most common cause (80% of cases); however a wide QRS could be supraventricular (SVT) and conducted with fixed or functional bundle branch block (BBB) (15–25% of WTC cases). The aberrancy may be favored by drug influences, electrolyte imbalances, or preexcitation (1–5%) [1].

A history of structural heart disease may be in favor of TV (>95% chances); however about 10% of patients with VT have no structural heart disease [1].

There are several electrocardiographic criteria for the differential diagnosis that are reported in Table 6.1 [6–10].

6.4.5 R Wave Peak Time (Intrinsicoid Deflection) Criterion

Intrinsicoid deflection, or R wave peak time, represents the early phase of ventricular depolarization and is defined as the time period from the onset of the QRS complex to the peak of the R wave (Figs. 6.8 and 6.9) [2–5].

The depolarization impulse travels through the normal His-Purkinje system faster than in the contractile myocardium and may be one reason why initiation of ventricular depolarization in VT

Table 6.1 Summary of the traditional criteria for the differential diagnosis of wide QRS tachycardias (WCT)

Atrioventricular dissociation	– No correlation between atrial and ventricular activity – It is diagnostic – 20–50% of VT	– P waves – Notches and irregularities repeated cyclically – 30% of VTs have 1:1 retrograde VA conduction
Capture beats	– Occur when a sinoatrial beat transiently captures the ventricles – It's an indirect sign of AV dissociation	– Narrow QRS complex usually close to normal duration
Fusion beats	– Occur when a sinoatrial beat and a ventricular beat coincide temporally in depolarizing the ventricles – It's an indirect sign of AV dissociation	– Hybrid QRS complex
Precordial concordance	– QRS complexes have the same orientation in all precordial leads	– Orientation of QRS complexes[a]
Morphology	– VT is suspected when QRS complexes do not resemble typical bundle branch block	RBBB morphology: – Wide R wave (V1) – qR or Rs (V1) – RSr′ (V1) – rS complex (V6) LBBB morphology: – Wide R wave (>30 ms) (V1 or V2) – A slurred or notched downstroke of the S wave (<60 ms) (V1 or V2) – Q wave, QS, or QR complexes (V6) [b] [c]

[a]QRS complexes have to be all positive or all negative from V1 to V6. A negative concordance has a higher specificity, while a positive concordance may also be caused by preexcited SVT through a left posterior accessory pathway

[b]In brackets is reported where to look for the morphologic features

[c]QR (but nor QS) complexes in any lead except aVR during WCT, especially when present in the same leads also in sinus rhythm; indicate a remote myocardial infarction and therefore are more suggestive for VT

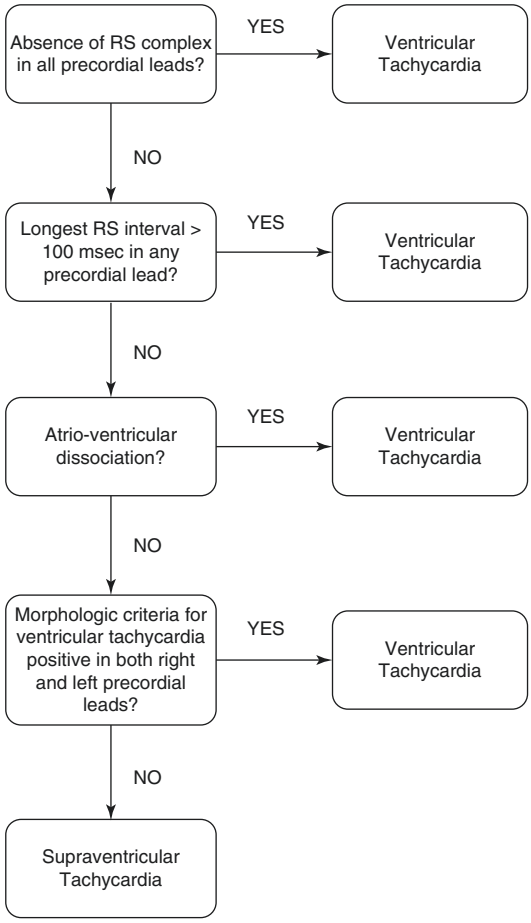

Fig. 6.8 Algorithm that summarizes Brugada's criteria for differential diagnosis of wide QRS tachycardias

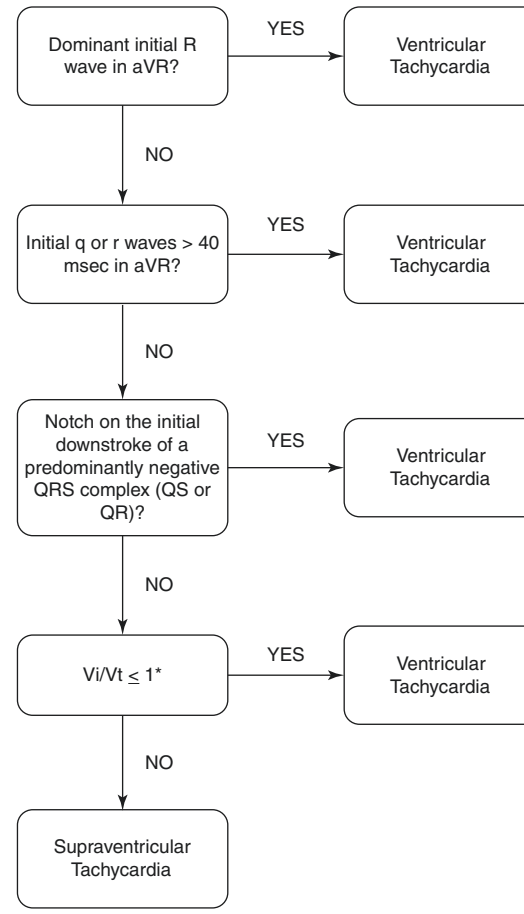

Fig. 6.9 Algorithm that summarizes Vereckei's criteria for differential diagnosis of wide QRS tachycardias. This algorithm adopts the conventional criteria of VA dissociation together with the morphologic analysis of the QRS complex in aVR. *v_i: total amplitude of the QRS in the first 40 ms; v_t, total amplitude of the QRS in the last 40 ms. During WCT due to VT, an initial slower muscle-to-muscle spread of activation occurs until the impulse reaches the His-Purkinje system, after which the rest of the ventricular muscle is more rapidly activated; thus, the $v_i/v_t \leq 1$ during VT

is longer than normal. The intrinsicoid deflection remains narrow or less wide during aberrance.

Pava et al. proposed that the deflection intrinsicoid in lead II (measuring the interval from the QRS onset to the peak of the first positive or negative wave) when ≥50 ms suggested VT and when <50 ms suggested aberrant conduction, with a high sensitivity (93.2%), specificity (99.3%), PPV (98.2%), and negative predictive values (NPV) (93.3%) for VT diagnosis [9].

Capucci et al. used a specific intracardiac ECG (iECG) to distinguish between an idioventricular activity (IVA) and an atrioventricular conduction (AVC) with wide QRS confirming that a prominent early iQRS velocity is generally observed with AVC and not with IVA [10].

Exception may occur depending on the underlying heart disease (MI scar, ventricular remodeling, or drug treatment) and a fascicular origin of the arrhythmia.

A precise evaluation of the initial ventricular activation represents therefore a strategic diagnostic criterion to differentiate any wide complex tachycardia. Indeed, the intrinsicoid deflection is an easy parameter to measure and therefore very much useful.

We believe that the integration of this parameter with the other reported ECG criteria (table) together with the patient history are of primary importance for a right etiologic definition of the tachycardia.

References

1. Vereckei A. Current algorithms for the diagnosis of wide QRS complex tachycardias. Curr Cardiol Rev. 2014;10(3):262–76.
2. Oreto G, Luzza F, Satullo G, Donato A, Carbone V, Calabrò MP. Wide QRS complex tachycardia: an old and new problem. G Ital Cardiol. 2009;10(9):580–95.
3. Barbarossa A, et al. Wide QRS complex tachycardias. In: Capucci A, editor. Clinical cases in cardiology. Cham: Springer; 2015.
4. De Ponti R, Bagliani G, Padeletti L, Natale A. General approach to a wide QRS complex. Card Electrophysiol Clin. 2017;9(3):461–85.
5. Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM. Application of a new algorithm in the differential diagnosis of wide QRS complex tachycardia. Eur Heart J. 2007;28:589–600.
6. Bauernfeind RA, et al. Retrograde block during dual pathway atrioventricular nodal reentrant paroxysmal tachycardia. Am J Cardiol. 1978;42:499.
7. Brugada P, Brugada J, Mont L, Smeets J, Andries EW. A new approach to the differential diagnosis of a regular tachycardia with a wide QRS complex. Circulation. 1991;83:1649–59.
8. Vereckei A, Duray G, Szénási G, Altemose GT, Miller JM. A new algorithm using only lead aVR for the differential diagnosis of wide QRS complex tachycardia. Heart Rhythm. 2008;5:89–98.
9. Pava LF, Parafán P, Badiel M, et al. R-wave peak time at DII: a new criterion to differentiate between wide complex QRS tachycardias. Heart Rhythm. 2010;7:922–6.
10. Capucci A, et al. The intracardiac ECG dynamics allows discriminating idioventricular activity from atrio-ventricular conduction with wide QRS. Europace. 2017;19(Supplement 3):iii88.