General Approach to a Wide QRS Complex

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KEYWORDS

• QRS complex • Wide complex tachycardia • Ventricular tachycardia • Supraventricular tachycardia • Aberrancy

KEY POINTS

- A wide QRS complex (≥120 ms) is present when the normal activation pattern (His–bundle branches–Purkinje) is modified.
- It is important to differentiate between ventricular tachycardia and supraventricular tachycardia conducted with aberrancy because this significantly influences the management.
- The presence of ventriculoatrial dissociation (either by identifying dissociated atrial activity or noting capture and fusion beats) is the most helpful electrocardiographic sign of ventricular tachycardia.
- The Brugada and Vereckei algorithms, which use a combination of ventriculoatrial dissociation and QRS morphology criteria, are commonly used for the differential diagnosis of wide QRS tachycardia.
- All the conditions modifying structurally the activation of the heart (pacemakers, ventricular preexcitation, severe myocardial disease, hyperkaliemia, antiarrhythmic or psychotropic drugs) should be known to provide a precise differential diagnosis of a wide QRS.

INTRODUCTION

The term "wide QRS complex" is generally used as an electrocardiographic (ECG) jargon in the presence of a QRS complex with increased duration, greater than or equal to 120 milliseconds. Although "wide QRS complex" frequently refers to a wide complex tachycardia (WCT), a wide QRS complex is present also in a single beat, and during bradycardia. Because duration is the only discriminant, it should be accurately measured on a standard 12-lead ECG, from the onset of the QRS complex to its terminal components. Indeed, measurement of the QRS duration in a single lead may frequently result in an inaccurate evaluation and, therefore, it should be avoided. The approach to an ECG showing wide QRS complexes frequently represents a great challenge in clinical practice. Wide QRS complex results from a variety of mechanisms and clinical conditions and its correct interpretation is crucial for differential diagnosis and appropriate decision making.

CAUSES OF A WIDE QRS COMPLEX

Narrow QRS complex is the ECG equivalent of a well-defined, ordered, and reproducible activation sequence of both ventricles through the His

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bundle, bundle branches, and Purkinje network. Every event that modifies this sequence results in QRS prolongation. More specifically, a wide QRS can be seen in the presence of the following causes:

- Ectopic ventricular beat/ventricular tachycardia (VT): when a beat originates in a ventricular focus, the activation wavefront spreads centrifugally from this site with a cell-to-cell propagation outside the specialized conduction tissue, producing a wide QRS complex (Figs. 1 and 2). As an exception, ectopic beats originating close to or in the bundle branches or Purkinje network show a narrower QRS complex, because a considerable part of the ventricular activation proceeds faster, over the specialized conduction tissue.
- Intraventricular conduction delay: conduction delay or block in one of the bundle branches or fascicles results in delayed activation of some regions of the ventricle, producing prolongation of the QRS complex; conduction delay is usually secondary to an anatomic/functional bundle branch block (Fig. 3). Rarely, it is an effect of drugs (most typically sodium channel blockers, such as class Ic antiarrhythmics or tricyclic antidepressants; Fig. 4) or electrolyte disturbances (eg, hyperkaliemia Fig. 5).
- Pacing: when a ventricular site (usually the right ventricle) is artificially paced, the depolarization wavefront proceeds centrifugally and slowly from the pacing site to the remaining ventricular myocardium (Fig. 6), similar to what is observed in ectopic ventricular beat. Therefore, the result of pacing is a wide QRS complex preceded by the pacing artifact. A narrower paced QRS complex is observed when the paced beat fuses with the normally conducted beat, when the pacing electrode is close to the His bundle area, or when the ventricles are simultaneously paced by two remote sites, as in biventricular pacing during cardiac resynchronization therapy.
- Ventricular preexcitation: a portion of the ventricular myocardium is activated early over an atrioventricular (AV) accessory pathway and fuses with the normal AV node-His-Purkinje activation wavefront to activate the entire ventricular myocardium (Fig. 7). The resulting fusion QRS complex is more or less wide, depending on the balance of the two AV conduction systems: the earlier the activation over the accessory pathway the wider the QRS complex.

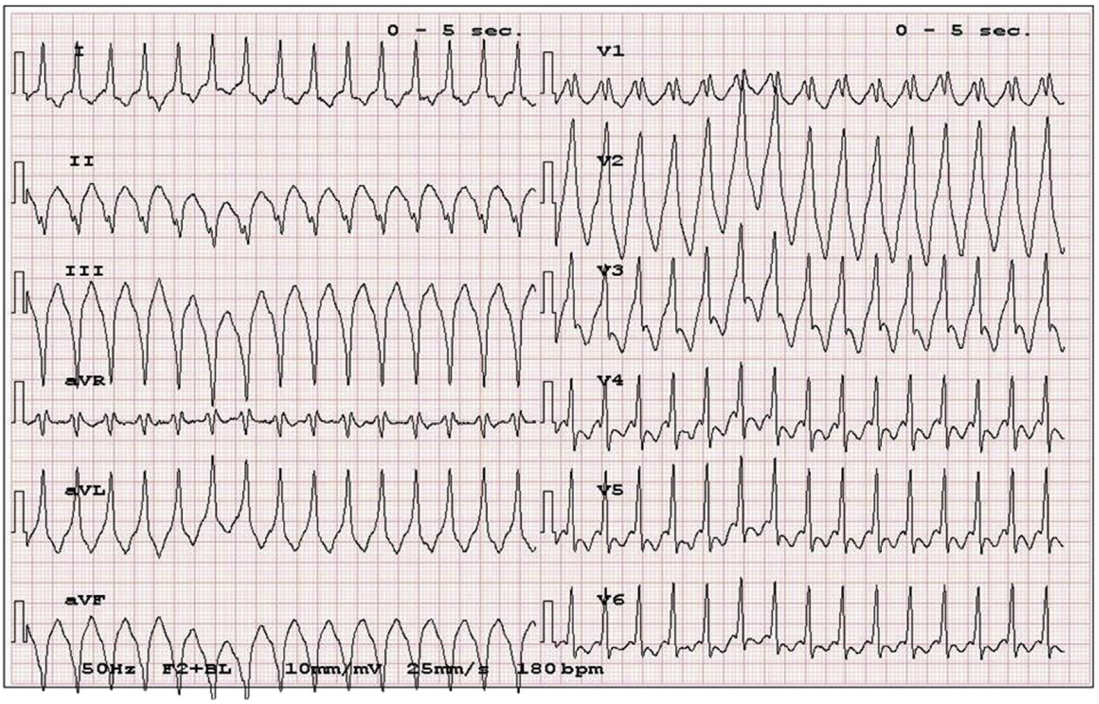

Fig. 1. Ventricular tachycardia with a right bundle branch pattern. Note the apparent "narrow" QRS complexes in the left precordial leads.