Wide QRS Complex Tachycardia in the Emergency Setting

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6.1 Wide QRS Complex Tachycardia

A wide QRS complex tachycardia can be (1) ventricular tachycardia (VT); (2) supraventricular tachycardia (SVT) with bundle branch block that may be either preexisting or due to aberrant conduction, namely, tachycardia-dependent abnormal intraventricular conduction; a further possibility is the effect of some antiarrhythmic drugs that slow down intraventricular conduction, resulting in marked QRS complex widening; and (3) supraventricular tachycardia with conduction of impulses to the ventricles over an accessory pathway (preexcited tachycardia).

In the presence of wide QRS tachycardia, the correct diagnosis is of paramount importance, since the treatment commonly used in SVT is different from that of VT, and some drugs useful in the former (e.g., verapamil) are harmful in the latter [1–3].

The origin of a wide QRS complex tachycardia can be reliably identified using a "holistic" approach, namely, taking into account all of the available items: no single criterion is able to provide a simple and quick solution of the problem in all cases. The available ECG signs are, without any exception, suggestive of ectopy, namely, ventricular origin of the impulses; SVT with aberrant conduction may only be diagnosed by excluding all of the items favoring VT. The recognition of ventricular or supraventricular origin of wide QRS complex tachycardias is not difficult if a

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detailed analysis is used, taking into account several diagnostic signs: [4–12] the idea that a single quick item can offer an immediate and reliable solution is something of an illusion.

If, despite a complete diagnostic approach, the dilemma cannot be resolved, it is necessary to assume a ventricular origin of the arrhythmia since (1) a wide QRS tachycardia is more likely VT than SVT and (2) it is less dangerous to treat an SVT like it were ventricular in origin than applying to a patient with VT the treatment commonly used for SVTs. In particular, intravenous verapamil should be avoided whenever SVT diagnosis is not certain, since this drug is harmful in some VT patients [1–3].

6.2 General Criteria

6.2.1 Atrioventricular Dissociation

Whenever the electrical activity of the atria is recognizable, two different situations may occur:

1. Atrioventricular (A-V) dissociation
2. Relationship between P waves and QRS complexes.

A-V dissociation demonstrates the ventricular origin of the wide QRS complexes and occurs in a percentage variable from 19 to 70 % of VT cases [5, 6, 10, 11]. Dissociation, however, is often difficult to be diagnosed since in several cases, sinus P waves are not easily recognizable, being simultaneous to QRS complexes or T waves. Moreover, in the presence of atrial fibrillation, A-V dissociation cannot be appreciated. Before excluding, in a wide QRS complexes tachycardia, the presence of P waves independent of QRS complexes, however, one should observe with great attention the configuration of several consecutive complexes in all 12 leads, paying the greatest attention to leads II and V1 (the ones where sinus P waves are usually evident). Aim of this analysis is comparing consecutive complexes searching for slight differences in QRS or T morphology: with this approach it is not rare to discover, in the presence of VT, that in some leads, slight variations in QRS complex or T wave configuration occur. To be sure that such differences express the presence of P waves dissociated from QRS complexes, and superimposed on these, it is necessary to measure the intervals separating the "disturbing" events: in case of A-V dissociation, they are separated from relatively constant intervals, being "long" intervals in multiples of the "short" ones (Fig. 6.1a). When, in contrast, the intervals separating the changes in morphology of T waves and/or QRS complexes are irregular, it is more likely that artifacts, rather than dissociated sinus P waves, are involved (Fig. 6.1b).

The best ECG leads to be analyzed, searching for "dissociated" P waves, are leads II and V1, the ones where sinus P wave voltage is usually relatively high; it is also advisable to observe the leads where the QRS complex and/or the T wave is of

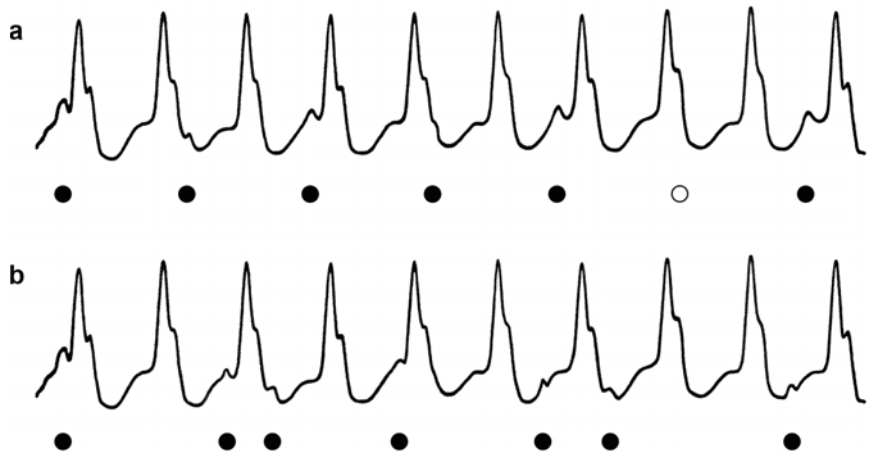

Fig. 6.1 Diagrams (**a**, **b**) show two wide QRS complex tachycardias. In both diagrams, small positive deflections, independent of QRS complexes, are present. In diagram (**a**) these deflections are rhythmic and separated by constant intervals; whenever a deflection is invisible, being coincident with a QRS complex (*circle*), the interval between two manifest waves is twice the basic interval. These small waves are, therefore, sinus P waves: accordingly, A-V dissociation can be diagnosed, revealing a ventricular origin of tachycardia. In diagram (**b**), in contrast, the small positive deflections are arrhythmic: they are not P waves but artifacts

low voltage, since it is relatively easy to detect the small atrial waves whenever these are not "buried" within large QRS or T deflections. This is expressed by the "haystack principle": if you are searching for a needle in a haystack, select a small haystack" (Fig. 6.2).

The bedside diagnosis of A-V dissociation can be improved by heart sound auscultation and arterial pulse palpation: whenever the atrial contraction is dissociated from ventricular activity, it is possible to appreciate a variable loudness of the 1st heart sound and variability in peripheral pulse amplitude. This is because (1) whenever atrial contraction occurs immediately before ventricular systole, the blood flow "opens" the atrioventricular valves, resulting in a relatively loud 1st heart sound, a phenomenon that does not occur if mitral and tricuspid valves are closed at the time of atrial systole, and (2) if atrial contraction occurs when the A-V valves are open, the diastolic ventricular filling is improved, resulting in a relatively increased stroke volume: accordingly, the pulse amplitude will be higher with respect to that of heart beats in which atrial systole occurs while the A-V valves are closed.

6.2.2 Second-Degree V-A Block

In ventricular tachycardia, atrial electrical activity may be not dissociated from ventricular one if retrograde ventricular-atrial (V-A) conduction occurs, as it happens in about one half of cases. The V-A ratio may be 1 (every QRS complex is followed by a

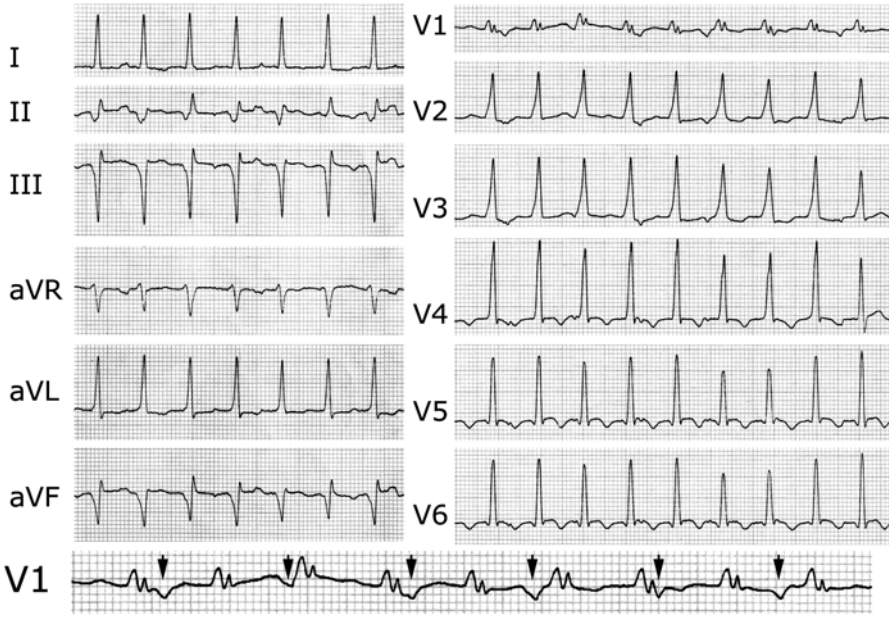

Fig. 6.2 Wide QRS complex tachycardia. QRS duration is 0.12 s, but since in some leads ventricular complexes are relatively narrow, a supraventricular tachycardia could be diagnosed at first glance. The ventricular origin of tachycardia is demonstrated by A-V dissociation; the P waves independent of ventricular complexes (*arrows*), and separated from constant intervals, are easily recognized in lead V1, since in this lead both QRS complexes ant T wave voltages are very low (the haystack principle)

retrograde P wave) or less than 1 when some ventricular impulses are not conducted to the atria. In a wide QRS complex tachycardia, a QRS/P ratio >1 (more QRS complexes than P waves) demonstrates the ventricular origin of the arrhythmia [6–8, 12], (Fig. 6.3), whereas a 1:1 ratio does not permit any definite conclusion since P waves may (a) express a supraventricular tachycardia with 1:1 A-V conduction or (b) represent the retrograde atrial activation during ventricular tachycardia. If analysis of P wave configuration is possible, a main P vector directed inferiorly demonstrates supraventricular origin of the arrhythmia, whereas a P vector directed superiorly (negative P waves in the inferior leads) does not permit any conclusion since not only VT but also several supraventricular tachycardias share a retrograde activation of the atria. In some cases of VT, however, retrograde P waves appear as positive in the inferior leads, a phenomenon that has been called "the illusion of retrograde positive P waves" (Fig. 6.4) [8, 13, 14].

6.2.3 Capture and Fusion Beats

The presence of narrow, or relatively narrow, beats during wide QRS tachycardia suggests a diagnosis of VT, *provided that narrow complexes are preceded by a P wave with an interval consistent with anterograde A-V conduction of the impulse*. The narrow, or less wide, complexes are *capture* or *fusion* beats that occur

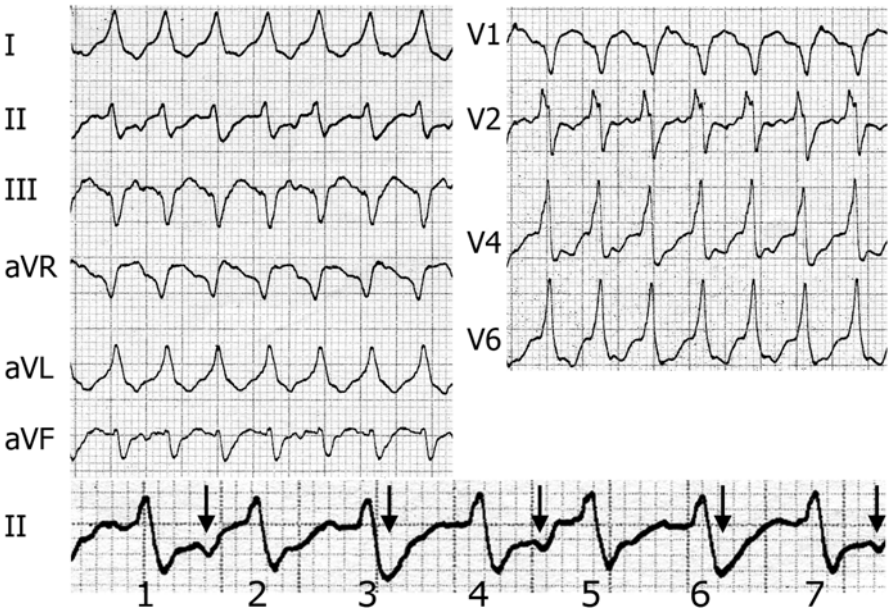

Fig. 6.3 Ventricular tachycardia with 3:2 retrograde block of the Wenckebach type. Lead II (enlarged in the *bottom row*) analysis reveals that ventricular complexes *1*, *4,* and *7* are followed by negative P waves occurring midway between two consecutive QRS complexes. Beats *3* and *6*, in turn, show very wide "S waves" that never occur in the other beats, whereas complexes *2* and *5* do not show any of the 2 above characteristics (negative P wave, wide "s wave"). It is, therefore, evident that in a group of 3 beats, the 1st one (complexes *3* and *6*) is followed by a retrograde P wave with a short R-P interval, whereas the P wave following the 2nd beat of the group (complexes *1*, *4*, *7*) occurs with a relatively long interval, and after the 3rd complex of the series (beats *2* and *5*), no P wave occurs. In other words, there is a retrograde 2nd-degree 3:2 Wenckebach type block, and this establishes the diagnosis of ventricular tachycardia. In this tracing, the r wave peak time in lead II is 30 ms

whenever, during VT, a sinus or supraventricular impulse succeeds in reaching the ventricles, whose depolarization is due totally (capture) or partially (fusion) to that impulse (Fig. 6.5). Capture and fusion beats are reliable signs of VT, but they are rare (4 % in a study based on 96 cases of proved VT [10]) and can be observed only in the presence of A-V dissociation. Since the latter phenomenon is, in itself, a clear sign of VT, the further help provided by capture and fusion beats is trivial, provided that these never occur whenever the heart rate is very high, being the A-V node made always refractory by ectopic ventricular impulses [6, 8].

6.2.4 Precordial QRS Concordance

A "concordant" QRS morphology in all the precordial leads, namely, ventricular complexes totally negative (QS pattern) or positive (R or qR pattern), demonstrates a ventricular origin of tachycardia, since no intraventricular conduction

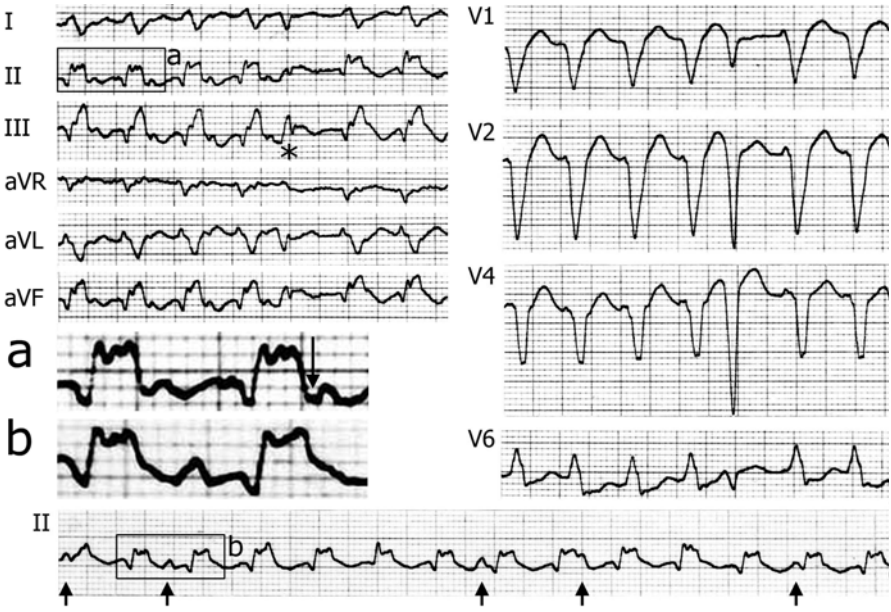

Fig. 6.4 Ventricular tachycardia with retrograde P waves apparently positive in the inferior leads. In all leads, that are simultaneous, apart from the tracing in the bottom strip, the 5th beat (*asterisk*) is a ventricular extrasystole. Beats 1–4 are followed by P waves that seem, at first glance, positive in the inferior leads and negative in aVR and aVL. The premature beat alters the relationship between QRS complexes and P waves, being the last two ventricular beats not followed by P waves. The bottom strip (lead II) has been recorded later with respect to the others and clearly shows A-V dissociation: P waves of sinus origin are positive (*arrows*) and independent of QRS complexes; some sinus P waves, occurring simultaneously with ventricular complexes, are invisible, being "buried" within the ventricular complexes. Panels (**a**, **b**) show enlarged beats: during retrograde V-A conduction, P waves are negative (*arrow* in section **a**), not positive as they seem at first glance. In section (**b**), a positive P wave is evident in between two QRS complexes, demonstrating A-V dissociation

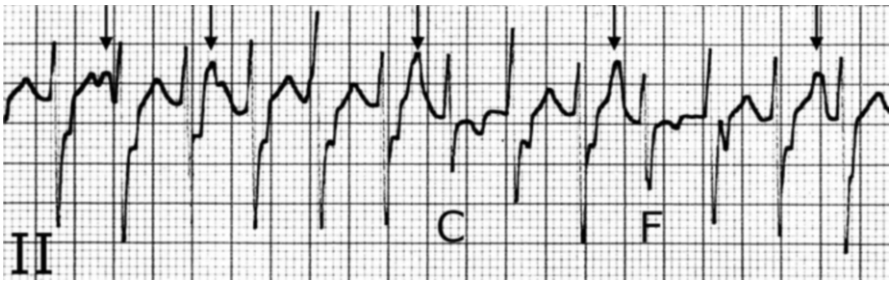

Fig. 6.5 Capture and fusion beats during ventricular tachycardia. In this ECG strip, A-V dissociation is evident (*arrows* point out sinus P waves that modify T wave morphology). In two occasions, the sinus impulse is conducted to the ventricles, giving rise to a narrow QRS complex (capture beat, labeled *C*) or to a fusion beat (labeled *F*). These are intermediate in configuration between ectopic wide complexes and capture beat

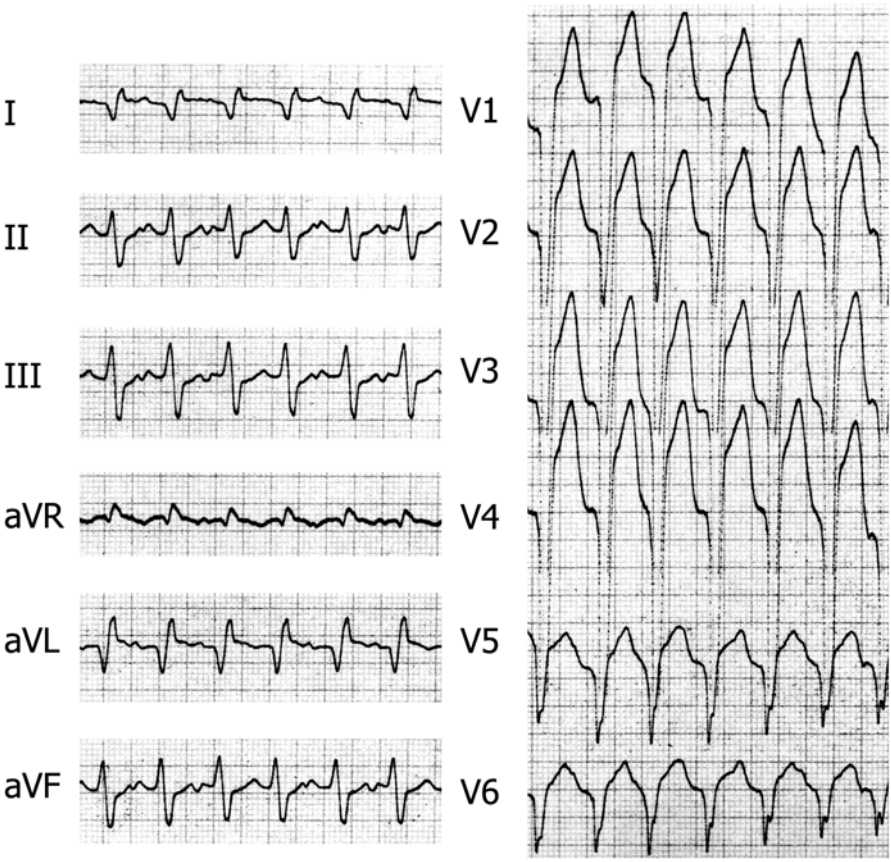

Fig. 6.6 Precordial concordance. In this tachycardia, wide QRS complexes with QS morphology are present in all precordial leads. This pattern demonstrates without any exception the ventricular origin of tachycardia. Analysis of the inferior leads also reveals a 2nd-degree V-A block with 3:2 ratio; the retrograde negative P waves modify the T wave configuration in two consecutive beats, whereas in the 3rd QRS complex, the T wave is not affected

disturbance can result in such a configuration [15]. Concordance, however, cannot be diagnosed if rS, Rs, or rs complexes occur even in one single precordial lead. In a study based on 232 electrocardiograms with bundle branch block analyzed during sinus rhythm, none showed precordial concordance, suggesting a 100 % specificity of this sign indicating the ventricular origin of the arrhythmia [16]. Negative concordance (QS morphology in all precordial leads, Fig. 6.6), however, is specific of VT, whereas positive concordance could be observed, although rarely, in a preexcited tachycardia due to a left-sided Kent bundle [7, 8]. Negative concordance in the bipolar limb leads (I, II, III) has also been proposed as a specific pattern suggesting VT; [17] such a configuration demonstrates an extreme right axis deviation, a phenomenon that never occurs in adults, apart from some cases of congenital heart disease or dextrocardia.

6.2.5 Absence of RS Complexes in the Precordial Leads

In several cases of VT, none of the precordial leads shows ventricular complexes with a configuration characterized by an R wave followed by an S wave (rs, RS, rS, or Rs). This sign, expressing in a slightly different manner the concept of "precordial concordance," suggests a ventricular origin of the arrhythmia. The sign specificity was 100 % both in the original study [5] and in another research based on 133 patients with wide QRS tachycardia; [10] in a different series, however, specificity was 81 and 98 % in the presence of positive and negative precordial QRS complexes, respectively [16].

6.2.6 Interval >100 ms from QRS Complex Beginning to S Wave Nadir in a Precordial Lead

It has been observed that whenever, in a wide QRS complex tachycardia, the interval from QRS complex beginning to S wave nadir exceeds 100 ms in a precordial lead, tachycardia is ventricular in origin [5]. The above criterion was fulfilled in 41 % of patients with previous myocardial infarction and VT [18]. In subjects with slowed down intraventricular conduction, however, leads V4–V6 show at times the above-mentioned sign even during sinus rhythm, particularly in the presence of left axis deviation. In a study based on electrocardiograms with left bundle branch block and sinus rhythm, 34 % of cases had an interval from QRS complex beginning to S wave nadir >100 ms [16], demonstrating a low specificity of this sign in revealing VT.

6.2.7 Vagal Stimulation Maneuvers

In the presence of QRS wide complex tachycardia, vagal stimulation can result in the following responses:

1. No change in tachycardia morphology or rate: the question remains open.
2. Sinus rhythm restoration: supraventricular reentrant tachycardia with a circuit incorporating the A-V node.
3. Variation in A-V conduction ratio, with appearance of P or F waves: atrial tachycardia or atrial flutter.
4. Variation in V-A conduction ratio, demonstrated by QRS complexes not followed by a retrograde P wave: ventricular origin of tachycardia.

6.3 The Electrocardiogram in the Absence of Tachycardia

An ECG recorded in the absence of tachycardia can be at times helpful, since a conduction disturbance or preexcitation observed during sinus rhythm can be the key to recognize the mechanism underlying the wide QRS complexes. In the great

majority of cases, however, no ECG recorded during sinus rhythm is available at the moment of the arrhythmic emergence. Whenever the ECG during tachycardia is identical to that obtained in sinus rhythm, the arrhythmia is supraventricular in origin, apart from a single exception: the bundle branch reentry tachycardia [19]. In the latter condition, a tracing in sinus rhythm can be misleading, since it suggests a supraventricular, rather than ventricular, origin of the arrhythmia [19].

6.4 QRS Complex Morphology in Leads V1 and V6

Analysis of QRS complex configuration represents an important tool in wide QRS complex tachycardia. Whenever other diagnostic signs (A-V dissociation, capture and fusion beats, precordial concordance, etc.) are either absent or controversial, the distinction between supraventricular and ventricular tachycardia lies on morphologic analysis of ventricular complexes, taking particularly into account leads V1 and V6. The 1st step is tachycardia classification based on QRS morphology in lead V1: whenever ventricular complex is mainly positive in this lead, tachycardia will be defined as "RBBB type," whereas if in that lead ventricular complexes are negative, tachycardia will be classified as "LBBB type." In any situation, the leads to be analyzed are V1 and V6.

6.4.1 Wide QRS Complex Tachycardia with Right Bundle Branch Block-Type Configuration (Positive QRS Complex in Lead V1)

V1. Ventricular complexes with morphology *R* or *Rr′* (the 1st R wave higher than the 2nd one), as well as *qR* or *RS* complexes, suggest VT, whereas both a triphasic (*rsR*Đ or *rSR*Đ) or biphasic configurations *rR*Đ with the 2nd R wave higher than the 1st one suggest SVT with aberrant conduction (Fig. 6.7).
V6. In this lead, *rS*, *QS,* or *qR* complexes are specific of VT (Fig. 6.8), whereas *qRs* complexes suggest aberrant conduction (specificity 95 %). Whenever the R/S ratio, however, is <1 (larger S wave than R wave voltage in lead V6), the ventricular, rather than supraventricular, origin of tachycardia is more likely [10, 16, 20].

6.4.2 Wide QRS Complex Tachycardia with Left Bundle Branch Block-Type Morphology (Negative QRS Complex in Lead V1)

V1. In the presence *of SVT with aberrant conduction and LBBB morphology, this lead shows either a QS configuration or a small and relatively narrow (≤30 ms) r wave* followed by a deep S wave with an early (<60 ms) nadir. R wave duration >30 ms or S wave nadir later than 60 ms from the QRS complex beginning suggest ventricular origin of the arrhythmia. Moreover, a notch in the proximal (descending) limb of S wave indicates VT (Fig. 6.8).

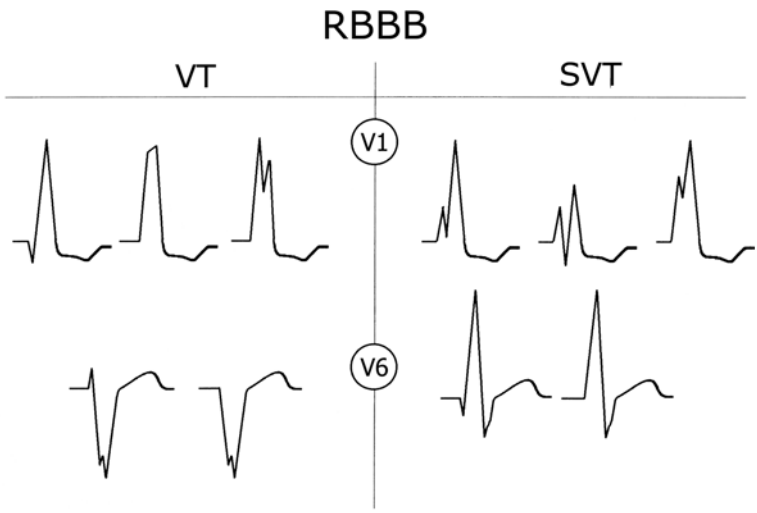

Fig. 6.7 Ventricular complex morphology suggesting either VT or SVT with aberrant conduction in wide QRS complex tachycardia with RBBB-like configuration (QRS complex mainly positive in lead V1)

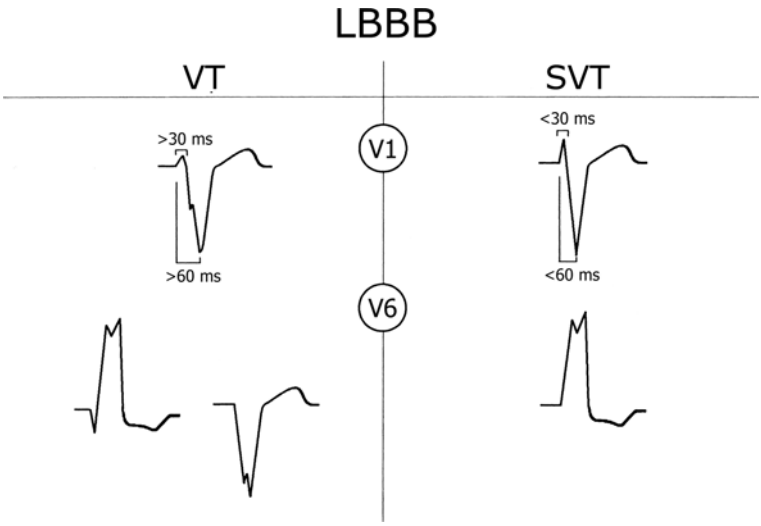

Fig. 6.8 Ventricular complex morphology suggesting either VT or SVT with aberrant conduction in wide QRS complex tachycardia with LBBB-like configuration (QRS complex mainly negative in lead V1)

V6. In a wide QRS complex tachycardia, negative or predominantly *negative QRS complexes in lead V6 suggest at first glance a ventricular origin* of the arrhythmia independent of the associated intraventricular disturbance pattern [20]. Impulses

of ventricular origin show often (69 % of cases) a negative net QRS amplitude (algebraic sum of positive and negative deflections) in V6, whereas this pattern is relatively rare (27 % of cases) in supraventricular tachycardia.

These signs based on QRS morphology in leads V1 and V6, introduced by Kindwall [21], express the slow initial progression of the ventricular wave front when the depolarization is not due to an impulse conducted over the His-Purkinje system. In true LBBB, thus, the 1st ventricular vector, expressing the right-to-left septal depolarization, is directed to the left and anteriorly: lead V1, thus, cannot show a *wide* r wave since the initial ventricular depolarization is fast, being the supraventricular impulse conducted over the Purkinje system. Accordingly, *in a broad QRS tachycardia with LBBB configuration, a relatively wide (≥30 ms) r wave in lead V1 strongly suggests a ventricular origin of the arrhythmia*. The same holds true for a relatively late (>60 ms) S wave nadir in lead V1 and for the presence of a notch in the descending S wave limb. The specificity of the above "ectopic" QRS morphologies in lead V1, however, is not 100 %: a study based on electrocardiograms with LBBB during sinus rhythm has reported a specificity of 78, 66 and 66 % for r wave duration in lead V1 >30 ms, S wave nadir >60 ms, and notch in the descending S wave limb, respectively [16].

6.4.3 Limitations of Criteria Based on QRS Morphology in Wide QRS Complex Tachycardia

Despite being useful, morphologic criteria are not absolute; the equations *typical bundle branch block = aberrant conduction, atypical bundle branch block = ventricular tachycardia* suffer some *limitations and may lead to wrong conclusions in patients treated with antiarrhythmic drugs, particularly those belonging to class 1C*. This is because these drugs slow down the intraventricular conduction, resulting in very abnormal ventricular complexes. Patients treated with 1C drugs may show extremely wide QRS complexes during SVT, to the extent that morphological analysis leads to a wrong diagnosis. This occurs not rarely in patients with atrial flutter treated with 1C antiarrhythmic drugs, that result in tachycardia rate reduction, favoring 1:1 A-V conduction of atrial impulses, and QRS complex widening.

6.5 Other Signs

6.5.1 QRS Duration >140 ms

It has been observed that a wide complex tachycardia with QRS duration >0.14 s is very likely ventricular in origin [11], but this criterion has a low specificity, ranging from 43 % [16] to 69 % [10]. Moreover, QRS complexes can be, although rarely, relatively narrow (<0.12 s) in ventricular tachycardia.

6.5.2 QRS Axis Deviation

In several cases of VT, left or right axis deviation occurs [9–11], and rarely the QRS axis is normal (between 0° and +90°) in VT. Superior QRS axis deviation, however, has been considered not suggestive of VT in the presence of ventricular complexes with LBBB configuration, whereas an RBBB morphology is often associated with VT (87 % specificity) [16].

6.5.3 Regularity

Apart from atrial fibrillation and atrial flutter or tachycardia with variable A-V conduction ratio, supraventricular tachycardias are usually regular. This is also true for sustained ventricular tachycardias, although irregularity in itself does not exclude VT, the variability of R-R intervals being not uncommon in focal tachycardias, particularly at the beginning or at the end of tachycardia. Sustained VT cycle variability has also been attributed to exit block [22] or longitudinal dissociation in the reentry pathway of tachycardia [23, 24].

6.6 Lead aVR Analysis

A new algorithm based on lead aVR analysis has recently been proposed to distinguish ectopy from aberrant conduction in wide QRS complex tachycardia [25, 26]. The procedure is based on four steps (Fig. 6.9): the first three ones are simple and quick, whereas the 4th step requires complicated voltage measurements. Whenever the sign looked for in steps 1, 2, or 3 is present, the diagnosis is VT, whereas only in step 4 becomes possible the recognition of SVT with aberrant conduction (Fig. 6.9).

6.6.1 Step 1: Dominant Initial R Wave

146 out of 482 cases of wide QRS tachycardia showed in lead aVR *a dominant initial R wave*, which was the largest ventricular complex deflection (Figs. 6.9 and 6.10). This pattern *demonstrated a ventricular origin* of the arrhythmia, confirmed by intracardiac recordings, in 144/146 cases (sensitivity 38.9 %, specificity 98.2 %).

6.6.2 Step 2: q or r initial Wave with Duration >40 ms in qR or rS complexes

A ventricular complex starting, in aVR, *not with a dominant R wave but with a low voltage q or r wave whose duration was ≥40 ms* was present in 74 out of 336 cases without a dominant initial R wave. In 65 of these, the diagnosis was VT (sensitivity 28.8 %, specificity 91.8 %).

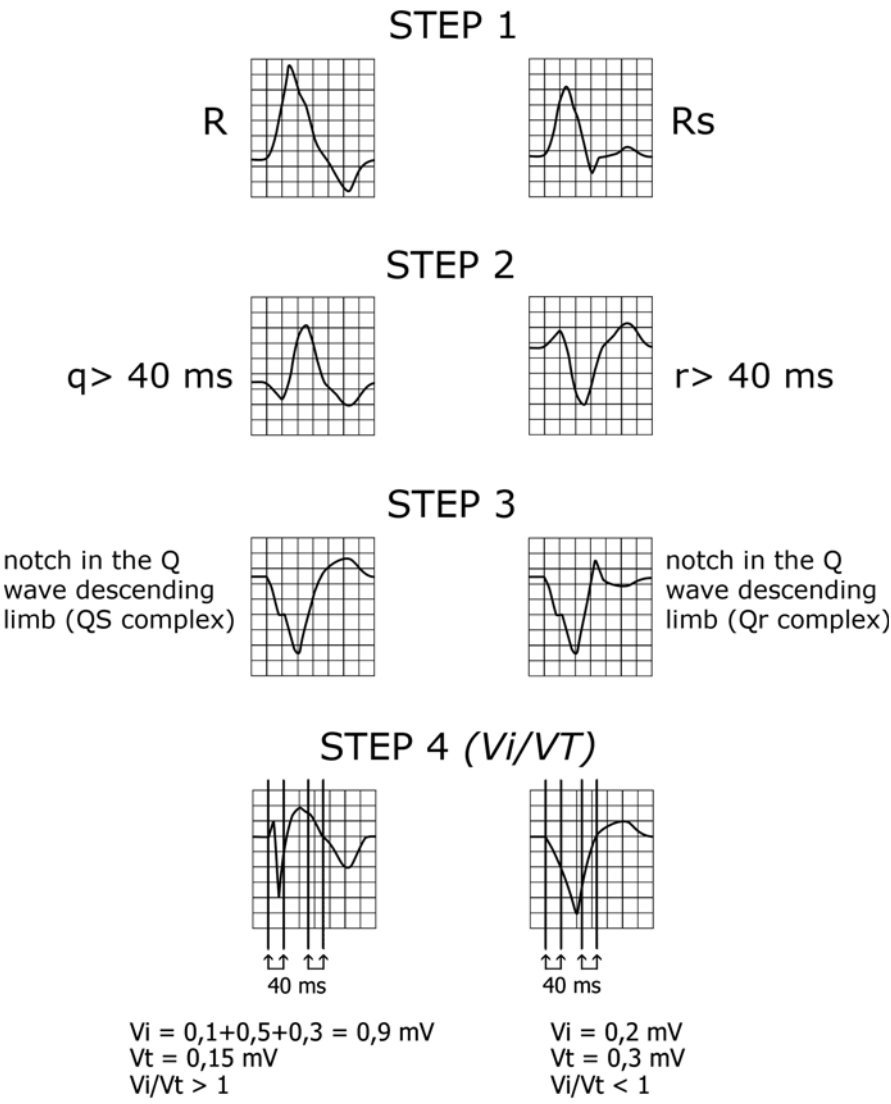

Fig. 6.9 Algorithm based on aVR analysis. *Vi* voltage of the first 40 ms, *Vt* voltage of the final 40 ms (see text)

6.6.3 Step 3: Notch in the Descending Q Wave Limb of a Negative (QS or Qr) Ventricular Complex

This pattern was present in 32 over 37 VT cases where the first 2 criteria were absent (sensitivity 19.9 %, specificity 95 %).

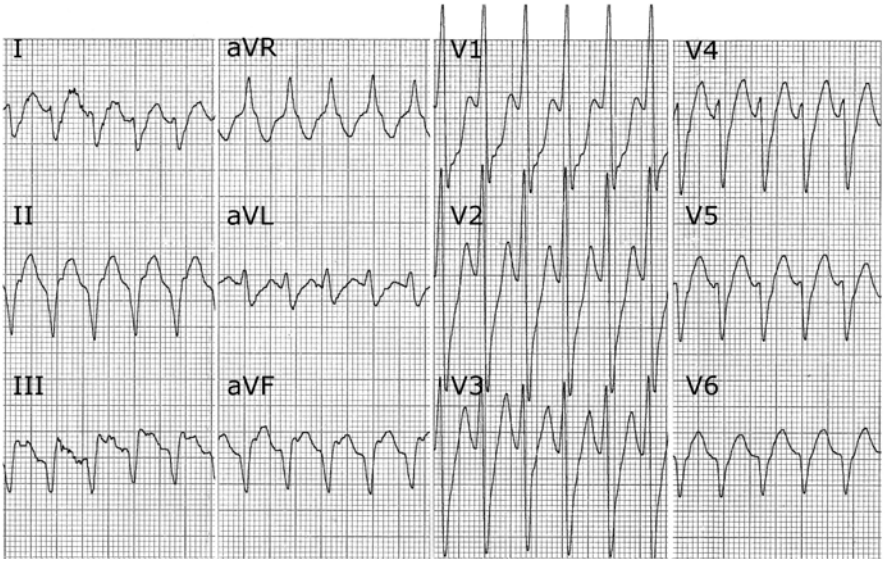

Fig. 6.10 Ventricular tachycardia. In this wide QRS complex tachycardia, negative P waves follow ventricular complexes in the inferior leads. The arrhythmia cannot be classified as "RBBB morphology" or "LBBB morphology" since lead V1 shows complexes with RS configuration. Both QS complexes in lead V6 and aVR analysis (entirely positive complexes) suggest VT

6.6.4 Step 4: *Vi*/*Vt* Ratio

The presence of any sign in steps 1–3 demonstrates the ventricular origin of the arrhythmia, but *the absence of these criteria does not permit to exclude VT and does not demonstrate SVT.* In this situation it is necessary to evaluate the *Vi/Vt ratio, namely, the ratio between the voltages recorded during the first 40 ms of the QRS complex (Vi) and those measured during the last 40 ms of the complex (Vt*). To calculate *Vi* (initial voltage) and *Vt* (terminal voltage), it is necessary to sum the amplitude of *all the deflections present in the first and the last 40 ms*, respectively (Fig. 6.10). This is not a very easy task, since the authors suggest to measure not the simple voltage of the deflection, but to take into account separately any limb of the waves. For example, if an R wave of 3 mm is present at the beginning of the ventricular complex, the corresponding calculated voltage is 6 mm (3 of the ascending limb + 3 of the descending limb). Unfortunately, it is not easy to distinguish exactly the moment of QRS complex beginning and/or termination, to the extent that the authors suggest to use a simultaneous recording of several leads (at least aVR, aVL, aVF) in order to make easier the identification of QRS complex beginning and/or termination.

A Vi/Vt ratio ≥1 suggests a diagnosis of SVT with aberrant conduction, while *a ratio ≤1 speaks in favor of VT.* This criterion has a logical basis: in VT, intraventricular conduction is totally independent of the conduction system, and the ectopic impulse is slowly conducted; accordingly, a small amount of ventricular muscle is

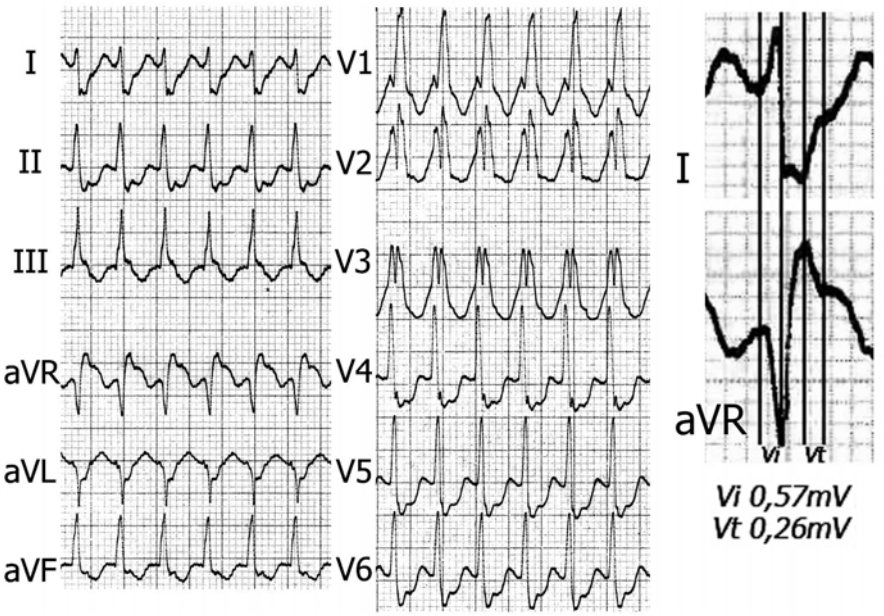

Fig. 6.11 Supraventricular tachycardia with aberrant conduction. Neither P waves nor signs of A-V dissociation are recognizable in this ECG. The morphology of ventricular complexes (rsR′ in V1, Rs with wide s wave in V6) suggests aberrant conduction. The same diagnosis results from lead aVR analysis, since neither dominant initial R wave nor wide q or r wave with duration >40 ms occurs, and is also absent a notch in the proximal q wave limb. Moreover, the ratio between the initial (first 40 ms, *Vi*) and the terminal (final 40 ms, *Vt*) voltage is >1, pinpointing the diagnosis of aberrant conduction

depolarized during the first 40 ms, resulting in a relatively low voltage of the corresponding ECG deflections. When, in contrast, the initial ventricular depolarization occurs using the Purkinje system, as it happens in bundle branch block, a relatively large myocardial mass is depolarized during the first 40 ms, resulting in relatively high voltage-related deflections. Examples of correct diagnosis arrived by means of aVR analysis are reported in Figs. 6.11, 6.12, and 6.13.

In conclusion, aVR analysis can be very helpful whenever one of the signs described in steps 1–3 is present, but becomes less useful in their absence.

6.7 R Wave Peak Time at Lead II

It has recently been reported that the *R wave peak time (RWPT) duration in lead II (the interval from the QRS complex beginning to the 1st change in polarity*) permits a quick and reliable distinction between VT and SVT [27]. The authors have observed that an *RWPT ≤50 ms demonstrates a supraventricular origin* of the arrhythmia, whereas an *RWPT ≥50 ms in lead II suggests VT*. The underlying reason why a long RWPT speaks in favor of ventricular origin of tachycardia is

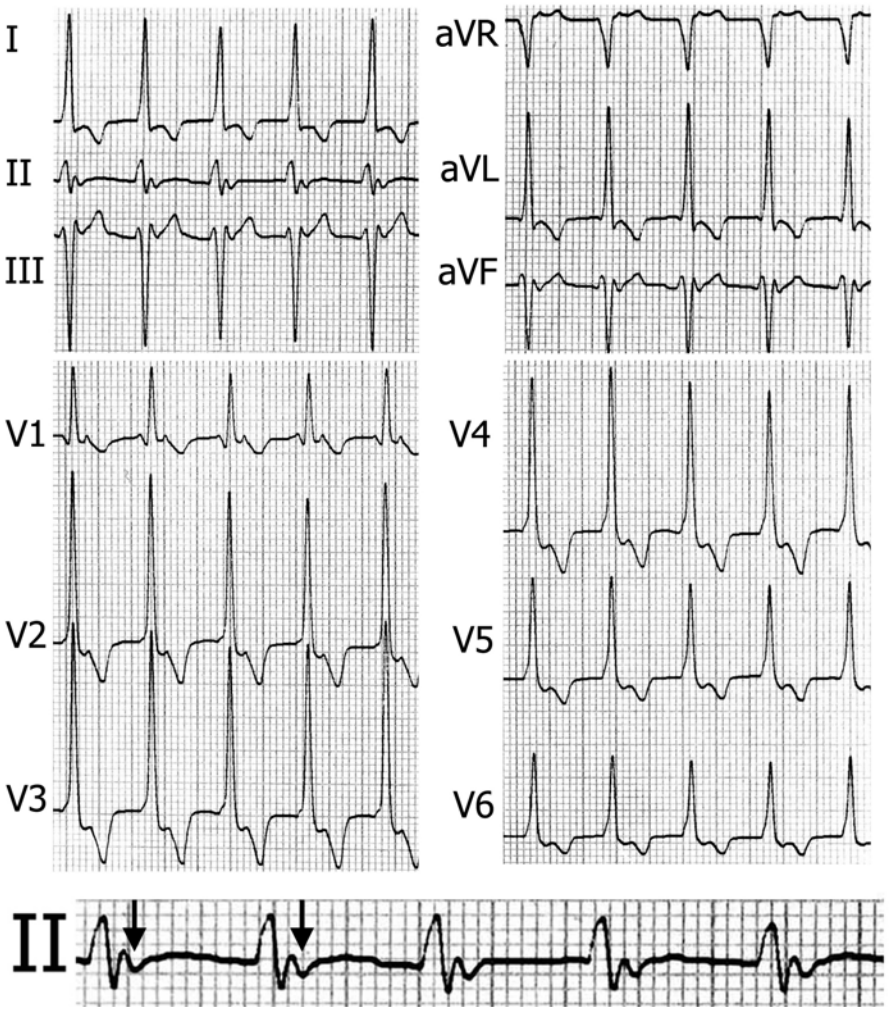

Fig. 6.12 Wide QRS tachycardia with 1:1 V-A conduction. Negative P waves follow ventricular complexes with constant R-P interval in the inferior leads (*arrows* in the *bottom strip*, representing lead II enlarged). This pattern suggests a diagnosis of ventricular (fascicular) tachycardia, despite the relatively narrow QRS complexes

likely the slow initial diffusion of the depolarization wave front in VT. In SVT, in contrast, at the beginning of ventricular depolarization, the supraventricular impulse is normally conducted over a bundle branch (or a fascicle) and the Purkinje system, in such a way that a relatively large myocardial mass is quickly depolarized, resulting in a short RWPT. In the case of VT, in contrast, the cardiac impulse slowly travels over the common myocardial tissue, resulting in a long RWPT. The efficacy of this sign in discriminating VT from SVT is very high in the study that has described such a criterion; the experience of our group,

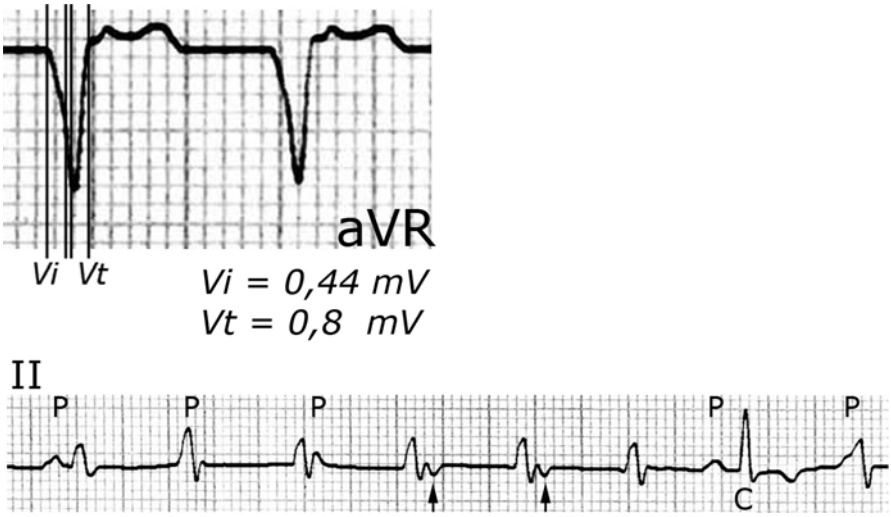

Fig. 6.13 Same case of the preceding figure. Analysis of lead aVR (*top*) and effect of verapamil administration (*bottom*). Despite the absence of (a) dominant initial R wave, (b) q wave with duration >40 ms, (c) notch in the q wave descending limb, the ratio between the voltages of the first and the final 40 ms (*Vi/Vt*) is less than 1, supporting the diagnosis of VT. The ECG recorded during intravenous verapamil administration (strip of lead II) shows A-V dissociation (the 1st three and the last QRS complex) and a capture beat (*C*); *arrows* indicate retrograde conduction restarting

however, is less satisfactory. Further research is necessary to evaluate the real efficacy of RWPT in lead II. Figure 6.3 shows a case of VT in which the RWPT does not exceed 30 ms.

In conclusion, the classic diagnostic criteria to distinguish VT from SVT with aberrant conduction include the following:

1. A-V dissociation, characterized by absence of any relationship between QRS complexes and P waves; this phenomenon is at times immediately recognizable but more often can be discovered only by means of a detailed analysis of the tracing.
2. Second-degree ventriculoatrial block, namely, a relationship between P waves and QRS complexes, but with more ventricular complexes than P waves.
3. Fusion and/or capture beats.
4. Concordant precordial pattern, a sign that can be also expressed as absence of RS (or even rs, Rs, rS) complexes in the precordial leads.
5. Analysis of QRS complexes aimed at discriminating a typical pattern of intraventricular conduction disturbance from a QRS configuration that is impossible or unlikely whenever the ventricular depolarization is dependent on a supraventricular impulse.

Several further criteria have been introduced to identify the origin of wide QRS complex tachycardia, but none of these is able to provide a simple and quick solution of the problem.

Vagal maneuvers and analysis of previous electrocardiograms recorded during sinus rhythm, if available, can provide further keys to the diagnosis. Some criteria proposed in the past, such as QRS axis or ventricular complex duration, are nowadays no longer considered; in addition, it has been demonstrated that items such as age, hemodynamic status, heart rate, and regularity of R-R intervals can be misleading and should not be taken into account.

Analysis of QRS configuration in leads V1 and V6 is a keystone in distinguishing the origin of wide QRS tachycardias: diagnostic criteria rely upon the assumption that aberration is due to a functional bundle branch block, whereas ectopy derives from a totally abnormal activation of the ventricles. Aberration, thus, results in a "typical" bundle branch block morphology, whereas ectopy is expressed by an "atypical" bundle branch block. Specific criteria, based on analysis of leads V1 and V6, have been developed to distinguish from each other the two conditions. Criteria based on QRS configuration, however, suffer from limitations since unexpected complicating factors, such as a previous myocardial infarction, can result in an "atypical" form of bundle branch block even in the presence of supraventricular tachycardia.

Recently proposed items (lead aVR analysis, peak R wave time in lead II) can be at times useful, but they need further investigation to confirm the results obtained by the authors that introduced such criteria.

6.8 Ventricular Versus Preexcited Tachycardia

In preexcited tachycardia, supraventricular impulses are conducted to the ventricles over an accessory pathway, resulting in wide QRS complexes; this is because ventricular depolarization is entirely (or, less commonly, partially) due to the impulse conducted over the abnormal conduction pathway. Such a situation may occur in the presence of (1) atrial tachycardia, (2) atrial flutter, (3) A-V nodal reentrant tachycardia, and (4) antidromic A-V reentrant tachycardia.

In all of these conditions, ventricular depolarization is due to the accessory pathway, and the QRS complexes are very wide ("pure" delta waves), mimicking a VT. It is, thus, necessary to distinguish VT from preexcited tachycardia, since at first glance QRS complex morphology suggests in the latter ventricular, rather than supraventricular, origin of the arrhythmia. In antidromic A-V reentrant tachycardia due to Mahaim fibers, however, the morphology of QRS complexes suggests supraventricular, rather than ventricular, origin of the arrhythmia, since the accessory pathway is usually connected with the right bundle branch, resulting in a typical left bundle branch block pattern [28].

Distinction between VT and preexcited tachycardia is based on the following concept: VT can originate from any part of the ventricular myocardium, whereas in preexcited tachycardia, ventricular depolarization starts (apart the rare exception represented by Mahaim fibers) from the atrioventricular rings, in correspondence of ventricular insertion of the Kent bundle. Accordingly, there is a relatively limited possible patterns, and each of them is characteristic of a defined Kent bundle

location. Since the ECG in sinus rhythm permits in most cases a reliable localization of the accessory pathways [29, 30], the question to be raised in the presence of a wide QRS complex tachycardia should be the following: "Is the morphology of this arrhythmia consistent with the typical pattern of ventricular depolarization due to an accessory pathway?" If the answer is "yes," a preexcited tachycardia is possible, whereas in the case of negative answer, such a diagnosis can be excluded.

It has been reported that in a wide QRS complex tachycardia, the following morphologies suggest VT rather than preexcitation: [4]

1. Negative QRS complexes in V4–V6
2. Negative precordial QRS concordance (negative QRS complexes in all precordial leads)
3. Deep Q waves or Qr complexes in a precordial lead except V1

Although Q waves in precordial leads other than V1 are virtually impossible in preexcitation, negative complexes in V4–V6 are not rarely observed in preexcitation due to a posteroseptal accessory pathway, since the superiorly directed vector can result in large S waves.

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