The QRS configuration during bundle branch block. What has rate got to do with it?

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This editorial refers to ‘Rate-related changes in QRS morphology in patients with fixed bundle branch block: implications for differential diagnosis of wide QRS complex tachycardia’† by T. Datino et al., on page 2351

Bundle branch block is a common cause of a widened QRS complex, and, as indicated in Table 1, different mechanisms may be responsible. Three are rate related and one is based upon retrograde invasion into the bundle branch. Those four mechanisms lead to the intermittent appearance of a bundle branch block pattern.1

Most commonly, bundle branch block is continuously present, so-called fixed bundle branch block, the patient showing a wide QRS all the time independent of the heart rate. When the heart rate is fast and the QRS wide, the challenge for the cardiologist is to differentiate bundle branch block from other causes such as ventricular pre-excitation, the use of class IC drugs leading to rate-related QRS widening, and in particular ventricular tachycardia.

Datino et al.2 describe QRS behaviour during incremental atrial pacing in 59 patients with fixed bundle branch block. In 14 of them they could also look at the QRS configuration during spontaneous supraventricular tachycardia. The question they wanted to answer was: ‘Does the bundle branch block pattern stays the same during rapid ventricular rates or do changes occur possibly leading to the incorrect diagnosis of a ventricular tachycardia?’ That is an important question because most cardiologists believe that in the patient with fixed bundle branch block the QRS does not change when the heart rate accelerates.

Datino et al.2 show that during pacing of the atrium at increasing rates, QRS changes, ranging from minor to major, were present in the majority of their patients. Changes were found in the width, voltage, and QRS configuration, occurring more commonly on increasing the atrial pacing rate. QRS changes similar to those during atrial pacing were observed when comparable ventricular rates occurred during supraventricular tachycardia, but one has to be careful here because a P wave falling in the QRS, as happens during the common type of AV nodal re-entrant tachycardia, may change the QRS configuration.

We know that under normal circumstances conduction over the bundle branch system results in early activation of the left ventricle at three sites by way of the left septal fibres, the left anterior and the left posterior branch, and early activation of the right ventricle at one site: the exit of the right bundle branch.3 Subsequent activation of the ventricles occurs by way of the Purkinje fibres. As beautifully shown by Tawara4 more than a century ago, the intraventricular conduction system is a delicate network consisting of many interconnected fibres, especially on the left side. These different fibres have different electrophysiological properties for conduction velocity and refractory period duration, and those properties may behave differently when heart rate changes occur.5,6

With this in mind, we should look at what may occur during rate acceleration in patients with a fixed block in one bundle branch. First of all, changes are more likely to occur in fixed right bundle branch block because of the subdivision of the left bundle branch into septal fibres, and a left anterior and posterior fascicle. Those changes can be based on increasing delay or block in one or more of these left-sided subdivisions.2 When during incremental atrial pacing a block takes place in the anterior or posterior fascicle, a marked shift in the QRS axis will occur. This was not observed in any of the patients with right bundle branch block in the Datino cohort; however, 17 out of the 29 already had an axis shift, because of additional block in either the left anterior or posterior fascicle. Surprisingly, none of these patients developed delay or block in the remaining conducting fascicle. This suggests that the patients studied, who, according to the Methods section, were referred for electrophysiological study, did not suffer from episodes of syncope because of sub-AV nodal block.

Delay or block in left septal fibres will not result in an important QRS axis shift but may lead to a change in the initial portion of the QRS, as was seen in several of the patients with right bundle branch block. Especially when this occurs during a rapid rhythm in a patient with right bundle branch block and left anterior fascicular block, this may wrongly suggest the presence of a ventricular tachycardia, when only the tachycardia ECG is available.

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During a rate increase, delay or block may also occur in the Purkinje tissue and ventricular muscle, for example in the presence of scar tissue or ischaemia. That may lead to changes in the mid and last part of the QRS complex.

The situation is different in left bundle branch block, when only one conducting bundle branch (the right one) is present. Rate-related QRS changes should primarily take place in Purkinje and ventricular muscle tissue. Figure 1 of the article by Datino et al. is a nice example.2

The marked QRS axis shift in the frontal plane and the clockwise rotation in the horizontal plane are caused by a rate-related delay in activation of the basolateral part of the left ventricle and the basal part of the right ventricle, indicating extensive cardiac damage with intramuscular delay.

Another mechanism which may lead to QRS changes during tachycardia is the change in ventricular dimensions during rate changes. A marked rate increase in the normal heart may result in a smaller ventricular size with changes in the QRS. In the sick heart, ventricular dilatation may occur when the heart rate increases, also possibly leading to changes in QRS configuration.

Although not mentioned by the authors, there is a situation where during both sinus rhythm and tachycardia in an identical bundle branch block pattern is present but the tachycardia is not supraventricular but rather ventricular in origin. This occurs when a patient with fixed bundle branch block during sinus rhythm develops a re-entrant tachycardia in the bundle branch system with a circuit with anterograde conduction over the non-blocked bundle branch and interventricular septal tissue, and retrograde conduction over the bundle branch which is anterogradely blocked during sinus rhythm.8

To make the correct diagnosis in a wide QRS tachycardia remains a challenge, and a 12-lead ECG during sinus rhythm of the same patient can be helpful, as is the case in the patient with fixed bundle branch block. However, as pointed out by Datino et al.,2 a ventricular rate increase may lead to QRS changes in patients with fixed bundle branch block, because of changes in the pattern of ventricular activation. Rate-related changes in conduction velocity and refractory period duration in the bundle branch–Purkinje system and ventricular muscle, and rate-related changes in ventricular dimensions are responsible.

The conclusion is that in patients with the same type of bundle branch block during sinus rhythm and tachycardia, there may be differences in QRS configuration without implying that the tachycardia is ventricular. An important point, as also indicated elsewhere, is that a tachycardia with a bundle branch block shape contralateral to the type of bundle branch block during sinus rhythm always has its origin in the ventricle.

Conflict of interest: none declared.

References

Table I Types and mechanisms of bundle branch (BB) block

<table>
<thead>
<tr>
<th>Occurrence</th>
<th>Mechanism</th>
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<tbody>
<tr>
<td>INTERMITTENT BUNDLE</td>
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<td>BRANCH BLOCK</td>
<td></td>
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<tr>
<td>At slow rates</td>
<td>Phase 4 block in BB</td>
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<td>At intermediate rates</td>
<td>Acceleration-dependent BB, because of diminished excitability of the BB</td>
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<td>resulting in block at rates well beyond the duration of the action potential of the BB</td>
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<td>At fast rates</td>
<td>Phase 3 block on reaching the refractory period of the BB</td>
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<tr>
<td>At normal or fast</td>
<td>Retrograde invasion in the BB: anterograde block in the BB by retrograde penetration in to that BB by an impulse conducted over the contralateral BB</td>
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<td>rates</td>
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<tr>
<td>FIXED BUNDLE BRANCH</td>
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<tr>
<td>BLOCK</td>
<td></td>
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<tr>
<td>At all rates</td>
<td>Complete block or marked delay in conduction in a BB leading to complete ventricular activation over the contralateral BB</td>
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