His Bundle Recordings in Patients with Bundle Branch Block and Transient Neurologic Symptoms

By Melvin Scheinman, M.D., Alan Weiss, M.D., and Frederick Kunkel, M.D.

SUMMARY
His bundle electrograms were recorded in 19 patients with bundle branch block (BBB) and transient neurologic symptoms allowing for determination of intra-atrial (P-A), atrioventricular (A-V), nodal (A-H) as well as infranodal conduction (H-Q) times. The patients were initially monitored in a coronary care unit and have been followed for a mean period of 8 ± 4 months. In six patients (Group I) neurologic symptoms were observed in the absence of electrocardiographic evidence of A-V block. In six patients (Group II) the cause of symptoms was uncertain; two of these patients had relief of symptoms after permanent cardiac pacemaker insertion and were presumed to have episodic high grade A-V block. In seven subjects (Group III) complete A-V block was documented as the cause of the symptoms; these patients were studied when 1:1 antegrade A-V conduction returned. There was no significant difference between mean P-A, A-H, and QRS durations among the patients in the three groups. Mean H-Q (89 ± 20 msec) for Group III was significantly longer than that for Group I (56 ± 9 msec) or Group II (64 ± 11 msec) (P < .001). All patients with presumed or documented episodes of high grade A-V block had abnormal H-Q intervals, and six of the nine patients with presumed or documented complete A-V block had H-Q intervals ≥80 msec. The present data suggest that patients with transient neurologic symptoms, bifascicular or left BBB associated with marked prolongation of H-Q (≥80 msec), should be seriously considered as candidates for insertion of a permanent cardiac pacemaker even in the absence of documented high grade or complete A-V block.

Additional Indexing Words:
Bifascicular block  A-V conduction disorders  Syncope  Seizures  Dizziness

A WEALTH of anatomic and clinical data support the contention that patients with the electrocardiographic findings of bundle branch block (BBB) (particularly partial bilateral bundle branch block) have an increased risk of developing complete atroventricular (A-V) block.1-7 Transient complete A-V block may occur in patients with BBB and result in transient neurologic symptoms. On the other hand, it is well appreciated that the BBB pattern is a not uncommon, oftentimes incidental, electrocardiographic finding and that higher degrees of A-V block may never develop in patients with this pattern. Moreover, the major neurologic symptoms associated with high grade A-V block (i.e., dizziness, syncope and seizures) may be unrelated to any cardiac arrhythmia or conduction disturbance. The clinician is frequently uncertain whether or not to ascribe these symptoms in patients with BBB to episodic complete A-V block and whether or not to recommend insertion of a permanent intracardiac pacemaker.

The ability to record His bundle potentials provides an important tool for localization and perhaps for quantification of A-V conduction disturbances. The present study is part of an ongoing cooperative prospective study of patients with partial bilateral BBB. The objective of this study is to define the value of His bundle recordings in identifying a subset of patients with BBB who have a great risk for the development of high grade A-V block or sudden death.

Materials and Methods
Over a period of ten months, 19 patients with BBB and symptoms of dizziness, seizures and/or syncope were studied. The only requirements for inclusion in the present study were 1) presence of BBB, 2) history of

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symptoms of dizziness, seizures or syncope, and 3) presence of 1:1 antegrade A-V conduction at the time of the His bundle study. Patients with acute myocardial infarction, however, were excluded. All patients were initially monitored from 3 to 7 days in the Coronary Care Unit and were followed at monthly intervals for 2 to 15 months following discharge from the hospital. A 12-lead electrocardiogram and a 60 sec rhythm strip were obtained during each follow-up visit. Subsequent to discharge from the hospital, all patients who did not undergo permanent cardiac pacemaker insertion underwent continuous 12 hour electrocardiographic monitoring (Holter System) on at least one occasion. Most patients were evaluated by a neurologist and further neurologic evaluation was obtained when indicated. The subjects were divided into three groups: Group I consisted of patients whose symptoms appeared to be unrelated to episodic A-V block; Group II was comprised of patients in whom the etiology of the neurologic symptoms was unclear; and Group III consisted of patients whose symptoms were clearly related to documented transient bouts of A-V block.

Recording of His bundle electrograms was performed according to the method of Damato et al. In brief, a hexapolar electrode catheter with electrodes set 10 mm apart was plugged into a six channel distribution switch box permitting recordings of any combination of bipolar leads. The junction box was connected to an Electronics for Medicine recorder with frequency response set at 40-500 cycles/sec and recordings made at a paper speed of 100 mm/sec. The electrode catheter was inserted into a femoral vein, advanced under fluoroscopic control into the right atrium and positioned across the tricuspid valve. The catheter was then manipulated until a discrete spike was displayed between prominent atrial and ventricular electrograms. It was then slowly withdrawn across the tricuspid valve, and the narrowest interval between the atrial electrogram and His bundle depolarization was recorded. The inflow portion of the right ventricle was similarly explored in order to record right bundle branch potentials (fig. 1). During the initial phase of our study, simultaneous His bundle electrograms (HBE) and precordial lead V1 were recorded in seven patients. During the last 6 months of the study, simultaneous HBE and scalar X, Y, and Z of the Frank lead system were recorded in 12 subjects. In 32% of studies, initial forces were first detected in leads X and Y of the Frank lead system. The difference between onset of ventricular activation in lead X or Y or Z compared with V1 ranged between 0 and 8 msec (unpublished observations). In view of the small differences it was felt acceptable to analyze the initial seven studies together with the latter 12. The A-H interval was measured from the initial rapid deflection of the atrial electrogram to the onset of the His bundle spike and was used as a measure of A-V nodal conduction time. The H-Q interval was measured from the onset of the His deflection to the earliest onset of ventricular activation as recorded in either lead V1 or the scalar X, Y, and Z leads and interpreted to reflect conduction in the His-Purkinje system. The scalar X, Y, and Z leads were introduced to define more precisely the earliest onset of ventricular activation. In our laboratory, the normal A-H time is 70 to 120 msec and the normal H-Q interval is 35 to 55 msec.

Our criteria for diagnosis of BBB and bilateral BBB have been reported previously. In brief, complete left BBB and right BBB were diagnosed according to the criteria of Goldman. Patients with incomplete right BBB or left BBB were excluded from the present study. Left anterior hemiblock was diagnosed when the electrocardiogram showed initial forces directed to the right (narrow q waves in leads I or aVL) and inferiorly (small r waves in leads II, III and aVF), and terminal forces directed superiorly and to the left with mean frontal plane QRS axis between \(-30^\circ\) and \(-90^\circ\).

![Figure 1](link)

*Simultaneous X, Y, and Z leads of the Frank system, right ventricular (RVE) and His bundle electrogram (HBE) showing His (H) and right bundle branch (RB) depolarizations.*
Electrocardiograms with superior axis due to deep Q waves in the inferior leads were not included in the left anterior hemiblock group. Left posterior hemiblock was diagnosed when the electrocardiogram showed small initial forces oriented superiorly and to the left, and terminal forces pointing to the right and inferiorly, with frontal plane QRS axis $> 120^\circ$. Patients with clinical evidence of pulmonary hypertension or lateral wall infarction were not included in the left posterior hemiblock group.

Informed consent was obtained from each patient prior to the study and the study protocol was approved by the University Committee on Human Experimentation.

Results

Nineteen patients with transient neurologic symptoms and electrocardiographic evidence of BBB were studied by means of His bundle electrograms and were followed for a mean ±SD time of 8 ± 3.7 months. These patients were further subdivided into three groups.

Group I

Group I consisted of six patients whose symptoms did not appear to be related to episodic high grade A-V block in the sense that they were observed to have symptoms in the absence of electrocardiographic evidence of worsening A-V block. The etiology of their symptoms as well as the results of the His bundle study are detailed in table I. Two patients (E.Y. and H.J.) showed prolonged A-V nodal conduction times while three patients showed abnormal infranodal conduction times. None of the patients had evidence of higher degree A-V block during the initial period of monitoring in the Coronary Care Unit nor did they show any progression of A-V block on repeated follow-up visits. This group has been followed for a mean time of 8.3 ± 3.2 months.

Group II

Group II (table I) consisted of six patients in whom the cause of the symptoms was unclear. All of these patients except one (A.R.) showed normal A-H but prolonged H-Q intervals. In three patients (A.R., D.G. and L.R.) symptoms were of sufficient concern to merit implantation of a permanent cardiac pacemaker; in two of these patients (L.R. and D.G.) the neurologic symptoms disappeared after insertion of the pacemaker. One patient, J.L., died of severe congestive heart failure 6 months following the study but never showed higher degrees of A-V block either during follow-up or in the terminal phase of his disease. The remaining two patients have been followed for a mean of 11 ± 6.4 months and have not shown evidence of increased A-V block on follow-up evaluation.

Group III

Group III (table I) consisted of seven patients whose symptoms were caused by episodic complete A-V block. Five patients showed complete A-V block on the admitting electrocardiogram but were studied several days later after return of 1:1 antegrade conduction. The remaining two patients showed episodes of complete heart block from 24 to 72 hours after the study. During complete heart block, the electrocardiogram showed a broad QRS with heart rate from 28-42 beats/min in five patients (fig. 2) while two patients had sinus rhythm with ventricular asystole (fig. 3). His bundle electrograms showed abnormal prolongation of the H-Q interval in all patients in this group while prolonged A-V nodal conduction was also documented in two patients. All patients in Group III underwent insertion of a permanent pacemaker; three of these patients (L.J., J.G. and C.S.) died of severe underlying heart disease within 14 months.

For the group as a whole, H-Q was prolonged in all patients with documented episodes of complete A-V block (Group III) and in the two patients whose symptoms disappeared after pacemaker insertion (patients D.G. and L.R., Group II). Furthermore, six of the nine patients who had either documented (Group III) or presumptive evidence (patients D.G. and L.R., Group II) of episodic A-V block had H-Q intervals of 80 msec or greater. The mean H-Q (±SD) interval of 89 ± 20 for patients in Group III was significantly greater than the mean H-Q intervals for either Groups I or II ($P < 0.025$), but there was no significant difference in mean H-Q interval between Groups I and II. Mean A-H intervals were not significantly different among the three groups, nor was there a significant difference in the QRS duration or in the interval from onset of the P wave of the surface electrogram to the onset of the atrial electrogram recorded in the His bundle electrogram among the three groups.

Discussion

The important therapeutic implications of the present study demand critical review of the technique we used for His bundle recording. Unfortunately, no universally acceptable method for validation of the His bundle spike is currently available. The technical limitations of both atrial and direct His bundle electrical pacing for differen-
tiating His from right bundle branch depolarizations have recently been discussed.13-16 We believe that the His spike was, in fact, recorded in the present report because prominent atrial electrograms were inscribed in the His bundle recordings18 as the multipolar electrode was carefully withdrawn across the tricuspid valve. Although unlikely, if on occasion right bundle branch rather than His bundle depolarizations were recorded, then, in fact, the true H-Q interval was underestimated and

**Table 1**

*Clinical and Electrophysiologic Data in 19 Patients with BBB and Transient Neurologic Symptoms*

<table>
<thead>
<tr>
<th>Patient</th>
<th>Symptoms</th>
<th>Cause of symptoms or cardiac diagnosis</th>
<th>Electrocardiogram*</th>
<th>P-P (msec)</th>
<th>P-R (msec)</th>
<th>A-H (msec)</th>
<th>H-Q (msec)</th>
<th>Length of follow-up (months)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Group I</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>E.Y.</td>
<td>syncope</td>
<td>cerebrovascular insufficiency</td>
<td>RBBB + LAH</td>
<td>930</td>
<td>240</td>
<td>160</td>
<td>50</td>
<td>5</td>
</tr>
<tr>
<td>Y.W.</td>
<td>syncope</td>
<td>cerebrovascular insufficiency</td>
<td>RBBB + LAH</td>
<td>770</td>
<td>175</td>
<td>85</td>
<td>50</td>
<td>13</td>
</tr>
<tr>
<td>L.B.</td>
<td>seizures, syncope</td>
<td>acute alcoholic intoxication</td>
<td>RBBB + LAH</td>
<td>710</td>
<td>198</td>
<td>95</td>
<td>65</td>
<td>11</td>
</tr>
<tr>
<td>F.J.</td>
<td>seizures, syncope</td>
<td>acute alcoholic intoxication</td>
<td>RBBB + LAH</td>
<td>950</td>
<td>210</td>
<td>95</td>
<td>58</td>
<td>8</td>
</tr>
<tr>
<td>P.D.</td>
<td>dizziness</td>
<td>hyperventilation</td>
<td>RBBB + LAH</td>
<td>670</td>
<td>190</td>
<td>100</td>
<td>45</td>
<td>8</td>
</tr>
<tr>
<td>H.J.</td>
<td>syncope</td>
<td>orthostatic hypotension</td>
<td>LBBB</td>
<td>848</td>
<td>258</td>
<td>170</td>
<td>70</td>
<td>5</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td></td>
<td></td>
<td></td>
<td>813</td>
<td>219</td>
<td>118</td>
<td>56</td>
<td>8.33</td>
</tr>
<tr>
<td><strong>Group II</strong></td>
<td></td>
<td></td>
<td></td>
<td>115</td>
<td>31</td>
<td>37</td>
<td>10</td>
<td>3.20</td>
</tr>
<tr>
<td>T.W.</td>
<td>dizziness</td>
<td>hypertensive cardiovascular disease</td>
<td>RBBB + LAH</td>
<td>880</td>
<td>192</td>
<td>80</td>
<td>58</td>
<td>15</td>
</tr>
<tr>
<td>A.R.</td>
<td>syncope</td>
<td>unknown</td>
<td>LBBB</td>
<td>720</td>
<td>150</td>
<td>90</td>
<td>50</td>
<td>2 p</td>
</tr>
<tr>
<td>A.U.</td>
<td>dizziness</td>
<td>cardiomyopathy</td>
<td>RBBB + LAH</td>
<td>685</td>
<td>217</td>
<td>87</td>
<td>62</td>
<td>6</td>
</tr>
<tr>
<td>J.L.</td>
<td>dizziness</td>
<td>rheumatic mitral valve disease</td>
<td>LBBB</td>
<td>760</td>
<td>195</td>
<td>106</td>
<td>66</td>
<td>9 (died)</td>
</tr>
<tr>
<td>D.G.†</td>
<td>syncope</td>
<td>unknown</td>
<td>LBBB</td>
<td>920</td>
<td>210</td>
<td>110</td>
<td>60</td>
<td>5 p</td>
</tr>
<tr>
<td>L.R.‡</td>
<td>seizures</td>
<td>unknown</td>
<td>RBBB + LAH</td>
<td>810</td>
<td>216</td>
<td>100</td>
<td>80</td>
<td>10 p</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td></td>
<td></td>
<td></td>
<td>779</td>
<td>198</td>
<td>96</td>
<td>64</td>
<td>11</td>
</tr>
<tr>
<td><strong>Group III</strong></td>
<td></td>
<td></td>
<td></td>
<td>92</td>
<td>28</td>
<td>11.7</td>
<td>11</td>
<td>6.4</td>
</tr>
<tr>
<td>L.J.§</td>
<td>dizziness</td>
<td>hypertension, past myocardial infarction</td>
<td>alternating RBBB + LBBB</td>
<td>610</td>
<td>235</td>
<td>100</td>
<td>120</td>
<td>2 p (died)</td>
</tr>
<tr>
<td>J.G.§</td>
<td>syncope, coma</td>
<td>past myocardial infarction</td>
<td>RBBB + LPH</td>
<td>710</td>
<td>242</td>
<td>100</td>
<td>90</td>
<td>14 p (died)</td>
</tr>
<tr>
<td>A.S.§</td>
<td>syncope</td>
<td>unknown</td>
<td>RBBB + LAH</td>
<td>880</td>
<td>290</td>
<td>190</td>
<td>85</td>
<td>9 p</td>
</tr>
<tr>
<td>J.D.§</td>
<td>seizures</td>
<td>hemodynamically insignificant aortic valve disease</td>
<td>RBBB + LAH</td>
<td>990</td>
<td>240</td>
<td>120</td>
<td>105</td>
<td>4 p</td>
</tr>
<tr>
<td>A.R.¶</td>
<td>syncope</td>
<td>hypertensive cardiovascular disease</td>
<td>RBBB + LAH</td>
<td>685</td>
<td>198</td>
<td>130</td>
<td>60</td>
<td>5 p</td>
</tr>
<tr>
<td>C.S.§</td>
<td>syncope, coma</td>
<td>coronary atherosclerosis (angina pectoris)</td>
<td>LBBB</td>
<td>630</td>
<td>185</td>
<td>70</td>
<td>90</td>
<td>6 p (died)</td>
</tr>
<tr>
<td>C.L.</td>
<td>syncope</td>
<td>unknown</td>
<td>RBBB + LAH</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td><strong>Mean</strong></td>
<td></td>
<td></td>
<td></td>
<td>751</td>
<td>232</td>
<td>118</td>
<td>89</td>
<td>7</td>
</tr>
</tbody>
</table>

* R = right, L = left, LAH = left anterior hemiblock, and LPH = left posterior hemiblock.
†† Permanent cardiac pacemaker inserted.
††† The neurologic symptoms disappeared after insertion of the cardiac pacemaker.
§§ Transient A-V block present on the admitting electrocardiographic record.
¶‡ A-V block occurred 24 hours after the His bundle study.
#‡ A-V block occurred 72 hours after study.
** ¶¶ Ventricular rate varied between 580 and 760 msec.

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Figure 2

(A) Lead I rhythm strip obtained on admission showing complete A-V block with idioventricular rate of 28 beats/min. (B) 12-lead electrocardiogram taken two hours later showing sinus rhythm, first degree A-V block, right BBB and left anterior hemiblock. (C) His bundle electrogram obtained at time of insertion of a temporary transvenous intracardiac pacemaker. The smaller terminal anterior forces (arrow) in V1 compared to V1 in section B suggest slight alteration in intraventricular conduction at the time of this study. A-H interval is within normal limits but there is marked prolongation of the H-Q interval. (HBE) = His bundle electrogram.

actually further supports the implication that patients with BBB and marked prolongation of the H-Q are at increased risk. Attempts were made to record the right bundle branch depolarization but were successful in only two studies. In addition since conduction times were often markedly prolonged, conceivably small differences in catheter position might reflect significant differences in measured A-H and H-Q intervals. We believe the latter possibility to be a very small source of error as the tricuspid valve was explored by means of a multipolar electrode catheter allowing for display and recording of His bundle activation from multiple bipolar leads and the smallest interval from atrial electrogram to His bundle depolarization was used in our measurements of A-V conduction times. Frequently, the His bundle potential was recorded in more than one bipolar lead and only minor (±5 msec) differences in A-H or H-Q intervals were noted between the different recording sites (fig. 4). Finally, a small error was introduced in the initial seven studies where V1
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alone was used to record the earliest onset of ventricular activation.

Measurement of infranodal conduction time (H-Q) is especially valuable in patients with BBB since it measures conduction characteristics of the His bundle and the remaining functional fascicles. Furthermore, post mortem anatomic studies of the conduction system have documented the relationship between bilateral bundle branch disease in patients with H-Q prolongation and BBB. In our study, all patients with documented complete A-V block (Group III) showed a markedly abnormal H-Q interval (with a mean value of 89 msec) when His bundle electrograms were obtained after return of sinus rhythm. Although others have reported comparable H-Q prolongation in patients with BBB, the magnitude of prolongation found for Group III patients is distinctly unusual and we believe reflective of severe impairment of conduction in the His-Purkinje system. Moreover, the significantly greater H-Q interval in patients with documented A-V block (Group III) compared with the other groups suggests that the specialized ventricular conduction system is involved in a progressive disease process, the end stage of which results in complete A-V block. These findings are in agreement with those of Narula and Samet who reported marked prolongation of the H-Q interval.

Figure 3

(A) 12-lead electrocardiogram obtained on admission shows left BBB with normal P-R interval. (B) His bundle electrogram taken on the same day shows normal A-H interval and marked prolongation of the H-Q interval. (C) MCL rhythm strip taken 24 hrs later shows complete heart block and ventricular standstill. Arrow indicates start of external cardiac compression.
Simultaneous X, Y, and Z leads of the Frank system and close bipolar His bundle electrograms showing close correspondence of His bundle depolarization (H) in each of the recordings. HBE₁ = bipolar electrogram recorded from the distal electrode pairs, HBE₂ = bipolar electrogram recorded from bipolar electrodes 10 mm from catheter tip.

(mean of 85 msec) in patients with right BBB and left anterior hemiblock who subsequently developed higher degrees of A-V block. Similarly, Ranganathan and associates⁶ found abnormal but less marked prolongation of the H-Q interval in four symptomatic patients with documented transient complete A-V block. In contrast, Kranz and Haft¹⁹ reported normal H-Q intervals in four of five patients with documented episodes of complete heart block. The reasons for the discrepant results are not immediately apparent.

In our study, patients with BBB whose symptoms appeared to be unrelated to episodically complete A-V block (Group I) showed either a normal H-Q interval or minimal prolongation of this interval. In none of these patients did higher degrees of A-V block develop during the follow-up period. Similarly, two patients in Group II with minimal H-Q prolongation did not show any increased impairment of A-V conduction on follow-up evaluation. However, two of three patients in Group II who underwent permanent pacemaker insertion experienced relief of symptoms, suggesting that these symptoms were in fact related to transient bradyarrhythmias.

His bundle electrograms were likewise useful in detecting concomitant block at the level of the A-V node. Two of seven patients in Group III showed prolongation of the A-H interval while two of six patients in Group I showed abnormally prolonged A-V nodal conduction times. Although none of the patients in the present study with prolonged A-H developed higher degrees of block, it is well appreciated that patients with A-V nodal disease may develop symptomatic high degree or complete A-V block.²⁰ Finally, this study is supportive of the previous observation²¹ that it is hazardous to make assumptions concerning infranodal conduction time on the basis of the P-R interval in the surface electrogram. Two of seven patients in Group III had normal P-R intervals but markedly prolonged H-Q times while six of the 12 patients in Groups I and II showed prolongation of the P-R interval that was primarily due to prolonged A-V nodal conduction. Quite clearly, a normal P-R interval does not exclude major block in the infranodal conduction system, nor does a prolonged P-R interval in a patient with BBB prove the existence of infranodal disease.

The electrocardiographic pattern of bifascicular block has been correlated with anatomic disease of the infranodal conduction system¹, ² and an increased risk for development of complete A-V block³–⁷ and/or sudden death²², ²³ This electrocardiographic pattern is not uncommon, being found in about 1% of electrocardiograms taken in hospital heart stations.³, ⁵ Most patients with this pattern remain asymptomatic for many years and prophylactic permanent pacemaker insertion appears to be unwarranted. Moreover, symptoms of dizziness,
seizures or syncope are also common and may be unrelated to transient A-V block. Both in the present study as well as in the larger series of patients with right BBB and left anterior hemiblock reported by Narula and Samet, a significant percentage of patients (25 and 28%, respectively) showed normal infranodal conduction. Therefore, the occurrence of these neurologic symptoms and an electrocardiographic pattern of bifascicular block do not invariably mean that the symptoms are related to impaired A-V conduction.

The relationship of the H-Q interval to the ultimate development of complete A-V block in patients with BBB remains uncertain. In our study, H-Q prolongation was found in all patients with documented or presumptive evidence of transient complete A-V block, and six of the nine patients with H-Q intervals greater than 80 msec showed documented or presumptive evidence of transient A-V block. Although mean H-Q was significantly longer in patients with documented A-V block, nevertheless, significant overlap of H-Q intervals between groups was noted. The studies of Kranz and Haft have clearly demonstrated that a normal H-Q interval in patients with BBB does not preclude development of complete A-V block, even within a few days of study. Further delineation of the value of His bundle studies in determining the precise risk of patients with BBB for development of complete A-V block and/or sudden death can only be obtained from carefully designed prospective studies. Until such data become available, we feel that the presence of H-Q prolongation does not, by itself, indicate the need for prophylactic cardiac pacemaker insertion. However, our data would suggest that it is prudent to strongly consider insertion of a permanent cardiac pacemaker in patients with transient neurologic symptoms, BBB and marked prolongation of H-Q (≥ 80 msec) even in the absence of documented episodes of complete A-V block.

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