Wide QRS Complex Tachycardias

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18.1 Case Report

A 72-year-old man was presented to the ER for an episode of palpitations, fatigue, and dyspnea lasting more than 6 h.

Medical History and Cardiovascular Risk Factors

- Arterial hypertension.
- Hypercholesterolemia (LDL 98 mg/dl on drug therapy).
- Stage 3 chronic kidney disease (GFR 40 ml/min).
- Previous inferior myocardial infarction (STEMI) 9 years ago with consequent coronary artery bypass graft (CABG) surgery: sequential venous graft (VG) between ascending aorta and right coronary artery (RC) and first left marginal artery (LMA1) and VG between ascending aorta and left anterior descending artery (LAD).
- Unstable angina (UA) 3 years ago. The patient was subjected to coronary arteriography that documented a total occlusion of the sequential VG to RC and LMA 1 and a stenosis of 95 % of the VG between aorta and LAD. For this reason, another CABG surgery consisting of left internal mammary artery (LIMA) to LAD was performed.
- Benign prostatic hypertrophy.

Allergies

None

Social History

- He never smoked.
- He never used illicit drugs.
- He has a sedentary lifestyle.

Home medications: pantoprazole 20 mg at 8:00 AM, ramipril 2.5 mg at 8:00 AM and 2.5 mg at 8:00 PM, metoprolol 50 mg at 8:00 AM and 50 mg at 8:00 PM, aspirin 100 mg at 12:00 AM, furosemide 50 mg at 8:00 AM and 25 mg at 4:00 PM, simvastatin 40 mg at 10:00 PM, tamsulosin 0.4 mg at 10:00 PM

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A. Capucci (ed.), *Clinical Cases in Cardiology: A Guide to Learning and Practice*,
DOI 10.1007/978-3-319-19926-9_18

Vital Signs at ER Entrance

- Temperature: 36.7 °C
- Heart rate: 120 bpm
- Blood pressure: 105/70 mmHg
- Respiratory rate: 16 breaths/min
- Oxygen saturation while breathing in ambient air: 91 %

Physical Examination

- General: fatigued, sweaty, alert, awake, and oriented
- Head, eye, ear, nose, and throat: normocephalic, atraumatic, mucous membranes moist, extraocular muscles intact, pupils equally round and reactive to light and accommodation bilaterally, bilateral tympanic membrane intact, bilateral sclerae anicteric, no conjunctival injection
- Neck: supple, no jugular venous distention, no lymphadenopathy, no carotid bruit
- Cardiovascular: regular rhythm, tachycardia rate, S1 and S2 are normal, holosystolic murmur III/VI on Levine scale at the apex and at the anterior axillary area
- Lungs: rales only at the pulmonary bases. No rhonchi or wheezes, no egophony, no alterations in tactile fremitus, normal percussion in remaining areas
- Abdomen: flat, no pulsatile masses, normal bowel sounds in all four quadrants, no high-pitched or tinkling sounds, resonant to percussion, soft, non-distended/non-tender, no rebound or guarding, no costovertebral angle tenderness, no hepatosplenomegaly
- Extremities: no cyanosis or clubbing, mild edema at both ankles
- Neurologic: cranial nerves I through XII intact, no focal deficit
- Psychiatric: normal affect, no hallucinations and normal speech
- Skin: intact, sweaty, no rushes, no lesion

Routine Laboratory Tests

- Complete blood count: normal (hemoglobin 12.9 g/dl)
- Inflammatory markers: ESR 46 mm/h, CRP 1.3 mg/dl
- Hepatic function (GOT, GPT, γ-GT, ALP, total bilirubin, direct and indirect): normal
- Renal function: creatinine 1.71 mg/dl, BUN 25.2 mg/dl
- Electrolytes (Na^+, K^+, Ca^{++}, Mg^{++}, Cl^-): normal
- Thyroid function (TSH, fT3, fT4): normal
- Fasting blood glucose: 131 mg/dl
- HbA1C: 7.0 % (53 mmol/mol)
- Hs-TnT: 12 pg/ml (highest value)

ECG

A standard 12-lead ECG, at rest, was performed. The ECG (Fig. 18.1) showed a wide QRS tachycardia. Heart rate was 120 bpm (RR 500 ms). QRS axis was about −160°. QRS duration was 200 ms. The QRS morphology was right bundle branch block type (Rr' in lead V1). P waves were not clearly visible. ST segment was not assessable.

What are the possible types of wide QRS complex tachycardia?

- Ventricular tachycardia (VT)
- Supraventricular tachycardia with aberrant AV conduction
  - Atrial fibrillation
  - Atrial flutter
  - Focal atrial tachycardia
  - Atrioventricular reciprocating tachycardia (ortho- or antidromic)
  - Atrioventricular nodal reciprocating tachycardia
- Pre-excited tachycardia (with anterograde conduction on the accessory pathway)
  - Atrial fibrillation
  - Atrial flutter
  - Focal atrial tachycardia

The presence of a regular rhythm excludes the hypothesis of atrial fibrillation and atrial flutter/tachycardia with variable AV conduction.

In the present ECG, there are not any visible P waves (or F wave); the P wave could be inside the terminal part of the ventricular complex or inside the T wave. So, a clear atrioventricular dissociation (suggestive for VT) is not demonstrable.

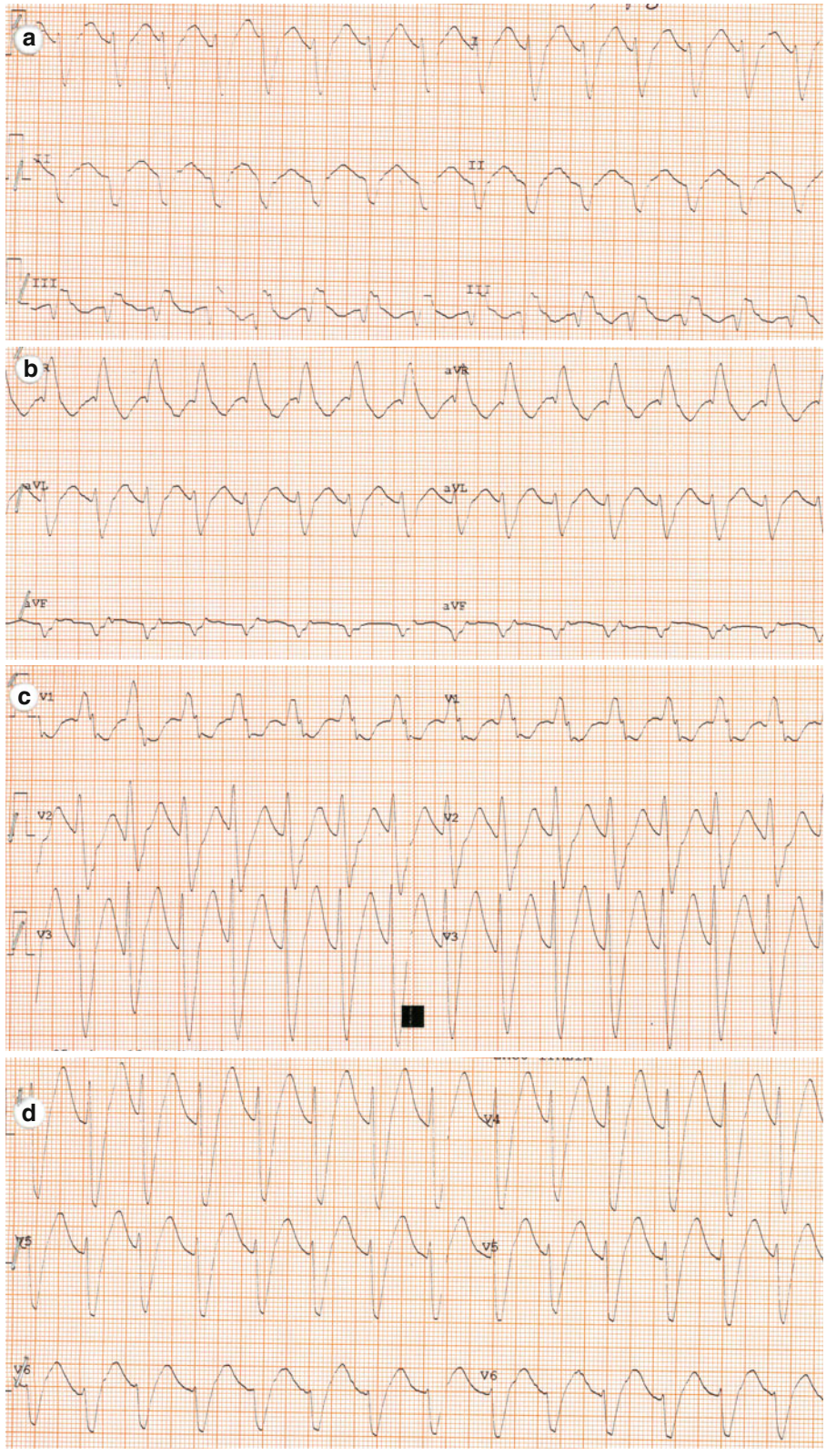

Fig. 18.1 (**a**–**d**) Rest ECG showing a wide complex tachycardia

Moreover, the heart rate (120 bpm) is too slow for a 2:1 atrial flutter. Even if P wave is not visible, hypothesis of a sinus tachycardia is unlikely because there were not physiological reasons for having an elevated rest heart rate.

There are not fusion or capture beats (diagnostic for VT).

There was hemodynamic stability, and therefore we performed carotid sinus massage that did not modify the tachycardia cycle. The QRS complexes in the precordial leads were not concordant: V1 positive and negative from V2 to V6 (concordance is high suggestive of ectopic ventricular beats, if negative specifically for VT). We measured the distance from the onset of QRS to the nadir of S wave in a precordial lead (we chose V2), and it is about 120 ms (when this interval is more or equal to 100 ms, you should consider VT). Finally, our ECG analysis ends with QRS morphological criteria: we are in front of a RBBB-type wide QRS tachycardia. In V1 the QRS complex morphology is Rr' and in V6 rS. This morphological pattern is suggestive for VT. The QRS axis at −160° and the QRS duration of 200 ms are other adjuvant criteria for VT.

Owing these criteria, we postulated the diagnosis of ventricular tachycardia.

The patient, after sedation with midazolam 5 mg IV, was successfully treated with electric cardioversion (DC shock 200 J) with an immediate return to sinus rhythm (Fig. 18.2).

At sinus rhythm, the QRS complex had a complete different morphology. The signs of the previous inferior MI were visible.

Admission to the Arrhythmology Department

The patient was then admitted to our cardiology and arrhythmology department to seek the cause of arrhythmia.

Electrolytec imbalance, hyper- or hypothyroidism, and coronary acute syndrome were excluded.

ECG: sinus rhythm, normal AV conduction, QRS axis +30°, Q wave in inferior leads as a previous inferior myocardial infarction, normal repolarization.

At echocardiography: mild dilatation of the left atrium. Moderate dilatation of the left ventricle (iEDV 92 ml/m^2). Severe left ventricular systolic dysfunction (EF 40 %). Akinetic and thinned inferior wall of the left ventricle. Normal dimensions of the right ventricular dimension and systolic function (TAPSE 19 mm). II grade diastolic dysfunction. Moderate mitral regurgitation. Mild tricuspid regurgitation with normal pulmonary arterial pressure. No pericardial effusion.

There were not any clues attributable to accessory AV pathways or to channelopathies (e.g., Brugada syndrome or long and short QT syndrome). Also, cardiomyopathies like hypertrophic cardiomyopathy, arrhythmogenic right ventricular dysplasia, or non-compacted myocardium were excluded. Finally, the presence of an inferior wall necrosis was confirmed.

Because of the low EF unknown in previous controls, we repeated a coronary arteriography that showed occlusion of VG between RC and LMA1, occlusion of VG between aorta artery and LAD, and a good flow on LIMA to LAD.

Amiodarone therapy was started (400 mg/die for the 1st week then 200 mg/die) to prevent recurrences. During hospitalization at ECG monitor, a lot of premature ventricular beats (PVBs), couple of PVBs, non-sustained VT (nsVT), and another VT (which required cardioversion with DC shock) were recorded. All these ventricular arrhythmias had the same QRS morphology of the first VT episode.

An invasive electrophysiological study (EPS) was performed and documented:

> […] easy induction by programmed ventricular pacing of a ventricular tachycardia coming from the inferior wall of left ventricle. A catheter ablation of the site of origin of ventricular tachycardia was performed successfully with no more induction of arrhythmia after programmed ventricular pacing of the area. […]

The final diagnosis was "ventricular tachycardia starting from the inferior wall of the left ventricle due to macro-reentrant circuits secondary to a myocardial scar."

VT did not return during hospitalization.

One month after discharge, we performed a 7-day-long ECG-Holter monitoring that did not document any VT recurrence.

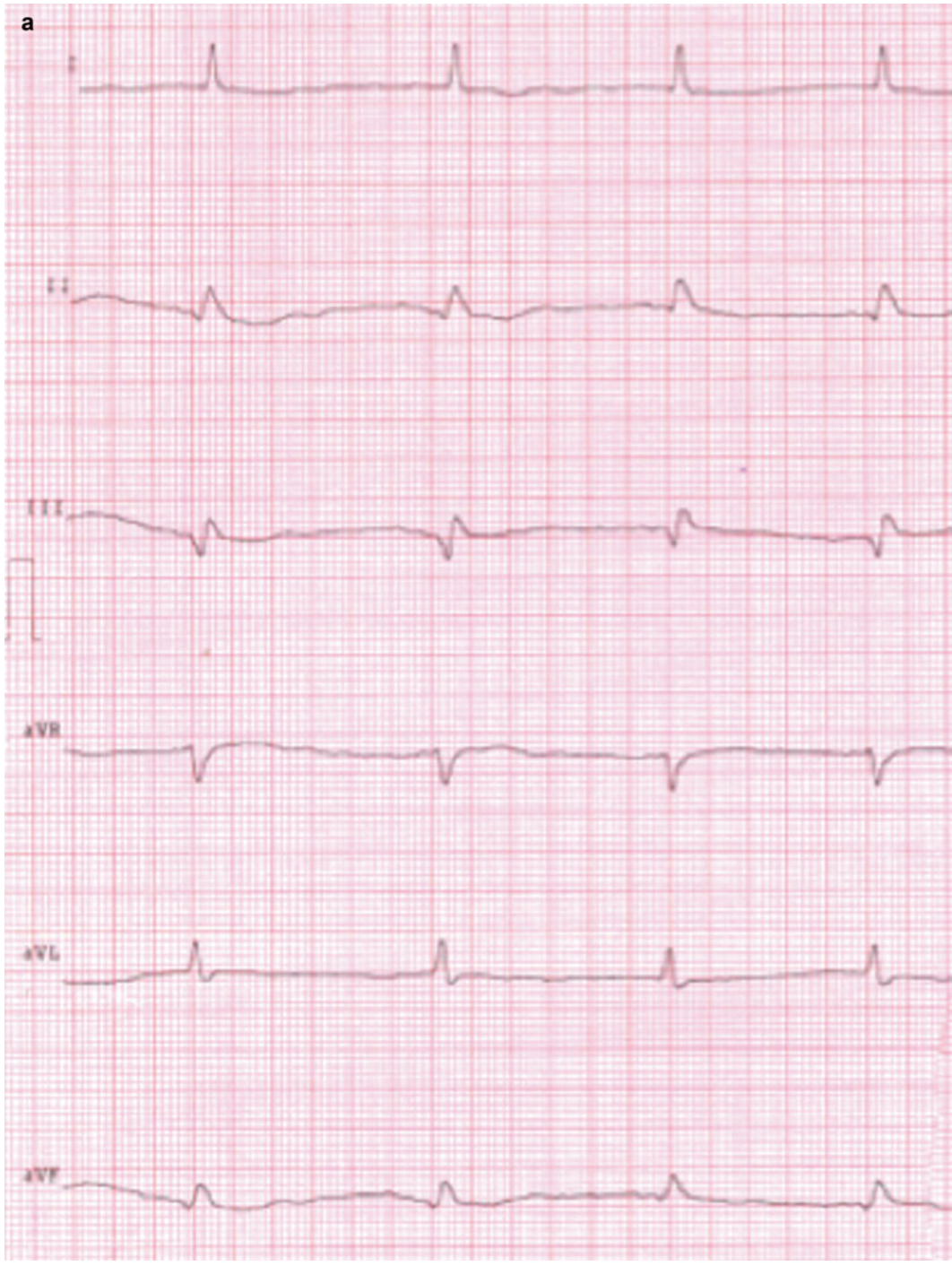

Fig. 18.2 (**a**, **b**) ECG performed after electrical cardioversion

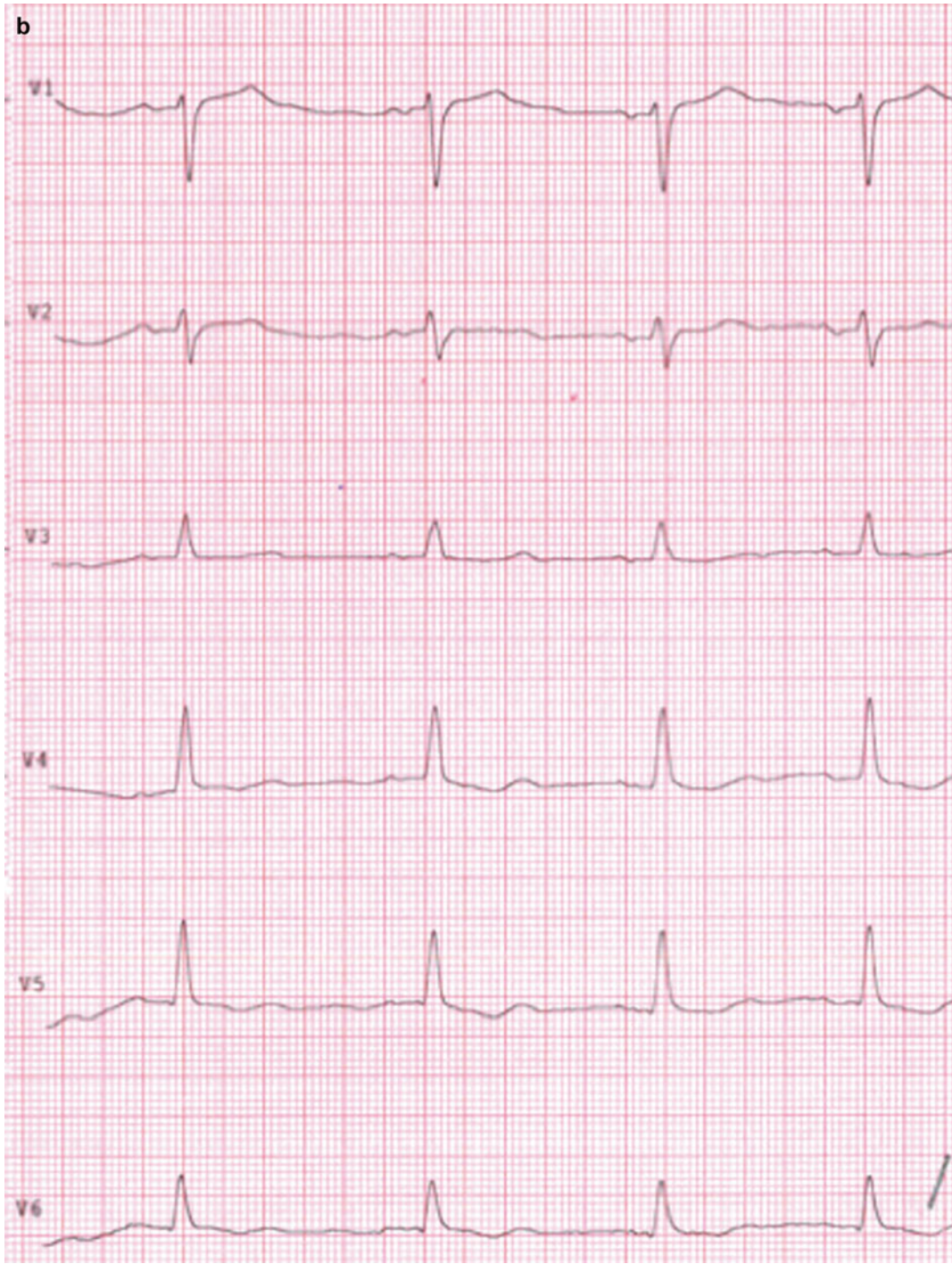

Fig. 18.2 (continued)

18.2 Ventricular Tachycardia

Definition

VT is a tachycardia that rises from the ventricles and lasts at least 3 beats. Non-sustained VT (nsVT) is a VT that lasts less than 30 s; contrarily, a VT that lasts longer than 30 s is called sustained ventricular tachycardia (sVT).

Epidemiology and Etiology

VTs are associated with a structural heart disease, but can occur also in its absence. Ischemic heart disease is the most common cause of VT. During the acute phase of ischemia, a polymorphic VT or a ventricular fibrillation (VF) is the principal cause of sudden cardiac death (SCD).

Monomorphic VT is usually a consequence of a myocardial scar zone that became a reentrant substrate in patients with structural heart disease. The scars can derive from an old myocardial infarction but also from nonischemic cardiomyopathies, including idiopathic dilated cardiomyopathy, hypertrophic cardiomyopathy (HCM), infiltrative heart disease (e.g., amyloidosis), and arrhythmogenic right ventricular dysplasia (ARVD).

Moreover, some genetic syndromes (channelopathies) are mainly associated with polymorphic ventricular arrhythmias: Brugada syndrome, long and short QT syndromes, and catecholaminergic polymorphic ventricular tachycardia (see specific chapters).

Finally, a VT that occurs in the absence of structural heart disease and genetic conditions is referred to as idiopathic VT. This form of VTs usually originates from the right ventricular outflow tract (RVOT) and is associated with good prognosis [1].

ECG Diagnostic Criteria

In the presence of a tachyarrhythmia with wide QRS, a possible diagnosis of ventricular tachycardia (VT) has to be taken into account. The mainstay of differential diagnosis is the ECG itself, but there are other elements that may help the diagnosis.

ECG in Wide QRS Tachycardias

Wide QRS complex tachycardias are one of the most challenging questions of ECG. A regular wide QRS complex tachycardia may represent one of the following rhythms:

- Aberrant atrioventricular conduction of supraventricular tachycardia (SVT)
- Ventricular tachycardia (VT)
- Preexisting right (RBBB) or left (LBBB) bundle branch block with SVT
- Anterograde conduction over the bypass tract of atrioventricular connection in patients with Wolff-Parkinson-White (WPW) syndrome

Irregular wide QRS complex tachycardia may represent one of the following rhythms:

- Atrial fibrillation with aberrant ventricular conduction
- Atrial fibrillation with ventricular pre-excitation
- Torsade de pointes
- Polymorphic VT

Differentiating ventricular tachycardia from other rhythms is of ut most importance for prognosis and therapeutic management. The following elements have been suggested for helping the differential diagnosis [2].

ECG During Sinus Rhythm

The presence of conduction defect during sinus rhythm in a previous or subsequent ECG and the comparison with the QRS complex during tachycardia may guide the diagnosis. Furthermore, the presence of PVCs with the same morphology of tachycardia may orientate to VT. If the onset of tachycardia is recorded, the initiation by a premature P wave suggests SVT, whereas initiation by PVC may be either SVT or VT. If the wide QRS tachycardia starts with shorter PR interval between last sinus P and first wide QRS complex, VT should be suspected.

Atrioventricular Dissociation During Tachycardia

AV dissociation, when present, is diagnostic for VT. Detection of P waves may be challenging. Notches and irregularities repeated cyclically may be the only indication of underlying P waves. AV dissociation also may be seen in junctional tachycardias with retrograde second-degree block, but this condition is rare [3]. Conversely a 1:1 AV correspondence is possible even during VT as 1:1 retrograde conduction may be present.

Precordial Concordance

When in all the six standard precordial leads the QRS complexes have the same polarity (all positive or all negative), that so-called concordance suggests VT diagnosis. Exceptions may occur: a negative concordance has a higher specificity; a positive concordance may be also observed in WPW during conduction through a left lateral pathway.

Captures and Fusions

During tachycardia a sinus beat may be conducted to the ventricle, resulting in a normal (capture) or a hybrid narrower (fusion) QRS complex. This finding is a strong evidence of VT. Exceptionally, fusion beats may be present during aberrant supraventricular tachycardia when in presence of BBB a contralateral PVC (i.e., PVC from the right ventricle during RBBB) results in pseudonormalization of the QRS complex.

Morphology of QRS Complex

The abovementioned findings are a strong evidence of VT but available only in a small number of cases. Most of the times, there are no clues for the diagnosis other than the QRS itself. Hence, QRS morphology analysis is of ut most importance. During aberrant conduction, the ventricle is activated through the left or right bundle, and VT is suspected when QRS complexes do not resemble typical LBBB or RBBB. The first portion of the QRS complex is called intrinsicoid deflection or R wave peak time and represents the conduction through the His bundle. Since aberrant conducted impulses follow the His pathway, the intrinsicoid deflection remains narrow or less wide during aberrance (about <0.04 s) but enlarges during VT. Exception may occur depending on the underlying heart disease (MI scar, ventricular remodeling, or drug treatment) and origin of arrhythmias (the closer the arrhythmia is to His bundle, the narrower the intrinsicoid deflection is). Wide QRS tachycardias have been classically divided in RBBB morphology depending on the polarity of the main QRS deflection in lead V_1 (positive deflection is RBBB and negative deflection is LBBB). Table 18.1 shows the morphology criteria for VT diagnosis, based on intracardiac studies [4, 5].

Diagnostic Elements Other than ECG

In diagnosis of wide-complex tachycardias, there are few elements other than ECG that may help the diagnosis. AV dissociation may be diagnosed during tachycardia with echocardiography: pulse Doppler of mitral inflow shows dissociation between A wave and QRS complexes. Clinically AV dissociation is suggested by first-tone intensity variability as clinical sign, but this finding has poor sensitivity.

Table 18.1 ECG findings indicating VT in wide complex tachycardia

	Lead V1	Lead V6
LBBB morphology	R wave >30 ms, onset of QRS to nadir of S wave >60 ms	Any Q wave
RBBB morphology	Monophasic R or biphasic qR, QR, RS	rS, QR, qr

Response to vagal maneuvers and adenosine may help the diagnosis. The arrhythmia interruption or variation in AV ratio with unmasking of underlying P or flutter waves favors VT.

What May Be Misleading

Age should not be considered. Older patients have higher probability to have both VT and SVT.

Heart rate should not be used for absolute diagnosis, but at a regular frequency of 150 bpm, atrial flutter with 2:1 VA conduction should be suspected. In this setting, vagal maneuvers or adenosine may unmask flutter waves through transient AV block.

Although the QRS tends to be wider during VT than during SVT, width itself should not be used as a diagnostic element.

Last, the hemodynamic tolerance of VT and SVT relies on several factors and is of most clinical importance but is not useful for differential diagnosis.

What If the Diagnosis Remains Unclear?

Despite accurate ECG analysis, differentiation between VT and SVT is not always clear. The patient should be treated as VT when the diagnosis remains unclear.

Clinical Presentation

Clinical symptoms and signs of VT presentation may have a wide variability. VT can be classified as hemodynamically stable and unstable. The patient with a hemodynamically stable VT can be totally asymptomatic or contrariwise show symptoms such as palpitations, dyspnea, chest pain, fatigue, dizziness, pre-syncope, and syncope. The hemodynamically unstable VT is characterized by systolic hypotension and all the signs of organ hypoperfusion up to pulseless VT and cardiogenic shock. Another important complication of high-rate VT is its degeneration into ventricular fibrillation, a condition that without a rapid treatment can cause patient's death.

Management of VT

Acute Management

The first step in the management of VTs is to assess the hemodynamic stability of the patient:

Unstable patient: hemodynamic compromise may occur with any VT, regardless of the etiology. Furthermore, patients who initially appear stable may deteriorate rapidly.

Unstable patients but still responsive and with an arterial pulse should undergo emergent synchronized cardioversion (100–200 j in monophasic shock or 50–100 j in biphasic shock) with uptitration of energy as needed [6]. If the QRS complex and T wave cannot be distinguished accurately, a synchronized shock could not be possible. Such patients should be treated with immediate defibrillation (unsynchronized shock using 360 J in monophasic shock or 200 J in biphasic shock).

Use of intravenous sedatives may be appropriate but must be balanced against the risks of further hemodynamic deterioration.

Patients who become unresponsive or pulseless should be managed according to ACLS resuscitation algorithms, with immediate high-energy defibrillation [7].

Stable patients: in hemodynamically stable patients, additional time can be given for the differential diagnosis between VT and SVT, and therapy may be targeted to the specific arrhythmia substrates.

Ventricular Tachycardia or Uncertain Diagnosis (Should Be Treated as a VT) [7]

- Elective electrical cardioversion:
  - Synchronized cardioversion (100–200 j in monophasic shock or 50–100 j in biphasic shock) with uptitration of energy as needed.
- Pharmacological cardioversion [7]:
  - Procainamide (10–15 mg/kg): proved to be superior to lidocaine (1.5 mg/kg) for termination of hemodynamically stable monomorphic VT. It can be administered at a rate of 20–50 mg/min until the arrhythmia is

suppressed. It should be avoided in patients with long QT or heart failure or low EF.
- Sotalol (100 mg IV over 5 min): also more effective than lidocaine in patients with sustained monomorphic VT. It should be avoided in patients with long QT.
- Amiodarone (150 mg IV over 10 min up to 1.2 g/24 h) is useful in recurrent monomorphic or refractory VTs.
- Lidocaine (1–1.5 mg/kg IV bolus): should be considered the second-line antiarrhythmic therapy for monomorphic VT and only in ischemic setting.

Supraventricular Tachycardia

Management is similar to an SVT with a normal QRS duration.

In SVT due to a reentrant circuit:

- Vagal maneuvers may be considered such as carotid sinus pressure (if no carotid bruits are present) or Valsalva maneuver as the initial intervention.
- Adenosine (6 mg IV over 1–2 s): highly effective in terminating many SVTs (e.g., AVNRT, AVRT), and for others (e.g., AF, atrial flutter), adenosine may facilitate the diagnosis by slowing the ventricular response to allow clearer assessment of atrial activity. If the initial dose is ineffective, a 12 mg dose may be given and repeated once if necessary. Adenosine has a very short half-life (less than 10 s), reducing the risk of an untoward reaction.
- Calcium channel blockers or beta-blockers: intravenous verapamil (2.5–5 mg IV) or beta-blockers (e.g., metoprolol 5–10 mg IV) may be given if the SVT persists after adenosine administration. These medications can terminate AVNRT as well as some atrial tachycardias. If the specific SVT diagnosis remains unknown, these drugs may slow the ventricular response and facilitate diagnosis. Finally, calcium channel blockers and beta-blockers should not be used in AVRT.
- Cardioversion is rarely necessary in patients with a stable SVT. However, if AVNRT or AVRT persist after the above interventions, synchronized cardioversion is usually effective in restoring sinus rhythm.

In atrial fibrillation, atrial flutter, or atrial tachycardia, a strategy of rate or rhythm control may be chosen according to clinical indications.

Chronic Management

Ventricular Tachycardia

In patients who survived a sudden cardiac arrest/VT, the first step is to exclude possible transient reversible causes. In the presence of sign of acute myocardial ischemia, myocardial revascularization (PTCA, CABG) may be performed. Myocardial revascularization may be sufficient in patients surviving VF in association with myocardial ischemia (normal ejection fraction and no history of MI). Instead, sustained monomorphic VT with prior MI is unlikely to be affected by revascularization [8]. Electrolyte abnormalities should be also excluded as the cause of the arrhythmias.

In sudden cardiac arrest/VT without removable cause, randomize controlled trials showed ICD superiority compared with antiarrhythmic drug therapy in prevention of sudden death [8].

2012 AHA/ACC/HRS guidelines recommended:

- ICD therapy is indicated in patients who are survivors of cardiac arrest due to VF or hemodynamically unstable sustained VT after evaluation to define the cause of the event and to exclude any completely reversible causes. (Class I, Level of Evidence: A)
- ICD therapy is indicated in patients with structural heart disease and spontaneous sustained VT, whether hemodynamically stable or unstable. (Class I, Level of Evidence: B)
- ICD implantation is reasonable for patients with sustained VT and normal or near-normal ventricular function. (Class IIa, Level of Evidence: C)
- ICD therapy is not indicated for patients with ventricular tachyarrhythmias due to a completely reversible disorder in the absence of structural heart disease (e.g., electrolyte imbalance, drugs, or trauma). (Class III, Level of Evidence: B)

Antiarrhythmic drug therapy may be considered as the second choice in patients who cannot receive ICD therapy or in patients with recurrent VT/VF associated with ICD shocks [9].

Supraventricular Tachycardia

WCTs due to SVT should be treated as SVT with narrow QRS complex (see Chap. 19).

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