The Effect of Intermittent Bundle Branch Block on the Coupling Interval of Ventricular Premature Depolarizations

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SUMMARY The mechanism of origin of ventricular premature depolarizations (VPDs) is explained by automaticity or reentry. We studied three patients with intermittent left bundle branch block (LBBB) and unifocal VPDs of right bundle branch block (RBBB) morphology (assumed left ventricular origin). We examined the effect of different types of intraventricular conduction on the coupling intervals of the VPDs. Specifically, we proposed that the coupling intervals of VPDs would be longer during LBBB conduction of sinus beats (ipsilateral to the ventricle of origin of VPDs) compared with the coupling intervals during normal intraventricular conduction. We found that the coupling intervals during LBBB were significantly longer than those during normal conduction in all three cases (patient 1, 596 ± 7 vs 484 ± 5 msec; patient 2, 639 ± 9 vs 534 ± 11 msec; patient 3, 444 ± 4 vs 382 ± 9 msec) (p < 0.005). We also examined the length of the preceding RR intervals and counted sinus beats intervening between successive VPDs (S values). One case demonstrated S values suggestive of concealed bigeminy; the other cases had S values suggesting concealed bigeminy and its variants. We have demonstrated that some VPDs are dependent on an initiating sinus beat. This dependence on a preceding beat is consistent with both a reentrant mechanism or triggered automaticity.

Methods
Twenty-four-hour, two-channel ambulatory electrocardiographic recordings were obtained using Del Mar Avionics models 445A and 445B Electrocardiographs. Scanning of the tapes was done with a Del Mar Avionics Electrocardioscanner model 660B. Tapes were scanned by technicians for the presence of VPDs. All selected rhythm strips were used for analysis. The paper speed was 25 mm/sec. Intervals were measured by the authors, using calipers, with an accuracy of 20 msec. Modified electrocardiographic leads $V_1$ and $V_2$ were used. Leads $V_1$ and $V_2$ were placed at the fourth right intercostal space parasternally, and at the fifth intercostal space at the anterior axillary line, respectively. The QRS morphology of VPDs was considered to be RBBB morphology if positive in lead $V_1$. Standard electrocardiographic criteria were used for diagnosis of LBBB. Statistical analysis was done using a $t$ test. The S values were examined by counting the number of sinus beats intervening between successive VPDs. When the numbers of intervening sinus beats (S values) are equal to $2n-1$ (n = any positive integer), concealed bigeminy is suggested. S values of $3n-1$ suggest the presence of concealed trigeminy.

Results
Two patients had rate-dependent phase 3 bundle branch block; one had non–rate-dependent intermittent bundle branch block. All three had VPDs with upright QRS complexes in $V_1$. All three were in sinus rhythm. Analysis of rhythm strips did not show evidence of ventricular parasystole (i.e., variable coupling with fusion beats and interectopic intervals that are multiples of a common denominator) in any of the cases. The range and mean of coupling intervals of VPDs and the preceding RR intervals during normal intraventricular conduction and LBBB for all three patients are shown in table 1. The coupling intervals of VPDs were significantly longer during LBBB conduction when
TABLE 1. Electrophysiologic Data

<table>
<thead>
<tr>
<th>Type of conduction</th>
<th>Preceding RR interval (range, msec)</th>
<th>Preceding RR interval (mean ± SEM, msec)</th>
<th>Coupling interval (range, msec)</th>
<th>Coupling interval (mean ± SEM, msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient 1</td>
<td>Normal</td>
<td>800–1000</td>
<td>896 ± 20*</td>
<td>460–530</td>
</tr>
<tr>
<td></td>
<td>LBBB</td>
<td>680–840</td>
<td>800 ± 15</td>
<td>540–600</td>
</tr>
<tr>
<td>Patient 2</td>
<td>Normal</td>
<td>640–740</td>
<td>696 ± 11†</td>
<td>480–600</td>
</tr>
<tr>
<td></td>
<td>LBBB</td>
<td>600–800</td>
<td>705 ± 13</td>
<td>560–720</td>
</tr>
<tr>
<td>Patient 3</td>
<td>Normal</td>
<td>600–1040</td>
<td>815 ± 17*</td>
<td>320–400</td>
</tr>
<tr>
<td></td>
<td>LBBB</td>
<td>580–800</td>
<td>693 ± 9</td>
<td>420–480</td>
</tr>
</tbody>
</table>

* p < 0.005.
† NS.
Abbreviation: LBBB = left bundle branch block.

compared to the coupling intervals during normal intraventricular conduction. This difference in the duration of coupling intervals did not relate to the length of the preceding RR intervals (figs. 1 and 2). S values were counted, and in case 3, they were consistent with concealed bigeminy. The other two cases showed S values that suggested concealed bigeminy and its variants.11,12

Discussion

The dependence of some VPDs upon the preceding sinus impulse has been recognized since early in this century.1 In the present report, a similar phenomenon is described in a clinical situation. The three patients had VPDs with upright configuration in lead V1, suggesting left ventricular origin.10 During intermittent LBBB, the coupling interval is prolonged, again presumably owing to the longer time required for the sinus impulse to reach the site of initiation of the VPDs.

The mechanism of supraventricular tachycardia (SVT) is better understood. Specific criteria have been developed to demonstrate the presence of a reentrant pathway.12 One of these relates to the effect of ipsilateral bundle branch block on the rate of SVT in the presence of an atrioventricular bypass tract.12-14

The cycle length of SVT is prolonged in the presence of
intermittent bundle branch block (ipsilateral to the bypass tract) compared with the cycle length of SVT during normal conduction. This lengthening of the cycle during bundle branch block is explained by the additional time required for the antegrade impulse to traverse the septum and return to the bypass tract to complete the retrograde limb of the circuit. This time interval has been described as the "transseptal conduction time." In the cases of the present report, as well as in the earlier experimental work of Scherf, the lengthening of coupling intervals of left ventricular VPDs during LBBB is analogous to the prolongation of the cycle length of reentrant SVT during ipsilateral bundle branch block. In our cases, the differences in

![Figure 2. Coupling intervals of ventricular premature depolarizations and preceding RR intervals of sinus beats. LBBB = left bundle branch block.](image)

![Figure 3. Proposed mechanism for ventricular premature depolarizations. (A) Normal atrioventricular conduction. The impulse reaches the reentrant pathway through the left bundle branch and Purkinje system. (B) Left bundle branch block. The impulse must traverse the myocardium from the right to left ventricle before entering the reentrant pathway. This transseptal conduction time causes a delay that is manifested as a prolonged coupling interval. An exact location of the reentrant pathway is not being proposed, but it is presumed to exist somewhere within the left ventricular myocardium. The presence of a triggered automatic focus rather than a reentrant pathway cannot be excluded.](image)
coupling intervals of VPDs during normal conduction and LBBB varied from 58 to 98 msec, which is consistent with the previously described transseptal conduction time in man. Figure 3 shows a proposed mechanism based on VPDs being reentrant in nature; however, we cannot exclude triggered automaticity (impulse formation from an ectopic focus) as an alternative mechanism of the arrhythmia. Since this was a retrospective study, no physiologic or pharmacologic maneuvers were performed to differentiate between the two mechanisms. The use of a slow-channel blocker, such as verapamil, in these patients may be helpful in differentiating triggered automaticity, which is slow-channel-dependent, from reentry.

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