Reevaluation of Enhanced Atrioventricular Nodal Conduction: Evidence to Suggest a Continuum of Normal Atrioventricular Nodal Physiology

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SUMMARY. The syndrome of enhanced atrioventricular nodal (AVN) conduction has been defined arbitrarily by: AH interval during normal sinus rhythm (AH-NSR) ≤ 60 msec; shortest right atrial pacing cycle length (PCL) maintaining 1:1 AVN conduction (shortest PCL 1:1) ≤ 300 msec; and at the shortest PCL 1:1, an increase in the AH interval from AH-NSR (ΔAH) ≤ 100 msec. We examined the relationship between AH-NSR, shortest PCL 1:1, and ΔAH in 160 consecutively studied patients who did not have accessory AV pathways or second-degree AV block to determine whether a distinct subgroup of patients with unusually rapid AVN conduction properties could be identified. The frequency distribution of each of the variables was unimodal and continuous. Cluster analysis, combining the three variables, failed to reveal a distinct subgroup at the lower end of the spectrum. Sixty-six patients (41%) had AH-NSR ≤ 60 msec, 36 (23%) shortest PCL 1:1 ≤ 300 msec, 76 (48%) ΔAH ≤ 100 msec, and 17 (11%) all three criteria. The shape of the AH vs atrial PCL curve was independent of shortest PCL 1:1. Neither ΔAH nor the terminal slope of the curve for AH vs atrial PCL (measured over the 20-40 msec below Wenckebach block) was related to AH-NSR or shortest PCL 1:1. We conclude that a subgroup cannot be identified by AH-NSR, shortest PCL 1:1, and ΔAH, and that enhanced AVN conduction as previously defined represents simply one end of the continuous spectrum of normal AVN physiology.

The syndrome of enhanced atrioventricular nodal (AVN) conduction was designated to describe a population of patients who, at electrophysiologic study, had a short AVN conduction time (AH interval) and were capable of 1:1 AVN conduction at rapid atrial pacing rates with minimal prolongation of AVN conduction time.\textsuperscript{1-4} Mechanisms proposed for this electrophysiologic response include: conduction over an accessory atrio-Hisian or atrionodal pathway, bypassing the region of major conduction delay in the AV node;\textsuperscript{5-14} an underdeveloped or anatomically small AV node;\textsuperscript{1, 2, 4, 9, 15} and an anatomically normal AV node with rapid conduction properties, either intrinsically or as a result of autonomic tone.\textsuperscript{1-4, 10, 16-18} Histologic correlation is not available.

The original criteria for enhanced AVN conduction were an AH interval ≤ 60 msec during sinus rhythm, 1:1 conduction from the atrium to His bundle at cycle lengths ≤ 300 msec during incremental right atrial pacing, and at the shortest pacing cycle length (PCL) maintaining 1:1 AVN conduction, an AH interval ≤ 100 msec longer than the AH interval recorded during sinus rhythm.\textsuperscript{7} The third criterion was later broadened, requiring AH prolongation of ≤ 100 msec at an atrial PCL of 300 msec rather than at the shortest conducting cycle length.\textsuperscript{2, 4} These criteria, arbitrarily established, have not been evaluated critically. Therefore, we examined multiple parameters of AVN conduction and refractoriness in a large group of adult patients undergoing electrophysiologic study to determine whether a distinct subgroup of patients who had unusually rapid AVN conduction properties could be identified, and in so doing, evaluated the specificity of the above criteria for enhanced AVN conduction.

Methods

Study Population

AVN function was evaluated in 160 consecutive patients (106 men and 54 women) undergoing electrophysiologic study for evaluation of brady- and tachyarrhythmias and syncope of undetermined etiology. Patients were 16–81 years (mean 45 years), were not receiving cardioactive drugs, did not have an accessory AV pathway (manifest or concealed), and did not have a history of second- or third-degree AV block. Twenty-nine patients had coronary heart disease, two hypertensive heart disease, 11 congestive and two hypertrophic cardiomyopathy, two rheumatic heart disease, 15 mitral valve prolapse, one patient atrial septal defect and one long QT syndrome. Ninety-seven patients had no evidence of structural heart disease by invasive or noninvasive evaluation. Before or during electrophysiologic study, nine patients had ectopic atrial tachycardia, 21 atrial fibrillation or flutter, 10 AVN reentrant tachycardia, eight supraventricular tachycardia of undetermined type, 84 ventricular tachycardia and 13 sinus node dysfunction. (Twenty-three of the above patients demonstrated two arrhythmias and were listed twice.) The remaining 38 patients demonstrated no arrhythmia during electrocardio-
graphic monitoring or electrophysiologic study, and the etiology of their symptoms of syncope or palpitations is not known.

Electrophysiologic Study

Patients were studied in the postabsorptive, nonseparated state after giving written informed consent. Two to four multipolar electrode catheters (10-mm inter-electrode spacing) were introduced percutaneously and guided fluoroscopically to the high right atrium, across the tricuspid valve (His bundle electrogram), to the right ventricular apex and, in many patients, into the coronary sinus. Intracardiac bipolar electrograms (filtered at 30–500 Hz) and standard ECG leads I, II, III and V1 (filtered at 0.1–20 Hz) were displayed simultaneously on a multichannel oscilloscope and recorded at paper speeds of 100 and 150 mm/sec.

Electrical stimulation was performed using a programmable stimulator (Medtronic model 5325) delivering 1.8-msec constant-current pulses at twice late diastolic threshold. The atrial stimulation protocol included high right atrial pacing at incremental cycle lengths, beginning with a cycle length just shorter than the spontaneous cycle length and shortening by 10–20 msec every eight to 12 complexes until AVN block occurred (141 patients) or until limited by patient symptoms (19 patients); and AVN refractory period determinations at one or more right atrial PCLs using extrastimulation. Atrial fibrillation was induced in 18 patients by atrial extrastimuli or short bursts of rapid atrial pacing.

Electrophysiologic Measurements

The AH interval was measured to the nearest 5-msec interval on the His bundle electrogram from the onset of the first rapid atrial deflection to the beginning of the His bundle deflection. The initial portion of the His deflection was chosen because it denotes that conduction has traversed the AV node and begun to activate the His bundle. The AH interval measured by this technique will be shorter than the AH interval measured from the first rapid atrial deflection to the first rapid His deflection. The AH interval was measured during sinus rhythm and during incremental atrial pacing at cycle lengths of 600, 500, 400 and 300 msec, and at each of the five 10-msec intervals before AVN block (fig. 1). The magnitude of AH prolongation during atrial pacing (DAH) was calculated by subtracting the AH interval during sinus rhythm from the AH interval at the shortest PCL maintaining 1:1 AVN conduction (shortest PCL 1:1). The slope of the curve of AH interval vs atrial PCL was measured at the terminal segment, just before AVN block. The slope was fit visually between the AH interval at the shortest PCL 1:1 and the AH interval at a PCL 10–40 msec longer.

The effective refractory period (ERP) of the AV node was defined as the longest A1,A2 interval, measured on the His bundle electrogram, at which A2 failed to produce His bundle depolarization. The AVN functional refractory period (FRP) was the shortest obtainable H1,H2 interval during atrial stimulation. If

the premature atrial complex at the shortest obtainable A1,A2 interval (atrial FRP) resulted in His bundle depolarization, the AVN ERP was listed as shorter than the shortest A1,A2 and the FRP as the same or shorter than the shortest H1,H2. These values were considered indeterminate (limited by atrial refractoriness) and were not used in statistical analyses. For comparison, only refractory periods determined at atrial PCLs of 450–549 msec or 550–649 msec were used.

Data Analysis

Parameters of AVN conduction and refractoriness were examined singly (frequency distribution), in pairs (x,y plots), and in groups of three to five variables (cluster analysis) in an attempt to identify a distinct subgroup of patients who had unusually rapid AVN conduction or short AVN refractoriness or both. Cluster analyses that forced the study population into two and three groups were used. Linear regression analysis was used to assess the relationship between pairs of variables. AVN refractory periods and shortest PCL 1:1 values that were not determined definitively (due to limitation by atrial refractoriness or premature termination of incremental atrial pacing) were excluded from linear regression analyses but were used in cluster analyses and are plotted as separate symbols in figures 2–7. An analysis of variance was used to evaluate the difference in slope of matched segments along the incremental atrial pacing curves between groups of patients in different ranges of shortest PCL 1:1.

Results

AH Interval During Sinus Rhythm and Shortest PCL 1:1

The distribution of AH interval measured during sinus rhythm (fig. 2) was continuous. No subgroup was evident at the lower end. The AH interval was linearly related to, but poorly predictive of shortest PCL 1:1 (fig. 2, table 1). Of 66 patients with an AH ≤
60 msec, only 27 (41%) maintained 1:1 AVN conduction to an atrial PCL ≤ 300 msec. Of 94 patients with an AH > 60 msec, 1:1 conduction was maintained to a cycle length ≤ 300 msec in nine. Two additional patients might have maintained 1:1 conduction to cycle lengths ≤ 300 msec since atrial pacing was terminated before AVN block at cycle lengths of 310 and 330 msec. Thus, the recorded AH interval during sinus rhythm does not appear to be a good criterion to identify patients who will exhibit 1:1 conduction at rapid atrial paced rates.

The AH interval was also linearly related to age and spontaneous sinus cycle length, although the rate of change in AH with each of these variables was small.
Figure 5. Magnitude of AH interval prolongation during incremental right atrial pacing (ΔAH) plotted as a function of shortest atrial pacing cycle length maintaining 1:1 atrioventricular nodal conduction (shortest PCL 1:1). Symbols are as in figure 3.

(slope = 0.42 msec/year and 0.02 msec AH/msec sinus cycle length, respectively) and scatter was large (table 1).

The frequency distribution for shortest PCL 1:1 is listed in figure 2. The necessity of terminating incremental atrial pacing before AVN block in 19 patients limits the definition of a distinct subgroup at the lower end. However, if a lower limit was to be defined, it would have to be at a value less than 270 msec, since the distribution is continuous at least to that level. Thirty-six patients (23%) maintained 1:1 AVN conduction to atrial PCLs ≤ 300 msec, including three patients who had an AH interval ≥ 95 msec during normal sinus rhythm.

The shortest PCL 1:1 was not related to age and was only poorly related to spontaneous sinus cycle length (fig. 3, table 1). The shortest PCL 1:1 correlated highly with AVN ERPs and FRPs, especially those determined at the shorter atrial PCL (fig. 4, table 1). Examining the shortest PCL 1:1 and ERP together, representing parameters of AVN conduction and refractoriness, failed to identify a distinct subgroup at the lower end. An AVN ERP shorter than the atrial FRP, even at the shorter atrial PCL, was not selective for patients who had a shortest PCL 1:1 ≤ 300 msec, as this was found in 14 of 46 patients (30%) with a shortest PCL 1:1 > 300 msec (fig. 4).

ΔAH

The magnitude of AH prolongation at the shortest
TABLE 1. Linear Regression Analyses (y = mx + b)

<table>
<thead>
<tr>
<th>Dependent variable (y)</th>
<th>Independent variable (x)</th>
<th>Slope* (m)</th>
<th>y intercept* (b)</th>
<th>No. of pts</th>
<th>r</th>
<th>R²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shortest PCL 1:1</td>
<td>AH&lt;sub&gt;NSR&lt;/sub&gt;</td>
<td>2.3 ± 0.2</td>
<td>216 ± 18</td>
<td>141</td>
<td>0.63</td>
<td>0.40</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AH&lt;sub&gt;NSR&lt;/sub&gt;</td>
<td>Patient age (years)</td>
<td>0.42 ± 0.11</td>
<td>52 ± 5</td>
<td>160</td>
<td>0.19</td>
<td>0.04</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>AH&lt;sub&gt;NSR&lt;/sub&gt;</td>
<td>SCL</td>
<td>0.02 ± 0.01</td>
<td>51 ± 9</td>
<td>160</td>
<td>0.19</td>
<td>0.04</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Shortest PCL 1:1</td>
<td>Age</td>
<td>—</td>
<td>—</td>
<td>141</td>
<td>—</td>
<td>—</td>
<td>NS</td>
</tr>
<tr>
<td>Shortest PCL 1:1</td>
<td>SCL</td>
<td>0.14 ± 0.38</td>
<td>268 ± 32</td>
<td>141</td>
<td>0.30</td>
<td>0.9</td>
<td>&lt;0.001</td>
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<tr>
<td>Shortest PCL 1:1</td>
<td>ERP (CL 450-549)</td>
<td>1.0 ± 0.8</td>
<td>56 ± 27</td>
<td>38</td>
<td>0.90</td>
<td>0.81</td>
<td>&lt;0.001</td>
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<tr>
<td>Shortest PCL 1:1</td>
<td>ERP (CL 550-649)</td>
<td>0.8 ± 0.1</td>
<td>149 ± 42</td>
<td>43</td>
<td>0.71</td>
<td>0.50</td>
<td>&lt;0.001</td>
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<tr>
<td>Shortest PCL 1:1</td>
<td>FRP (CL 450-549)</td>
<td>1.0 ± 0.1</td>
<td>-33 ± 42</td>
<td>38</td>
<td>0.86</td>
<td>0.74</td>
<td>&lt;0.001</td>
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<tr>
<td>Shortest PCL 1:1</td>
<td>FRP (CL 550-649)</td>
<td>0.8 ± 0.1</td>
<td>30 ± 40</td>
<td>43</td>
<td>0.81</td>
<td>0.65</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ΔAH</td>
<td>AH&lt;sub&gt;NSR&lt;/sub&gt;</td>
<td>—</td>
<td>—</td>
<td>160</td>
<td>—</td>
<td>—</td>
<td>NS</td>
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<tr>
<td>ΔAH</td>
<td>Shortest PCL 1:1</td>
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<td>—</td>
<td>141</td>
<td>—</td>
<td>—</td>
<td>NS</td>
</tr>
<tr>
<td>Slope</td>
<td>Shortest PCL 1:1</td>
<td>—</td>
<td>—</td>
<td>137</td>
<td>—</td>
<td>—</td>
<td>NS</td>
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</table>

During atrial fibrillation

<table>
<thead>
<tr>
<th>Dependent variable (y)</th>
<th>Independent variable (x)</th>
<th>Slope* (m)</th>
<th>y intercept* (b)</th>
<th>No. of pts</th>
<th>r</th>
<th>R²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shortest RR interval</td>
<td>AH&lt;sub&gt;NSR&lt;/sub&gt;</td>
<td>3.0 ± 1.0</td>
<td>188 ± 71</td>
<td>18</td>
<td>0.61</td>
<td>0.37</td>
<td>&lt;0.005</td>
</tr>
<tr>
<td>Shortest RR interval</td>
<td>Shortest PCL 1:1</td>
<td>1.1 ± 0.2</td>
<td>12 ± 64</td>
<td>15</td>
<td>0.88</td>
<td>0.77</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mean RR interval</td>
<td>AH&lt;sub&gt;NSR&lt;/sub&gt;</td>
<td>3.7 ± 1.7</td>
<td>242 ± 123</td>
<td>18</td>
<td>0.47</td>
<td>0.22</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Mean RR interval</td>
<td>Shortest PCL 1:1</td>
<td>1.4 ± 0.3</td>
<td>31 ± 105</td>
<td>15</td>
<td>0.82</td>
<td>0.67</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

Slope and intercept values are mean ± SEM.

Abbreviations: Shortest PCL 1:1 = shortest right atrial pacing cycle length maintaining 1:1 atrioventricular (AV) nodal conduction (msec); AH<sub>NSR</sub> = AH interval measured during sinus rhythm (msec); SCL = spontaneous sinus cycle length (msec); ERP = AV nodal effective refractory period (msec); FRP = AV nodal functional refractory period (msec); CL = cycle length (msec); ΔAH = magnitude of AH prolongation during incremental right atrial pacing (msec); Slope = negative value of the slope of the terminal segment of the incremental atrial pacing curve (AH vs atrial pacing CL) (mm/mm).

PCL 1:1 (ΔAH) varied widely, from 15 to 315 msec, and was not related to the AH interval during sinus rhythm or the shortest PCL 1:1 (table 1, fig. 5). The distribution of ΔAH was continuous at the lower end to at least 50 msec. Of the eight patients who had ΔAH < 50 msec, only four maintained 1:1 AVN conduction to atrial PCLs ≤ 300 msec. In three of these four patients, incremental atrial pacing was terminated before AVN block, precluding exact determination of ΔAH. The ΔAH ≤ 100 msec was observed in 76 of 160 patients (48%), 21 of whom maintained 1:1 AVN conduction to cycle lengths ≤ 300 msec (fig. 5). Therefore, the criterion of ΔAH ≤ 100 msec was not useful in defining a subgroup of patients with rapid AVN conduction.

The AH interval measured at the shortest atrial PCL that maintained 1:1 AVN conduction demonstrated a near-normal distribution, continuous at the lower end (fig. 8).

Values for the terminal slope of the incremental atrial pacing curves (AH vs cycle length, see figure 1)
were continuous at the lower end, and were totally unrelated to the AH interval measured during sinus rhythm or the shortest PCL 1:1 (fig. 6, table 1). Since "relatively" small changes in AH interval during incremental pacing has been used as a criterion for enhanced AVN conduction, one might have expected the terminal slope of the AVN curves to be relatively flat only in these patients who had a PCL 1:1 ≤ 300 msec. However, relatively flat terminal segments were found in many patients (slope ≤ 0.33 in 11 patients and ≤ 0.5 in 29), spanning the range of shortest PCL 1:1 from 250 to 600 msec (fig. 6).

The observation that neither ΔAH nor terminal slope of the incremental atrial pacing curves correlated with shortest PCL 1:1 implied that the shape of the curves was independent of shortest PCL 1:1. The similarity in shape of the curves throughout the range of shortest PCL 1:1 can be demonstrated by dividing the population into four groups based on shortest PCL 1:1 and plotting mean values for AH interval at selected pacing cycle lengths (fig. 9). Group 1 had shortest PCL 1:1 ≤ 600 msec but > 500 msec, group 2 the shortest PCL 1:1 ≤ 500 msec but ≥ 400 msec, group 3 the shortest PCL 1:1 < 400 but ≥ 300 msec, and group 4 the shortest PCL < 300 msec. AH interval (mean ± SEM) is plotted for each group at cycle lengths of 600, 500, 400, and 300 msec, and at the mean shortest PCL 1:1. There was no significant difference between groups in the slope of segment between the last full 100-msec interval and the mean shortest PCL 1:1 (segment A, groups 1–4) the 100 msec interval before that segment (segment B, groups 2–4) or the 100-msec interval before that segment (segment C, groups 3 and 4). These data are important, for they demonstrate the universality of the shape of AVN conduction curves regardless of whether patients can sustain 1:1 AVN conduction at rapid or only slow atrial pacing rates.

Enhanced AVN Conduction

Cluster analysis using AH interval during sinus rhythm, the shortest PCL 1:1 and ΔAH, alone and in combination with terminal slope of incremental atrial pacing curve, AVN ERP (cycle lengths 450–549 msec and/or 550–649), or spontaneous sinus cycle length failed to identify a distinct subgroup of patients with unusually short values for these variables of AVN conduction and refractoriness. Each of the three original criteria for enhanced AVN conduction (AH ≤ 60 msec, shortest PCL 1:1 ≤ 300 msec, and ΔAH ≤ 100 msec) fell within the continuous portion of the respective frequency distribution (figs. 2 and 5). Forty-nine patients (31%) fulfilled at least two criteria, and 17 patients (11%) fulfilled all three criteria (fig. 7). For those 17 patients, the sinus cycle length ranged from 515 to 920 msec (median 645 msec). Only six of the 17 patients underwent electrophysiologic study for evaluation of supraventricular tachyarrhythmias.

AH Interval and Shortest PCL 1:1 as Predictors of Ventricular Response During Atrial Fibrillation

The shortest and mean RR intervals during induced atrial fibrillation in 18 patients correlated highly with the shortest atrial PCL maintaining 1:1 AVN conduction and poorly with the AH interval measured during sinus rhythm (fig. 10, table 1). The mean RR interval ≤ 380 msec (rate ≥ 158/min) occurred in five of seven patients with shortest PCL 1:1 ≤ 300 msec and in none of 11 patients with shortest PCL 1:1 > 300 msec.

Discussion

After examining measurements of AVN function in 160 patients undergoing electrophysiologic study, we could not identify a subgroup of patients demonstrating unusually rapid AVN conduction and short AVN refractoriness. The distribution of each of the variables was continuous and unimodal, whether examined singly or in combination. Each of the arbitrary criteria for enhanced AVN conduction fell well inside the lower end of the respective distribution. We conclude that enhanced AVN conduction as previously described represents simply one end of the continuous spectrum of normal AVN physiology. The normal AVN response to propranolol16 and verapamil17 in patients who have these criteria further supports our conclusion.

Our findings do not exclude the occurrence of true atrio-Hisian accessory pathways. Brechenmacher identified tracts connecting the atrium with the penetrating portion of the His bundle in two of 687 hearts (0.03%) studied postmortem. However, to our knowledge, patients reported to have electrophysiologic properties strongly suggestive of a functioning atrio-Hisian pathway are very uncommon.22, 23
The data in figure 9 reflect the extremely wide spectrum of human AVN physiology. The shape of the atrial pacing curves are similar regardless of the range of shortest PCL 1:1. It has been postulated that the AVN functional properties of patients at the shorter end of the spectrum (and fulfilling the three prior criteria for enhanced AVN conduction) are the result of increased sympathetic or decreased parasympathetic tone. This may be the case for some patients, since nine of 17 patients had sinus rates $\geq$ 93/min (cycle length $\leq$ 645 msec), possibly reflecting increased sympathetic tone, at the time of electrophysiologic study. However, the effects of the autonomic nervous system on sinus nodal automaticity do not necessarily correlate with autonomic effects of AVN conduction. Further, rapid AVN conduction represents intrinsic AVN function in some patients, as two of nine such patients continued to exhibit equally rapid conduction after autonomic blockade with atropine and propranolol. Two patients we previously reported also had rapid AVN conduction after i. v. atropine (0.03 mg/kg) and propranolol (0.15 mg/kg).

Several other observations are noteworthy. AVN refractoriness is less in children than adults, suggesting that age may be related to AVN function. However, in this series of patients, ages 16–81 years (mean 45 years), the shortest PCL 1:1 was unrelated to age. In fact, 10 of 36 patients with a shortest PCL 1:1 $\leq$ 300 msec were older than 50 years. The AH interval measured during sinus rhythm also correlated with shortest PCL 1:1. A similarly weak relationship between AH interval and shortest PCL 1:1 ($r = 0.41$) was found for the fast AVN pathway in a large population of patients with dual functional AVN pathways. AH interval was also a poor predictor of ventricular response during atrial fibrillation. Conversely, the shortest PCL 1:1 correlated highly with the shortest and mean RR intervals during atrial fibrillation, confirming the findings of an earlier report.

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