Prognostic Value of Infranodal Conduction Time in Patients with Chronic Bundle Branch Block

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SUMMARY  His bundle recordings were obtained in 121 patients with chronic bundle branch block and the patients were followed for a mean period of 18 months. Seventy-nine patients had an infranodal conduction time (H-Q) < 70 msec while 42 had H-Q ≥ 70 msec. There was no significant difference in mean age, smoking history, diabetes, syncope, dizziness, blood pressure, and serum cholesterol or triglyceride levels between the two groups. There was a significantly greater incidence of progression to second degree or third degree atrioventricular block (9/42, 21%), and of severe congestive heart failure (16/42, 38%) in patients with H-Q ≥ 70 compared with those with H-Q < 70 (1/79, 1.3%; and 13/79, 16%, respectively). The risk of sudden death was significantly greater only in the group with H-Q ≥ 70 and severe congestive heart failure. There was no correlation between the presence of first degree atrioventricular block and/or any particular type of bundle branch block pattern with sudden death and/or progression to second degree or third degree atrioventricular block.

Analysis of the surface electrocardiogram is only of limited value in predicting high risk patients with chronic bundle branch block. Electrophysiologic studies are of greatest value in patients with bundle branch block with transient neurologic symptoms in whom no cause for the symptoms is evident.

THE PROXIMAL PORTION of the infranodal conduction system is comprised of the His bundle and right and left bundle branches. The left branch divides into at least two fascicles that supply the anterior-superior and posterior-medial portions of the left ventricle. Recently, electrocardiographic criteria were suggested for the diagnosis of conduction blocks in either the left anterior or posterior fascicles. The diagnosis of conduction failure in two of three fascicles suggests an impaired safety margin for atrioventricular (A-V) conduction. Moreover, a wealth of anatomic and clinical studies supports the relationship between intraventricular conduction disturbances and progression to high grade or complete A-V block. Progression to complete A-V block may be sudden or may occur episodically and result in sudden death and/or Stokes-Adams attacks. The ability to recognize these patients with intraventricular conduction disturbances at high risk for progression to complete A-V block is especially important as these patients may benefit from prophylactic intracardiac pacemaker insertion. The purpose of this study was to assess the usefulness of His bundle recordings in detecting patients with bundle branch block at risk of developing high grade A-V block. In addition, we evaluated the known risk factors for sudden death in an effort to determine whether or not the H-Q interval was an independent risk factor.

Materials and Methods

Materials

In March 1972, a prospective study of patients with intraventricular conduction delay was instituted among hospitals in San Francisco. Twelve-lead electrocardiograms were screened for evidence of intraventricular conduction block and patients with the disturbance were invited to volunteer for the study. Patients from other San Francisco hospitals who were referred to us for electrophysiologic evaluation were also included in the study. The University of California Committee on Human Experimentation approved the study, and informed consent was obtained from all patients.

Methods

A complete history, chest roentgenogram, and serum cholesterol and triglycerides were obtained and a physical examination was performed. His bundle electrograms were obtained using standard techniques. In brief, a multipolar electrode catheter was inserted into the right femoral vein and positioned across the tricuspid valve. The His bundle electrogram and surface leads X, Y, and Z of the Frank orthogonal lead system were simultaneously displayed on an oscilloscope and recorded using an Electronics for Medicine (DR-12) recorder. The A-V nodal conduction time was measured from the initial high frequency deflection of the low right atrial electrogram to the first high frequency His bundle deflection (A-H), whereas infranodal conduction time was measured from the initial high frequency deflection recorded from the His bundle to the onset of ventricular activation determined from the surface leads (H-Q). Patients who had second or third degree A-V block before or during the His bundle recording were excluded from study. Similarly, patients with acute myocardial infarction were excluded.

Follow-Up. Patients were followed every three months either in the pacemaker or arrhythmia clinic at San Francisco General Hospital Medical Center or by referring physicians. Follow-up information was obtained from patients who did not keep scheduled appointments by either a public health nurse or health care technician. Where necessary, home visits were made to obtain additional information including 12-lead electrocardiograms. In the event of diagnosed progression to higher degrees of A-V block, efforts were made to obtain electrocardiographic tracings showing the A-V block. In the event of death, hospital and/or coroner's reports were reviewed and observers were interviewed to determine if the death was sudden.

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Definitions

For purposes of this study, the following definitions of terms were used: sudden death, those deaths occurring unexpectedly and either instantaneously (or during sleep) or within one hour after onset of symptoms; bundle branch block, left and right bundle branch block defined according to Goldman; left anterior hemiblock, when initial frontal plane forces were directed inferiorly but the mean QRS vector was superior to \(-30^\circ\); left posterior hemiblock, initial forces directed superiorly with mean frontal plane QRS vector \(\geq 100\) degrees in the absence of right ventricular hypertrophy or anterolateral wall myocardial infarction; and intraventricular conduction delay, when the surface electrocardiogram showed prolongation of the QRS (\(\leq 0.12\) sec) without specific bundle branch block pattern.

Congestive heart failure was diagnosed on the basis of physical findings and chest roentgenograms, and the patients were categorized according to the New York Heart Association functional classifications. Valvular heart disease was diagnosed on the basis of the physical examination or results of cardiac catheterization. The diagnosis of cardiomyopathy was made in those patients with cardiac failure without demonstrable cause. For purposes of this study, patients with a history of smoking in excess of ten cigarettes per day were classified as cigarette smokers.

All data were stored on punch cards and later retrieved using a program written for the IBM Model 360/75 computer. All nominal data were tested with the chi-square test or Fisher's exact probability test. Correlation of morbid events with H-Q intervals was tested with a one-way analysis of variance with Scheffe's method for multiple contrasts.

A total of 171 patients entered into the study, but 50 patients were excluded from this report. Of the 50, 24 underwent prophylactic cardiac pacemaker insertion at the discretion of their referring physicians. Ten were excluded because of insufficient follow-up information, and 11 had not completed their first three months follow-up. Five additional patients were excluded because a permanent pacemaker was inserted because of documented symptomatic sinus node dysfunction.

Results

The pertinent clinical and electrocardiographic data for 121 patients with bundle branch block are summarized in tables 1 and 2. The patients were divided into two subgroups; those with infranodal conduction time \(<70\) msec (H-Q \(\leq 70\)) (79 patients) and those with infranodal conduction times \(\geq 70\) msec (H-Q \(\geq 70\)). This division was made on the basis of correlating serial 5 msec sequential analyses of the H-Q interval and the incidence of either sudden death or progressive A-V block.

Clinical Comparisons

There was a greater percentage of males in the H-Q \(\geq 70\) group than in the H-Q < 70 group. However, there was no significant difference between the groups for the other recognized risk factors for sudden death (i.e., blood pressure, history of cigarette smoking, incidence of coronary artery disease, diabetes, or elevated serum cholesterol or triglyceride levels). The incidence of syncope or dizziness was similar for both groups. A significantly greater percentage of H-Q \(\geq 70\) patients were in functional class III or IV than H-Q < 70 patients (P < 0.05).

Analysis of the Electrocardiographic Patterns

The presenting electrocardiographic patterns are detailed in table 2. Fifty-two of 121 (43%) patients had a pattern of first degree A-V block (P-R \(\geq 0.20\) sec), and the incidence of this pattern was greater in the H-Q \(\geq 70\) (25/42; 59%) group than in the H-Q < 70 (27/79; 34%) group. The presence of first degree A-V block, however, was a poor indicator of infranodal conduction delay as 36/52 (69%) had A-V nodal block. Similarly, there was no correlation between this electrocardiographic finding and the incidence of progressive A-V block or sudden death. In addition, there was no significant difference in the incidence of specific bundle branch block patterns between the groups (table 2) nor was there any correlation between the bundle branch block pattern (or combination with first degree A-V block) and the incidence of progression to higher degrees of A-V block or sudden death.

H-Q and Progressive A-V Conduction Disturbances

There was a significant correlation between the incidence of H-Q \(\geq 70\) msec and progression to high grade A-V block (table 3). Patients in this group showed a relative risk of progressive A-V block of 26.7:1\textsuperscript{st} compared with patients with H-Q < 70 msec. The pertinent clinical and electrocardiographic findings for the 10 patients who progressed to

<table>
<thead>
<tr>
<th>Table 1. Clinical Description of Subjects</th>
<th>H-Q (\leq 70) msec</th>
<th>H-Q (\geq 70) msec</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male/female</td>
<td>56/23</td>
<td>38/4</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Mean ((\pm) sd) age (yr)</td>
<td>67 (\pm) 15</td>
<td>67 (\pm) 11</td>
<td>N.S.</td>
</tr>
<tr>
<td>Syncope</td>
<td>20</td>
<td>14</td>
<td>N.S.</td>
</tr>
<tr>
<td>Dizziness</td>
<td>17</td>
<td>10</td>
<td>N.S.</td>
</tr>
<tr>
<td>CAD</td>
<td>64</td>
<td>31</td>
<td>N.S.</td>
</tr>
<tr>
<td>Smokers</td>
<td>40</td>
<td>20</td>
<td>N.S.</td>
</tr>
<tr>
<td>Diabetes</td>
<td>12</td>
<td>28</td>
<td>N.S.</td>
</tr>
<tr>
<td>NYHA class III or IV</td>
<td>15</td>
<td>16</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Mean (\pm) sd systolic blood pressure (mm Hg)</td>
<td>136 (\pm) 25</td>
<td>136 (\pm) 25</td>
<td>N.S.</td>
</tr>
<tr>
<td>Cardiomyopathy or valvular heart disease</td>
<td>18</td>
<td>13</td>
<td>N.S.</td>
</tr>
<tr>
<td>Mean (\pm) sd serum cholesterol</td>
<td>217 (\pm) 47</td>
<td>183 (\pm) 41</td>
<td>N.S.</td>
</tr>
<tr>
<td>Mean (\pm) sd serum triglycerides</td>
<td>129 (\pm) 71</td>
<td>118 (\pm) 54</td>
<td>N.S.</td>
</tr>
<tr>
<td>Follow-up (months)</td>
<td>19 (\pm) 14</td>
<td>16 (\pm) 13</td>
<td>N.S.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Table 2. Electrocardiographic Findings</th>
<th>H-Q (\leq 70) msec</th>
<th>H-Q (\geq 70) msec</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>1\textsuperscript{st} atrioventricular block</td>
<td>27</td>
<td>25</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Right bundle branch block</td>
<td>18</td>
<td>9</td>
<td>N.S.</td>
</tr>
<tr>
<td>Right bundle branch block + left anterior hemiblock</td>
<td>26</td>
<td>13</td>
<td>N.S.</td>
</tr>
<tr>
<td>Right bundle branch block + left posterior hemiblock</td>
<td>3</td>
<td>4</td>
<td>N.S.</td>
</tr>
<tr>
<td>Left bundle branch block</td>
<td>28</td>
<td>14</td>
<td>N.S.</td>
</tr>
<tr>
<td>Intraventricular conduction delay</td>
<td>4</td>
<td>2</td>
<td>N.S.</td>
</tr>
<tr>
<td>QRS duration</td>
<td>138 (\pm) 20</td>
<td>138 (\pm) 24</td>
<td>N.S.</td>
</tr>
</tbody>
</table>
TABLE 3. Prognostic Indexes

<table>
<thead>
<tr>
<th></th>
<th>H-Q &lt; 70 msec</th>
<th>H-Q ≥ 70 msec</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Progression to 2° or 3° atrioventricular block</td>
<td>1</td>
<td>9</td>
<td>10⁻⁴</td>
</tr>
<tr>
<td>Sudden death</td>
<td>1</td>
<td>7</td>
<td>10⁻⁴</td>
</tr>
<tr>
<td>Total deaths</td>
<td>13</td>
<td>15</td>
<td></td>
</tr>
</tbody>
</table>

high grade A-V block are tabulated in table 4. Six of the 10 patients developed progressive A-V block within one month of study. The site of the block was verified by repeat electrophysiologic studies in three patients (Nos. 4, 7, 8) (table 4). Patient 4 showed 2:1 A-V block with block occurring in the His-Purkinje system. Patient 7 showed high grade A-V block with conducted beats showing alternate right bundle branch block and left bundle branch block patterns. Patient 8 showed complete A-V block with an idioventricular rhythm, with QRS morphology different from the preblock pattern. In the last two patients, His bundle electrograms confirmed a block in the His-Purkinje system. Repeat studies were obtained in one additional patient (No. 1) after he presented with a documented Stokes-Adams attack. The patient resumed sinus rhythm with 1:1 A-V conduction during the study with an H-Q interval of 80 msec (which was identical to that of the control study performed six months prior to his admission). A presumptive diagnosis of infranodal block was made on the basis of surface electrocardiographic recordings in the remaining patients (table 4). These patterns included Mobitz II type block, third degree A-V block with idioventricular focus, and third degree A-V block associated with ventricular standstill. In three patients (Nos. 2, 3, and 5), high grade A-V block occurred during a subsequent admission for acute myocardial infarction. The correlation between H-Q ≥ 70 and progressive A-V block remains even if the last three patients are omitted from analysis.

Relationship of H-Q Interval and Mortality

There was a statistically significant increase in the incidence of sudden death for the H-Q ≥ 70 group compared with the H-Q < 70 group (table 3). Further analysis showed that this correlation was strongly influenced by the presence or absence of severe congestive heart failure. For patients with no or mild left ventricular failure (NYHA class I or II), there was no significant difference in incidence of sudden death in those with H-Q ≥ 70 msec (2/25, 8%) compared with those with H-Q < 70 msec (1/65, 1.5%). On the other hand, for patients in functional class III or IV, there was a significantly greater incidence of sudden death in the group showing H-Q ≥ 70 (5/17, 42%) than in those showing H-Q < 70 (0/14) (P < 0.001). The association of H-Q ≥ 70 msec and severe congestive heart failure emerges as an additional risk factor for sudden death that is independent of the previously described risk factors (e.g., serum cholesterol level, blood pressure, cigarette smoking). Similarly, the total mortality was significantly greater only in patients with H-Q ≥ 70 msec in the presence of moderate or severe congestive heart failure.

Discussion

Progression to High Grade A-V Block

Our study showed that the H-Q interval in patients with chronic bundle branch block is an independent risk factor for progression to second or third degree A-V block. We found, moreover, that marked prolongation of H-Q (≥ 70 msec) was

TABLE 4. Electrocardiographic Findings in Patients with Progressive Atrioventricular Block

<table>
<thead>
<tr>
<th>Patient no.</th>
<th>Preblock electrocardiogram</th>
<th>Preblock H-Q interval (msec)</th>
<th>Type of block</th>
<th>Time after study</th>
<th>Circumstances of detection</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Right bundle branch block + left anterior hemiblock</td>
<td>105</td>
<td>3° atrioventricular block + ventricular standstill (8 sec)</td>
<td>6 months</td>
<td>Hospital admission for evaluation of grand mal seizures</td>
</tr>
<tr>
<td>2</td>
<td>Right bundle branch block + left anterior hemiblock</td>
<td>95</td>
<td>Mobitz II</td>
<td>13 months</td>
<td>Hospital admission for acute anterior myocardial infarction</td>
</tr>
<tr>
<td>3</td>
<td>Right bundle branch block + left anterior hemiblock</td>
<td>98</td>
<td>3° atrioventricular block with idioventricular rate of 42 beats/min</td>
<td>22 days</td>
<td>Hospital admission for acute anterior myocardial infarction</td>
</tr>
<tr>
<td>4</td>
<td>Right bundle branch block + left anterior hemiblock</td>
<td>82</td>
<td>2:1 atrioventricular block</td>
<td>36 months</td>
<td>Follow-up electrocardiograms (outpatient), periodic dizzy spells</td>
</tr>
<tr>
<td>5</td>
<td>Right bundle branch block + left posterior hemiblock</td>
<td>90</td>
<td>Mobitz II 3° atrioventricular block</td>
<td>18 days</td>
<td>Hospital admission for acute anterior myocardial infarction and severe heart failure</td>
</tr>
<tr>
<td>6</td>
<td>Right bundle branch block</td>
<td>45</td>
<td>Mobitz II</td>
<td>11 days</td>
<td>During hospital admission for treatment of osteomyelitis</td>
</tr>
<tr>
<td>7</td>
<td>Right bundle branch block</td>
<td>120</td>
<td>High grade atrioventricular block (conducted beats showed alternate right and left bundle branch block)</td>
<td>4 days</td>
<td>Monitored in a coronary care unit (severe heart failure)</td>
</tr>
<tr>
<td>8</td>
<td>Left bundle branch block</td>
<td>73</td>
<td>3° atrioventricular block, idioventricular rhythm rate 37 beats/min</td>
<td>12 days</td>
<td>In hospital observation, thrombophlebitis, Stokes-Adams attack</td>
</tr>
<tr>
<td>9</td>
<td>Left bundle branch block</td>
<td>95</td>
<td>3° atrioventricular block + ventricular standstill</td>
<td>6 days</td>
<td>Emergency room, after Stokes-Adams attack</td>
</tr>
<tr>
<td>10</td>
<td>Left bundle branch block</td>
<td>70</td>
<td>Mobitz II block</td>
<td>10 months</td>
<td>Electrocardiogram taken in convalescent hospital</td>
</tr>
</tbody>
</table>
associated with a 21% (9/42) incidence of progression to high grade A-V block within a mean follow-up period of 18 months. In contrast, only one of 79 patients with H-Q < 70 msec showed progressive A-V conduction disturbances. These conclusions remain valid even if the three patients who showed progressive A-V block during subsequent admission for acute myocardial infarction are excluded from the study. Our findings support the concept that the safety margin for continued successful A-V conduction is impaired in those patients with marked prolongation of infranodal conduction time. It also suggests that these patients suffer from a progressive disorder involving the His-Purkinje system that may result in complete failure of infranodal conduction. This conclusion remains tentative because the site of block was documented by repeat studies in only three of 10 patients. In addition, the bulk of patients showing progression (60%) did so within 22 days of study, and we cannot exclude the possibility that the symptoms in these patients were related to undetected episodic A-V block.

Review of the literature reveals widely differing incidences of progression to second or third degree A-V block among patients with chronic bundle branch block. Furthermore, differing etiologies and/or associated disease processes appear to play an important role in the rate of progression. For example, Rotman and Trimbawasser studied 519 mostly young asymptomatic Air Force personnel (93% of whom had no evidence of cardiovascular disease) and found only two instances of progression to complete A-V block within approximately 10 years. In that study, progression to complete A-V block occurred in only one of 96 patients with either right bundle branch block and left anterior hemiblock or right bundle branch block and right axis deviation and in only one of 125 patients with left bundle branch block. In contrast, DePasquale and Bruno studied 85 patients with right bundle branch block and left anterior hemiblock and found progression to second degree A-V block in 15 patients and complete A-V block in two over a mean follow-up time of 3.1 years. In their series, all but five had associated organic cardiac disease and 77% had coronary artery disease. It is conceivable that patients with idiopathic sclerodegenerative disease of the ventricular conduction system alone have a much slower rate of progression than those patients with bundle branch block related to either ischemia alone or ischemia plus idiopathic degenerative disease involving the conduction system.

Previously reported studies of patients with bundle branch block and documented second or third degree A-V block who underwent His bundle recordings show marked prolongation of infranodal conduction time during 1:1 A-V conduction. These findings, though retrospective, support our results with respect to the importance of marked prolongation of H-Q and development of progressive A-V block. Similarly, the observed increased mortality in patients with marked prolongation of the H-Q interval supports the findings of Narula et al. They, for example, found a five-fold difference in mortality between patients with prolonged H-Q and those with normal H-Q intervals. Our study design differed from that of Narula et al. in that they included 14 patients (of 58 with prolonged H-Q) with documented second degree A-V block that occurred before or during the electrophysiologic study.

It is particularly important to compare our study with similarly designed recent studies by Denes et al. and Dhirgra et al. Denes et al. followed 119 patients with right bundle branch block and left anterior hemiblock for a mean follow-up time of approximately 18 months; the incidences of progression to advanced A-V block, sudden death, or nonsudden death were similar to ours. In addition, we confirmed their findings of a higher incidence of congestive heart failure as well as a higher mortality rate in patients with a prolonged H-Q interval. The studies differed, however, in that they found no significant difference in the incidence of sudden death or progression to second or third degree A-V block in patients with or without abnormal infranodal conduction time. Furthermore, in an earlier report by Dhirgra et al. of 18 patients with H-Q ≥ 80 msec, there was only one instance of progression to second degree A-V block within a mean follow-up period of 711 days.

In our study, an H-Q ≥ 70 msec was associated with an increased incidence of sudden death (and was independent of other known risk factors for this complication) only in those patients with moderate or severe congestive heart failure. There are insufficient data in the reports by Denes et al. and to compare these findings. Somewhat more difficult to explain is the discrepancy in the incidence of progression to second or third degree A-V block in patients with abnormal H-Q intervals between our study and that of Denes et al. The difference may be related to different types of patients. For example, the incidence of coronary artery disease was higher in our series (57%) than in theirs (27%). In addition, 43% of our patients presented with syncope compared with approximately 10% in their studies. Finally, we included all patients with intraventricular conduction delay, whereas their series was restricted to patients with bifascicular block. Our series was clearly biased in the preselection of patients with transient neurologic symptoms because these patients were more apt to be referred for study.

Our studies highlight the difficulties in predicting those patients with bundle branch block who are at high risk of sudden death or A-V block on the basis of the surface electrocardiogram. Although patients with first degree A-V block had a significantly longer H-Q interval compared with those without first degree A-V block, in the majority of patients with first degree A-V block the site of the block was localized to the A-V node. Similar findings were reported by others. In addition, we found no significant correlation between the type of bundle branch block pattern and the incidence of sudden death, the progression to second or third degree block, or an H-Q interval ≥ 70 msec. The H-Q duration was, therefore, a more accurate predictor of major complications than the surface recordings.

**Limitations of the Study**

Several important limitations of our study are apparent. The assumption of progressive infranodal conduction block remains inferential because the site of the block was located by repeat studies in only three of the 10 patients who showed progressive disease of the conduction system. Furthermore, the bulk of patients (6/10) developed A-V block within 22 days of study, while three additional patients progressed to A-V block in the course of a subsequent myocardial infarc-
tion. It is likely that in the six patients showing early progression, symptoms were related to episodic high grade A-V block that was not detected in spite of repeated continuous electrocardiographic monitoring. The most valuable use of electrophysiologic study would appear to be in symptomatic patients with bundle branch block in whom no other cause of symptoms is found. The value of the H-Q interval in predicting development of remote A-V block awaits further studies. In addition, the mechanism of death in those who died suddenly is unknown. Others have found a high incidence of premature ventricular depolarizations and ventricular tachycardia in patients with bundle branch block. We have insufficient continuous 24-hour electrocardiographic recordings to comment on the relationship of premature ventricular depolarizations to sudden death. Finally, our results are applicable only for a rather select subgroup of patients with bundle branch block and cannot be extrapolated to all patients with bundle branch block.

Clinical Implications

Although our findings cannot be extrapolated to reflect on the natural history of unselected patients with bundle branch block, nevertheless our study group represents a reasonable sample of patients with bundle branch block likely to be referred for electrophysiologic studies. Our results lead us to the following conclusions. His bundle electrograms should be obtained in patients with bundle branch block and transient neurologic symptoms in whom prolonged continuous electrocardiographic monitoring fails to reveal evidence of second or third degree A-V block. These studies should be obtained irrespective of the type of bundle branch block pattern or the presence or absence of first degree A-V block. Prophylactic insertion of a permanent pacemaker is warranted in those patients with marked prolongation of H-Q (≥70 msec) in whom no other cause for the neurologic symptoms is found. Symptomatic patients with H-Q <70 msec appear to have a low (short-term) incidence of either sudden death or progression to second or third degree A-V block. The relatively small sample size of asymptomatic patients with H-Q ≥70 msec precludes firm conclusions, but our study indicates that these patients should be followed carefully because they appear to be at increased risk for progressive A-V block. Finally, our results support the need for a prospective study to determine whether or not prophylactic insertion of a permanent cardiac pacemaker in symptomatic patients with bundle branch block and marked prolongation of the H-Q interval will result in alleviation of neurologic symptoms or decreased incidence of sudden death.

Acknowledgment

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References

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