Significance of the HV Interval in 517 Patients with Chronic Bifascicular Block

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SUMMARY In January 1975, we reported results of a prospective follow-up study (mean 538 ± 42 days) of 119 patients with chronic bifascicular block (BFB), and concluded that BFB patients with normal and prolonged HV (NHV and PHV) had a similar incidence of atrioventricular (AV) block and mortality. In this report, we update these findings in 517 patients with a follow-up of 21 days to 9.8 years (mean 3.4 ± 0.2 years). Three hundred nineteen patients (61%) had NHV and 198 (39%) had PHV (>55 msec). The NHV and PHV groups were similar in regard to age (NHV vs PHV, 61 ± 1 vs 62 ± 1 years) and sex (80% male, 20% female vs 82% male and 18% female). The following were more common (p < 0.05) in patients with PHV (percent of patients with finding in NHV vs PHV groups): angina (18% vs 27%), congestive failure (27% vs 42%), cardiomegaly (48% vs 66%), New York Heart Association functional class II-IV (34% vs 56%), premature ventricular complexes (20% vs 29%), and organic heart disease (OHD) (75% vs 85%). Spontaneous trifascicular block (TFB) developed in two patients (0.6%) with NHV and nine patients (4.5%) with PHV (p < 0.05). Cumulative 7-year incidence of TFB was 3% with NHV and 12% with PHV (p < 0.01). Seven-year cumulative cardiovascular mortality was 32% in NHV patients and 57% in PHV patients (p < 0.005).

In conclusion, PHV in patients with chronic BFB was associated with a greater incidence and severity of OHD, and higher total and sudden death mortalities. The risk of spontaneous TFB was small in patients with either NHV or PHV, although it was significantly higher in the latter.

THE CLINICAL SIGNIFICANCE of a prolonged HV interval in patients with chronic bifascicular block has been controversial. Scheinman et al., 3 Narula et al., 4 and McAnulty and co-workers confirmed our findings.5 To resolve this controversy, we analyzed our experience in our series of patients with chronic bifascicular block. In this study, we examine and compare clinical, electrocardiographic and electrophysiologic variables in bifascicular block patients with normal and prolonged HV intervals. We also report prospective observations concerning life history of these patients to determine the prognostic significance of the HV interval.

Our results suggest that the HV interval has prognostic significance. Using Bayesian theory, we believe that our data can be reconciled with previous data concerning the significance of the HV interval in patients with chronic bifascicular block.

Materials and Methods

Definitions are based on the recommendations of the Criteria Committee of the New York Heart Association.6 The criteria for electrocardiographic diagnosis of right bundle branch block included a QRS duration of 0.12 second or greater, with an rsR or qr configuration of the QRS complex in lead V1. The criteria for diagnosis of left bundle branch block included a QRS duration of 0.12 second or greater, with the presence of a broad monophasic R wave or Rs in lead V6 with ST depression and T-wave inversion in leads V5 and V6.

In the patients with right bundle branch block, left anterior fascicular block was diagnosed if the mean frontal QRS axis was more negative than −30°, with small q and tall R wave in lead I, and a small r and deep S wave in lead III. Left posterior fascicular block was diagnosed if the mean frontal QRS axis was more positive than 90° in the absence of right ventricular hypertrophy, with a small r and deep S wave in lead I, and a small q and tall R wave in lead III.

Bifascicular block was defined as right bundle branch block with left anterior fascicular block, right bundle branch block with left posterior fascicular block, or left bundle branch block. The latter was included as a bifascicular block; we consider left bundle branch block to be equivalent to involvement of two fascicles (left anterior and posterior).

Patient Selection

Patients with chronic bifascicular block were detected through screening of inpatient and outpa-
tient ECGs in the Chicago West Side Medical Center (University of Illinois Hospital, West Side Veterans Administration Hospital, and Cook County Hospital). Additional patients with bifascicular block were also referred to us for electrophysiologic study and follow-up from physicians in the Chicago area.

Criteria for inclusion in this study were: presence of chronic bifascicular block with intact AV conduction; age 18 years or older; informed consent for and performance of electrophysiologic studies; and voluntary agreement to periodic follow-up in a conduction disease clinic. Patients with a history of second- or third-degree AV block and patients with acute myocardial infarction were excluded.

Initial evaluation of patients included history, physical examination, serial ECGs, chest roentgenogram and routine laboratory tests. Based on this evaluation, a clinical diagnosis was established for each patient. Previously described criteria were used for diagnosis of organic heart disease. Primary conduction disease was diagnosed if radiographic heart size was normal and there was no clinical evidence of organic heart disease, with the exception of an intraventricular conduction defect.

Electrophysiologic Studies

Each patient gave informed written consent before the electrophysiologic study. His bundle (H) electrograms were recorded, using previously described catheter techniques, at the time of entry into the study. Cardiac drugs were withheld for at least 48–72 hours before the study. AH and HV interval measurements were made at paper speeds of 200 mm/sec and reflected the mean of 10 consecutive sinus beats. The normal AH interval is 54–130 msec; the normal HV interval 31–55 msec. HV intervals alone were measured in patients with atrial fibrillation.

Patient Follow-up

Patient follow-up was similar to that previously described. After initial study, all patients were prospectively followed in a conduction disease clinics at intervals of 1–3 months. At each clinic visit, history, physical examination, and ECGs were performed. Portable tape-recorded monitoring and/or prolonged inpatient electrocardiographic monitoring were used when history suggested transient bradyarrhythmias. In patients who developed AV block, we attempted to localize the site of block, using clinical data, electrocardiographic criteria and repeat His bundle recording, whenever possible. Permanent pacemakers were implanted in symptomatic patients with documented bradyarrhythmias or in patients with recurrent syncope of unknown cause. The term "trifascicular block" was used to imply second- or third-degree AV block distal to the His bundle recording site.

Deaths during the follow-up were classified as either sudden or not sudden. Sudden death was defined as unexpected death due to natural causes occurring within 24 hours of the onset of acute symptoms or within 24 hours of being seen alive without symptoms. Deaths that did not fulfill these criteria were classified as nonsudden. Deaths were further subcategorized as deaths due to cardiovascular or noncardiovascular causes. Cardiovascular deaths included all sudden deaths and deaths occurring due to cardiac related causes. Deaths unrelated to cardiac events were classified as noncardiovascular.

Analysis of Data

All information at the time of initial evaluation and subsequent clinic visits was keypunched and stored on IRS database discs. Specifically designed programs were used for data recall and statistical analysis. Data on cumulative risk of AV block and survival were analyzed using life-table methods. A standard, unpaired t test was used to test the significance of differences in means and two-by-two chi-square method for frequency data analysis.

Results

The study group consisted of 517 patients with chronic bifascicular block. Three hundred twenty-nine patients had right bundle branch block and left anterior fascicular block, 46 right bundle branch block and left posterior fascicular block and 142 left bundle branch block.

On the basis of electrophysiologic studies, the 517 patients were classified into two groups: the normal HV group, 319 (61%) patients with a normal HV interval (55 msec or less); and the prolonged HV group, 198 (39%) patients with a prolonged HV interval (56 msec or greater).

Clinical Data

The mean age (± SEM) was 61 ± 1 years for the normal HV group and 62 ± 1 years for the prolonged HV group (NS). There were 254 males (80%) and 65 females (20%) in the normal HV group and 163 males (82%) and 35 females (18%) in the prolonged HV groups (NS).

The prevalences of specific categories of heart disease in the 517 patients are presented in table 1. Hypertensive cardiovascular disease and primary conduction disease were significantly more frequent in the normal HV group (p < 0.01 and < 0.001), while arteriosclerotic heart disease and primary myocardial disease were more frequent in the prolonged HV group (p < 0.001 and p < 0.01). There were no significant differences in the prevalence rates of valvular and other heart diseases in patients with normal and prolonged HV intervals.

The frequency of cardiovascular symptoms and selected historical findings are presented in table 2. Angina, incidence of old myocardial infarction, dyspnea, third and fourth heart sounds, congestive heart failure and cardiomegaly by chest roentgenogram were significantly more common in the
prolonged HV group. Most of the patients with a normal HV interval were asymptomatic (New York Heart Association functional class I) and the majority of the patients with a prolonged HV interval were symptomatic (classes II to IV) \( p < 0.001 \).

**Electrocardiographic Data (table 3)**

PR intervals were significantly longer in the patients with HV prolongation \( p < 0.001 \). Premature ventricular complexes detected on 12-lead resting ECGs were more common in the prolonged HV group \( p < 0.025 \). Right bundle branch block was significantly more prevalent in the normal HV group and left bundle branch block was more prevalent in the prolonged HV group \( p < 0.001 \) and \( p < 0.001 \).

**Electrophysiologic Data (table 4)**

In patients with a normal HV interval, HV ranged from 24–55 msec (mean 46 ± 0.36 msec). In the prolonged HV group, the HV interval ranged from 56–125 msec (mean 69 ± 0.86 msec). By study design, this difference was statistically significant \( p < 0.0001 \). There were no significant differences in the two groups in regards to AH interval, frequency of AH prolongation, AV nodal as well as ventricular specialized conduction system effective refractory periods and sinus nodal recovery time.

**Follow-up Data**

The follow-up ranged from 21 days to 9.8 years (mean 3.7 ± 0.1 years) for the normal HV group and 37 days to 9.4 years (mean 3.1 ± 0.2 years) for the prolonged HV group (NS). Second- or third-degree AV block developed in 15 of 319 patients (4.7%) with normal HV and 19 of 198 patients (9.5%) with prolonged HV \( p < 0.05 \). Of these, the AV block was spontaneous and not due to an apparent cause in 10 patients (3%) with a normal HV interval and 3 patients (6.5%) with prolonged HV (NS). AV block was secondary to an apparent cause in five patients (1.7%) with normal HV and six patients (3%) with prolonged HV interval (NS). Spontaneous trifascicular block developed in two patients (0.6%) in the normal HV group and nine patients (4.5%) in the prolonged HV group \( p < 0.02 \).

The annual total cumulative incidence of all AV block, as well as the cumulative incidence of spontaneous block and trifascicular block for the first 7 years, using actuarial life-table methods, is presented in figure 1. The cumulative 7-year incidence of all AV block was 12% in the normal HV group and 28% in prolonged HV group \( p < 0.001 \). The cumulative 7-year incidence of spontaneous AV block was 10% in the normal and 20% in the prolonged HV group \( p < 0.005 \). The cumulative 7-year incidence of spontaneous trifascicular block was 3% in the normal HV and 12% in the prolonged HV group \( p < 0.001 \). The incidence of spontaneous AV block and trifascicular block was significantly higher in the prolonged HV group from the third year on.

Two hundred seventeen of 517 patients died during the follow-up, 122 (38%) from the normal HV group and 95 (48%) from the prolonged HV group \( p < 0.05 \). Of these, sudden death occurred in 49 (15%) in the normal HV group and 40 (20%) in the prolonged

**TABLE 1. Classification of Heart Disease in 517 Patients with Normal and Prolonged HV Intervals**

<table>
<thead>
<tr>
<th>Heart disease</th>
<th>Normal HV (n = 319)</th>
<th></th>
<th>Prolonged HV (n = 198)</th>
<th></th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Primary conduction disease</td>
<td>79</td>
<td>25</td>
<td>22</td>
<td>11</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Organic heart disease (total)</td>
<td>240</td>
<td>75</td>
<td>176</td>
<td>89</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>HCVD</td>
<td>119</td>
<td>37</td>
<td>51</td>
<td>26</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>ASHD</td>
<td>74</td>
<td>23</td>
<td>78</td>
<td>39</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>VHD</td>
<td>14</td>
<td>4</td>
<td>15</td>
<td>8</td>
<td>NS</td>
</tr>
<tr>
<td>PMD</td>
<td>17</td>
<td>5</td>
<td>24</td>
<td>12</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Other</td>
<td>16</td>
<td>5</td>
<td>8</td>
<td>4</td>
<td>NS</td>
</tr>
</tbody>
</table>

Abbreviations: HCVD = hypertensive cardiovascular disease; ASHD = arteriosclerotic heart disease; VHD = valvular heart disease; PMD = primary myocardial disease.

**TABLE 2. Clinical Data in 517 Patients**

<table>
<thead>
<tr>
<th>Clinical finding</th>
<th>Normal HV (n = 319)</th>
<th></th>
<th>Prolonged HV (n = 198)</th>
<th></th>
<th>( p )</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n</td>
<td>%</td>
<td>n</td>
<td>%</td>
<td></td>
</tr>
<tr>
<td>Syncope</td>
<td>37</td>
<td>12</td>
<td>33</td>
<td>17</td>
<td>NS</td>
</tr>
<tr>
<td>Angina</td>
<td>58</td>
<td>18</td>
<td>53</td>
<td>27</td>
<td>&lt;0.02</td>
</tr>
<tr>
<td>Previous MI</td>
<td>45</td>
<td>14</td>
<td>47</td>
<td>24</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Dyspnea</td>
<td>107</td>
<td>34</td>
<td>105</td>
<td>53</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>S1</td>
<td>34</td>
<td>11</td>
<td>48</td>
<td>24</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>S2</td>
<td>155</td>
<td>49</td>
<td>114</td>
<td>58</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>NYHA class I</td>
<td>211</td>
<td>66</td>
<td>87</td>
<td>44</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>NYHA class II-IV</td>
<td>108</td>
<td>34</td>
<td>111</td>
<td>56</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Cardiomegaly (radiographic)</td>
<td>151</td>
<td>48</td>
<td>130</td>
<td>66</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Clinical CHF</td>
<td>86</td>
<td>27</td>
<td>83</td>
<td>42</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Abbreviations: NYHA = New York Heart Association functional classification; CHF = congestive heart failure; MI = myocardial infarction.
HV group (NS). Cardiovascular mortality (including sudden death) occurred in 72 patients (22%) from the normal HV group and 76 (38%) from the prolonged HV group ($p < 0.001$).

Figure 2 presents the total cumulative mortality, cumulative sudden death mortality, and total cardiovascular mortality (including sudden death). At 7 years, total cumulative mortality was 48% in the normal HV group and 66% in the prolonged HV group ($p < 0.001$). Cumulative 7-year total sudden death mortality was 22% in the normal HV group and 35% in the prolonged HV group ($p < 0.005$). Cumulative 7-year total cardiovascular mortality (including sudden death) was 32% in the normal HV group and 57% in the prolonged HV group ($p < 0.001$).

**Discussion**

In patients with bifascicular block, the HV interval is a measure of conduction time in the distal His bundle and the remaining functioning fascicle. Prolongation of the HV interval in patients with bifascicular block suggests trifascicular disease. Patients with bifascicular block and prolonged HV interval should be at higher risk for subsequent trifascicular block than those with bifascicular block and normal HV interval.

The prognostic significance of the HV interval in patients with chronic bifascicular block is controversial. Many workers have reported a higher incidence of progression of AV block and a higher mortality in patients with bifascicular block and prolonged HV interval than those with a normal HV interval. Narula et al. reported the results of follow-up in 95 patients with chronic bifascicular block. Of these, 26 had normal HV and 69 had prolonged HV intervals (> 45 msec). AV block was documented in two patients (8%) with normal HV and 29 (42%) with prolonged HV intervals over a mean follow-up of longer than 3 years. Mortality was significantly higher in the patients with prolonged HV. At least 14 of 69 patients with prolonged HV intervals had a history of second- or third-degree AV block either before or at the time of electrophysiologic study.

Scheinman and co-workers reported results of follow-up (mean 8 months) in 19 patients with bundle branch block and transient neurologic symptoms (dizziness and/or syncope). Of these, the cause of symptoms was clearly related to documented complete AV block in seven. HV intervals in these patients were prolonged (range 60-120 msec; mean 89 msec). In contrast, the HV in the other 12 patients in whom cause of symptoms was unclear ranged from 45-80 msec (mean 61 msec). In a subsequent study, Scheinman et al. reported 88 patients with bundle branch block, 57 with HV intervals less than 70 msec and 31 with HV intervals 70 msec or longer. Eight of 88 patients (9%) developed AV block over a mean follow-up of 18 months. In the patients who developed AV block, the HV interval was more than 70 msec. AV block was not noted in the patients with HV less than 70 msec.

The association between a prolonged HV interval and development of AV block in patients with bifascicular block has also been supported by several retrospective studies. Gupta and associates documented HV prolongation in 15 of 16 patients (92%)

**Table 3. Electrocardiographic Findings in 517 Patients**

<table>
<thead>
<tr>
<th></th>
<th>Normal HV (n = 319)</th>
<th>Prolonged HV (n = 198)</th>
<th></th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SEM</td>
<td>n</td>
<td>%</td>
<td>Mean ± SEM</td>
</tr>
<tr>
<td>PR interval (sec)</td>
<td>0.18 ± 0.002</td>
<td>319</td>
<td>-</td>
<td>0.21 ± 0.003</td>
</tr>
<tr>
<td>QRS duration (sec)</td>
<td>0.14 ± 0.003</td>
<td>307</td>
<td>-</td>
<td>0.14 ± 0.002</td>
</tr>
<tr>
<td>PVC</td>
<td>-</td>
<td>63</td>
<td>20</td>
<td>-</td>
</tr>
<tr>
<td>LBBB</td>
<td>-</td>
<td>51</td>
<td>16</td>
<td>-</td>
</tr>
<tr>
<td>RBBB</td>
<td>-</td>
<td>268</td>
<td>84</td>
<td>-</td>
</tr>
</tbody>
</table>

**Table 4. Electrophysiologic Data in 517 Patients**

<table>
<thead>
<tr>
<th></th>
<th>Normal HV (n = 319)</th>
<th>Prolonged HV (n = 198)</th>
<th></th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SEM</td>
<td>n</td>
<td></td>
<td>Mean ± SEM</td>
</tr>
<tr>
<td>HV (msec)</td>
<td>46 ± 0.4</td>
<td>319</td>
<td></td>
<td>69 ± 0.9</td>
</tr>
<tr>
<td>AH (msec)</td>
<td>105 ± 2.4</td>
<td>307</td>
<td></td>
<td>105 ± 2.6</td>
</tr>
<tr>
<td>AH (&gt; 130 msec)</td>
<td>-</td>
<td>57</td>
<td>(18%)</td>
<td>-</td>
</tr>
<tr>
<td>Atrial ERP (msec)</td>
<td>265 ± 3.7</td>
<td>202</td>
<td></td>
<td>260 ± 4.6</td>
</tr>
<tr>
<td>AV nodal ERP (msec)</td>
<td>357 ± 7.1</td>
<td>115</td>
<td></td>
<td>359 ± 7.9</td>
</tr>
<tr>
<td>HPS ERP (msec)</td>
<td>430 ± 31.5</td>
<td>16</td>
<td></td>
<td>405 ± 27.4</td>
</tr>
<tr>
<td>SRT (msec)</td>
<td>1005 ± 18.7</td>
<td>258</td>
<td></td>
<td>1039 ± 22.3</td>
</tr>
</tbody>
</table>

*By study design.

**Abbreviations:** ERP = effective refractory periods; SRT = sinus node recovery time; HPS = His-Purkinje system.
with bifascicular block associated with intermittent Mobitz type II block or complete AV block. Vera and co-workers\textsuperscript{23} reported findings in 50 patients with transient episodes of second or third-degree AV block distal to the His bundle. During periods of intact AV conduction, His bundle recordings revealed HV prolongation in 49 of 50 (98%) and 37 (74%) had HV intervals of 75 msec or greater. Studies by Narula and Samet\textsuperscript{24} in a small number of cases with AV block tend to support the close relationship between HV prolongation and subsequent risk of AV block in patients with chronic bifascicular block.

There are several studies concerning life history of chronic bifascicular block not demonstrating HV prolongation as a predictor of subsequent AV block. One of these was an early report of follow-up of part of the present group of patients.\textsuperscript{4} We demonstrated development of spontaneous AV block in two of 86 (3%) bifascicular block patients with normal HV and one of 33 (3%) with a prolonged HV interval over a mean follow-up of 563 \pm 34 days (NS). The incidence of total cumulative mortality and mortality due to sudden death was similar in the two groups. In another early study from our laboratories, we demonstrated that markedly prolonged HV interval (80 msec or greater) in patients with bifascicular block was associated with a low incidence (6%; one of 18 patients) of subsequent AV block over a mean follow-up of 711 \pm 118 days.\textsuperscript{13}

McAnulty and co-workers\textsuperscript{25} reported prospective follow-up of 242 patients with chronic bifascicular block who underwent electrophysiologic study. Of these, 132 had HV prolongation and 110 a normal HV interval. The incidence of AV block was 5% (six of 132) in the group with HV prolongation and 7% (seven of 110) in those with normal HV intervals over a mean follow-up of 2 years (NS). Cumulative 3-year sudden death mortality was 23% in patients with a prolonged HV and 21% with a normal HV (NS). Haft et al. reported five patients with intermittent AV block who had normal HV intervals during periods of intact AV conduction.\textsuperscript{26}

We believe that the present study delineates the clinical associations of prolonged HV interval in patients with chronic bifascicular block. We demonstrate a strong association of prolonged HV interval and severity of organic heart disease in patients with chronic bifascicular block. Patients with prolonged HV have a significantly higher incidence of angina, previous infarction, symptoms and signs of congestive failure, cardiomegaly, more advanced cardiac functional class and premature ventricular contractions on resting ECG. The specific association of prolonged HV with a greater incidence of arteriosclerotic heart disease and primary myocardial disease probably reflects the characteristic of the population being studied, e.g. the extent of disease in our patients with these two disease entities. The significantly higher
cumulative total, cardiovascular, and sudden death mortalities noted in the patients with bifascicular block and prolonged HV, is consistent with the more extensive heart disease seen in this group.

We can reconcile our data concerning HV interval with previous data using Bayesian theory. According to the Bayes theorem, the predictive value of a given test will relate to the prevalence of the tested disease in the population being studied. Prolonged HV appears to predict development of AV block in patients with bifascicular block when the incidence of AV block is substantial, as in the previous series of Scheinman et al. and Narula and in the present series. In contrast, a prolonged HV interval did not predict development of AV block when the risk of AV block was very small, as in our early series and that of McAnulty et al.

One can ask why the HV interval is now predictive of trifascicular block in our present study. Our study incorporates 517 bifascicular block patients with a mean follow-up of 3.7 years. Although the incidence of spontaneous AV block is still low, we have accumulated enough patients with long enough follow-up to allow us to delineate the predictive capability of HV interval. We have not performed multivariate analysis to determine the independence of HV as a predictor of either AV block or mortality. We have also noted a significant difference in the incidence of AV block in regard to specific types of bifascicular block.

We believe that the HV interval is useful for evaluating patients with chronic bifascicular block and suspected AV block. In bifascicular block patients with idiopathic recurrent syncope, prolonged HV is a finding implicating intermittent trifascicular block as a cause of symptoms. However, recent evidence suggests that permanent pacing of patients with bundle branch block and prolonged HV interval does not ameliorate the future risk of sudden death. The finding of a normal HV interval in a patient with chronic bifascicular block suggests a very low risk of trifascicular block.

In asymptomatic subjects with chronic bifascicular block, the risk of AV block is low, and the sensitivity and specificity of a prolonged HV interval in predicting AV block would presumably be low, so that His bundle recording would not be indicated as a routine diagnostic procedure. In specific patient groups with a high risk of AV block, such as patients with calcific aortic stenosis, myotonia dystrophica, polymyositis, and Kearns-Sayre syndrome (ophthalmoplegia, retinitis pigmentosa, and distal conduction disease), prolonged HV interval might be sensitive and specific in predicting risk of AV block.

However, in each of these disease categories, the value of HV should be prospectively determined.

References

Left Fascicular Blocks During Right-heart Catheterization Using the Swan-Ganz Catheter

AGUSTIN CASTELLANOS, M.D., ANTONIO V. RAMIREZ, M.D., ALVARO MAYORGA-CORTES, M.D., KYRIACOS PEFKAROS, M.D., JOHN J. ROZANSKI, M.D., CHARLES SPRUNG, M.D., AND ROBERT J. MYERBURG, M.D.

SUMMARY During insertion of Swan-Ganz catheters, mechanical right bundle branch block occurred in association with left posterior fascicular block in two patients and with left anterior fascicular block in two. None of the four patients had acute myocardial infarction or acute (spontaneous or iatrogenic) pulmonary disease. In two cases, electrophysiologic studies demonstrated the coexistence of intra- and infra-Hisian conduction delays and blocks. Although the right bundle branch block may have resulted from injury to the central or peripheral right branch, the left fascicular blocks could not be explained by direct trauma to these left-sided structures. Our findings support the recent clinical and experimental reports that show that left fascicular block (as well as right bundle branch block) may be due to lesions involving the His bundle; presumably because of longitudinal dissociation of this structure affecting the transverse interconnections. In one patient, 2:1 intra-Hisian block may have coexisted with bradycardia-dependent (phase 4) right bundle branch block. More studies are required to determine the implications of catheter-induced conduction disturbances in other clinical settings, such as acute myocardial infarction.

RARELY, complications result from the use of the Swan-Ganz catheters. Recent communications have reported distal migration of catheters with pulmonary artery injury or pulmonary infarction. Rhythm disorders and conduction disturbances, such as ventricular ectopic beats and traumatic right bundle branch block (RBBB), have also been noted. Because the catheter is confined to the right-heart cavi- ties, axis shifts characteristic of left anterior fascicular block (LAFB) and left posterior fascicular block (LPFB) would not be expected to develop coincident with traumatic RBBB. However, these unusual associations can occur and are discussed in this report. We also examine possible electrophysiologic mechanisms.

Materials and Methods

Table 1 lists clinical data for four patients who, during insertion of a Swan-Ganz catheter, developed RBBB with LPFB (cases 1 and 2) or with LAFB (cases 3 and 4). Although one patient had old anteroseptal (and possibly inferior) myocardial infarctions, none had acute myocardial infarction by clinical, enzymatic or electrocardiographic findings, or acute pulmonary disease by clinical or x-ray findings or lung
Significance of the HV interval in 517 patients with chronic bifascicular block.
R C Dhingra, E Palileo, B Strasberg, S Swiryn, R A Bauernfeind, C R Wyndham and K M Rosen

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