Alteration of human right bundle branch refractoriness by changes in duration of the atrial drive train

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ABSTRACT We studied the effect of drive train duration on human right bundle branch refractoriness. Seven patients with a QRS duration of 0.10 sec or less and no preexcitation had functional right bundle branch block induced reproducibly with premature atrial stimulation. Refractoriness of the right bundle branch was defined as the longest \( H_1H_2 \) interval resulting in right bundle branch block and was determined in each patient at a constant pacing cycle length but at five to seven different atrial drive train durations varying from four to 99 complexes. In all seven patients, right bundle branch refractoriness decreased with increasing drive train duration (mean 15 msec). One-third of this decrease (5.0 msec) occurred between drive train durations of four and eight complexes, and close to two-thirds (9.3 msec) occurred between drive train durations of eight and 64 complexes. Very little further decrease (0.7 msec) occurred between 64 and 99 complexes. We conclude that right bundle branch refractoriness shortens progressively as the preceding drive train duration increases. This phenomenon may in part explain the disappearance of functional right bundle branch block during supraventricular tachycardia after a variable number of complexes without a change in cycle length of tachycardia.


REFRACTORINESS of various cardiac tissues is a function of the drive train cycle length at which the tissue is paced, as noted in animal\(^1\)–\(^4\) and human\(^5\)–\(^8\) atrial, atroventricular nodal, His-Purkinje, and ventricular tissue. The refractory period may also be altered by sudden changes in drive train cycle length before the premature impulse is delivered.\(^2\)–\(^4\), \(^7\), \(^8\) For example, Wiener et al.\(^7\) showed in human ventricular tissue that termination of a drive train by a single complex at a shorter interval produced 60% of the shortening of refractoriness produced by eight complexes at the identical shorter interval. However, Janse et al.\(^1\) demonstrated in canine ventricular tissue that although a sudden increase or decrease in heart rate changed the duration of refractoriness immediately, the steady-state value of refractoriness at the new heart rate was achieved only after a few hundred complexes, implying that not only the cycle length of the drive train but also its duration is important in determining the refractoriness of cardiac tissue. The purpose of this investigation was to test the hypothesis that prolongation of drive train duration shortens human right bundle branch refractoriness.

Methods

Four men and three women underwent control electrophysiologic study in the absence of antiarrhythmic drug therapy (table 1). The mean age was 45 years with a range of 32 to 56. Each patient had a QRS duration of 0.10 sec or less with no evidence of ventricular preexcitation but had functional right bundle branch block reproducibly initiated during premature atrial stimulation.

All patients underwent a standard electrophysiologic study after giving written and oral informed consent.\(^9\) Two or three multipolar electrode catheters were introduced percutaneously into the femoral vein and positioned under fluoroscopic guidance in the high right atrium, across the tricuspid valve in the region of the His bundle, and in the right ventricle. Intracardiac recordings filtered at 30 to 500 Hz and standard electrocardiographic leads I, II, III, and \( V_1 \) filtered at 0.1 to 20 Hz were displayed simultaneously on a multichannel oscilloscope (Electronics for Medicine VR-12) and recorded at paper speeds of 100 mm/sec. High right atrial pacing was performed with a custom-built programmable stimulator using 2 msec rectangular pulses at twice late diastolic threshold. For each patient, after establishing that functional right bundle branch block reproducibly occurred at a critical atrial premature coupling interval during a standard drive train duration of eight complexes, the refractoriness of the right bundle branch was determined with the use of five to seven different atrial drive train durations.
varying from four to 99 complexes. The cycle lengths of the drive trains for all patients varied from 600 to 900 msec but cycle length was constant for each patient. At each atrial drive train duration (S₁) the premature atrial stimulus (S₂) was decreased by 5 msec until right bundle branch block appeared. A 1 min rest period was allowed between each atrial drive train run. The right bundle branch refractory period was defined as the longest H₁H₂ interval resulting in right bundle branch block.

The decrease in right bundle branch refractoriness with increasing drive train duration was analyzed by repeated-measures analysis of variance.

Results

Figure 1 illustrates analog data from a patient who had a 15 msec decrease in right bundle branch refractoriness when the atrial drive train was increased from four to 64 complexes. Surface lead V₁ and intracardiac high right atrial and His bundle electrograms are displayed for atrial drive train durations of four, eight, 32, and 64 complexes. On the left are the premature intervals that resulted in narrow QRS complexes, and on the right the premature intervals that resulted in right bundle branch block. At each drive train duration illustrated, the longest H₁H₂ interval that resulted in a right bundle branch block progressively lessened by 5 msec.

Figure 2 illustrates graphically the refractory period of the right bundle branch vs atrial drive train duration for each of the seven patients. All patients demonstrated a decrease in right bundle branch refractoriness as atrial drive train duration increased; four patients had relatively flat curves and three patients relatively steep curves. The range of shortening of right bundle branch...
refractoriness was 5 to 30 msec, with a mean of 15. Note that the two patients with the greatest change in right bundle branch refractoriness were the two with the greatest difference between spontaneous sinus cycle length and atrial pacing cycle length (table 1).

Figure 3 illustrates mean cumulative shortening of right bundle branch refractoriness with increasing atrial drive train duration for the seven patients. The shortening of the right bundle branch refractory period is illustrated on the ordinate and the interval of atrial drive train duration over which this shortening occurred appears on the abscissa. The unfilled bars with numbers inside illustrate the decrease in right bundle branch refractory period for each change in drive train duration, and the filled bars represent the cumulative change in refractoriness. Note that right bundle branch refractoriness shortened progressively as the drive train duration increased, but only one-third of the 15 msec mean shortening occurred between drive train durations of four and eight complexes (5.0 msec), whereas almost two-thirds of the shortening occurred between drive train durations of eight and 64 complexes (9.3 msec). Little additional change in right bundle branch refractoriness occurred between 64 and 99 complexes. The overall decrease in right bundle branch refractoriness with increasing drive train dura-

**TABLE 1**

<table>
<thead>
<tr>
<th>Patient No.</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Structural heart disease</th>
<th>Clinical arrhythmia</th>
<th>SCL</th>
<th>APCL</th>
<th>ΔERP</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>41</td>
<td>M</td>
<td>None</td>
<td>VF</td>
<td>1030</td>
<td>600</td>
<td>25</td>
</tr>
<tr>
<td>2</td>
<td>52</td>
<td>M</td>
<td>CAD</td>
<td>Syncope, VT-NS</td>
<td>950</td>
<td>900</td>
<td>5</td>
</tr>
<tr>
<td>3</td>
<td>32</td>
<td>M</td>
<td>None</td>
<td>Syncope</td>
<td>880</td>
<td>700</td>
<td>30</td>
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<tr>
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<td>50</td>
<td>F</td>
<td>MVP</td>
<td>Syncope</td>
<td>700</td>
<td>600</td>
<td>10</td>
</tr>
<tr>
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<td>38</td>
<td>F</td>
<td>None</td>
<td>Syncope, VT-S</td>
<td>900</td>
<td>800</td>
<td>10</td>
</tr>
<tr>
<td>6</td>
<td>44</td>
<td>F</td>
<td>None</td>
<td>Syncope</td>
<td>850</td>
<td>800</td>
<td>15</td>
</tr>
<tr>
<td>7</td>
<td>56</td>
<td>M</td>
<td>CAD</td>
<td>VT-NS</td>
<td>980</td>
<td>850</td>
<td>10</td>
</tr>
</tbody>
</table>

SCL = sinus cycle length (msec); APCL = atrial pacing cycle length (msec); ΔERP = decrease in right bundle branch refractoriness with increased drive train duration (msec); CAD = coronary artery disease; MVP = mitral valve prolapse; VF = ventricular fibrillation; VT-S = sustained ventricular tachycardia; VT-NS = nonsustained ventricular tachycardia.
tion was significant ($p < .001$), but the decrease occurring at drive train durations longer than 32 complexes did not reach statistical significance ($p = .08$).

**Discussion**

It has been shown previously that human His-Purkinje refractoriness shortens with faster atrial pacing rates, but this study shows for the first time that the magnitude of decrease in refractoriness depends on the duration of the drive train at the newly established rate. Thus, several complexes at the shorter cycle length appear to be required for the maximum change in the effective refractory period to occur.

Previous investigations in a variety of cardiac tissues showed conflicting results in the number of beats required to reach steady-state refractoriness. Mendez et al. concluded that refractoriness varied only with the one preceding pacing interval, whereas Han and Moe described a cumulative change in refractoriness after the onset of pacing. Janse et al. demonstrated in canine ventricle that a sudden increase in pacing rate shortened refractoriness immediately, but the “minimum” steady-state refractoriness often required a few hundred beats to be established. Our study demonstrated that there was progressive shortening of human right bundle branch refractoriness as the atrial drive train duration preceding refractory period determination was increased from four to up to 99 complexes. Only one-third of the overall refractory period shortening observed in this study occurred between drive train durations of four and eight complexes, whereas two-thirds occurred when the drive train duration was greater than eight complexes. However, drive train durations greater than 32 complexes resulted in minimal further shortening of right bundle branch refractoriness. In this regard it is interesting to note the similarity of our results in His-Purkinje tissue to those of Janse et al. in ventricular tissue, in which 40% to 60% of the total refractoriness shortening occurred within the first 20 beats, and close to 80% occurred within 60 beats. Note that much of the shortening observed in this study occurred at drive train durations longer than eight complexes, the usual drive train duration used to establish refractory periods in the clinical electrophysiology laboratory. This study was not designed to determine the total change in right bundle branch refractoriness after a sudden increase in heart rate since drive train durations less than four complexes were not tested.

Some patients had small changes in right bundle branch refractoriness with increasing drive train duration, whereas others demonstrated more dramatic decreases. Although this may be attributed in part to differences among patients, it may also be related to the difference between spontaneous sinus rate and atrial pacing rate; that is, the greater the difference, the greater total steady-state decrease in refractoriness one may expect. In our study, we were limited to relatively slow atrial pacing cycle lengths in order to preserve the ability to induce functional right bundle branch block with premature stimulation. The patients with the greatest shortening of refractoriness (patients 1 and 3) were those with the greatest difference between spontaneous and paced atrial rates. One may postulate that with minimal differences in sinus and atrial pacing cycle lengths, the heart would sense little rate change.
and shortening of refractoriness would be small. On the other hand, with a greater change in cycle length the heart would recognize a distinctly new rate and one would expect refractoriness to require a greater amount of time to accommodate.

The most likely mechanism for the shortening of right bundle branch refractoriness noted in this study is a progressive decrease in action potential duration during atrial pacing. Carmeliet studied the transmembrane action potential of the frog ventricle and noted that pacing up to 40 or 50 complexes after a change in cycle length was necessary to achieve the maximum change in action potential duration. Another mechanism for the shortened refractory periods might be an increase in sympathetic tone brought about by the long atrial paced drive trains. We think this is unlikely because (1) it would not explain the changes in refractoriness at the shorter drive train durations, and (2) the pacing cycle lengths are all relatively slow and the longer runs are not associated with blood pressure changes. Of note, previous data from our laboratory evaluating ventricular refractoriness showed no effect of propranolol on ventricular effective refractory periods at drive train durations of 60 or more complexes.

Our observations on the relationship of drive train duration and right bundle branch refractoriness may explain in part the mechanism for the disappearance of functional right bundle branch block that is often present at the initiation of supraventricular tachycardia but that resolves shortly thereafter. For example, in patient 1 from this study the initiation of atrial pacing mimicked the onset of a supraventricular tachycardia (figure 4). The occurrence of right bundle branch block of the first "tachycardia" complex can be explained by a relatively long right bundle branch refractoriness due to the preceding slow heart rate. However, as the simulated tachycardia continues, right bundle branch block is present for seven complexes but then normalizes with no measurable change in the HH intervals. One explanation for persistence of right bundle branch aberrancy is concealed transseptal conduction; that is, the right bundle branch distal to the site of block is activated late within each QRS complex via transseptal conduction from the left, and thus the subsequent "right bundle to right bundle" interval is shorter than the manifest RR interval. Although this seems a likely explanation when right bundle branch block does not resolve at a new faster heart rate, it is unclear why this example concealed transseptal conduction should suddenly fail within a few beats, leading to QRS normalization. An alternative mechanism for the subsequent disappearance of right bundle branch block is accommodation of right bundle branch refractoriness with increasing drive train duration at a new rate (i.e., tachycardia), as demonstrated in this study. Thus, after the first right bundle branch block complex upon initiation of tachycardia, aberration may persist because of relatively long right bundle branch refractoriness (with or without a contribution from concealed transseptal conduction); as tachycardia continues, refractoriness gradually shortens and the QRS normalizes.

We conclude that right bundle branch refractoriness shortens progressively as the preceding drive train duration increases. This change in right bundle branch refractoriness with drive train duration may in part explain the change from right bundle branch block to normal QRS configuration during supraventricular tachycardia.

We thank Debbie Carter for her secretarial assistance in the preparation of this manuscript.

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Alteration of human right bundle branch refractoriness by changes in duration of the atrial drive train.

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Circulation. 1986;73:244-248
doi: 10.1161/01.CIR.73.2.244

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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