Effects of the Pacing Site on A-H Conduction and Refractoriness in Patients with Short P-R Intervals

JUAN ARANDA, M.D., AGUSTIN CASTELLANOS, M.D., FEDERICO MOLEIRO, M.D., AND BENJAMIN BEFEKER, M.D.

SUMMARY His bundle recordings were studied in four patients with short P-R and A-H intervals, and narrow QRS complexes, who had experienced several episodes of supraventricular tachyarrhythmias. The heart was paced from the high right atrium (HRA) and the coronary sinus (CS). In three patients the A-H Wenckebach phenomenon occurred at higher rates (greater than 200 pacing beats/min) when the CS was paced than when pacing was performed from the HRA. Moreover, CS stimulation produced smaller increments in the A-H interval than did pacing from HRA. The extrastimulus method of testing was done. In cases 1 and 2 the functional refractory period of the A-H tissues was 15 to 25 msec shorter during CS pacing than when pacing from the HRA. In case 3, the low right atrium (LRA) as well as the other two sites were paced. A type 1 gap was seen from HRA, a type 2 gap from CS, and both types appeared when the LRA was paced. Case 4, in which the mid-right atrium (MRA) was also stimulated, had a double pathway from HRA and CS with conduction through the accessory pathway late in the cycle and through the A-V node earlier in the cycle. However, the A-V node could not be penetrated during MRA stimulation. It appeared that the pacing site influenced the A-H conduction pattern and refractoriness, possibly by changing the site and/or mode of entry of the stimulus into the pathways that are responsible for this syndrome.

Material and Methods

Conventional cardiac catheterization and specialized conducting system studies were performed in four patients with short (less than 120 msec) P-R intervals and narrow QRS complexes. Informed consent was obtained after explaining the procedures. Pertinent clinical and surface electrophysiological information is presented in table 1. Anti-arrhythmic drugs and digoxin were withheld for five days prior to the study.

Definition of Terms and Graphs

The A-H interval was the time interval between atrial and His bundle deflections recorded from the bipolar lead located over the His bundle area. In our four patients, unlike those with normal P-R intervals, the A-H interval did not necessarily reflect A-V nodal conduction time.

The terms A-H Wenckebach phenomenon and A-H tissues will be used because we could not be certain which structure(s) (A-V node, accessory pathway, or both) caused the conduction disturbance. The H-V interval gave a measure of His-Purkinje conduction time.

The St-A interval reflected conduction time from the paced site to the right atrium in the vicinity of the A-V node as recorded in the His bundle lead.

DCL = driving cycle length.

From the Cardiovascular Laboratory, Veterans Administration Hospital, and the Division of Cardiology, Department of Medicine, University of Miami School of Medicine, Miami, Florida.

Address for reprints: Dr. Agustin Castellanos, Section of Cardiology, Department of Medicine, University of Miami School of Medicine, P.O. Box 875, Biscayne Annex, Miami, Florida 33152.

Received March 14, 1975; revision accepted for publication July 2, 1975.
A1-H1, H1-V1, A2-H2, H2-V2 were the A-H intervals for driven (basic) and testing (premature) response, respectively. The A1-A2 interval measured the time that elapsed between driven and testing atrial deflections. The H1-H2 interval was that between driven and testing His bundle deflections.

The effective refractory period (ERP) of the atria was the longest St1-St2 interval at which St1 failed to depolarize the atria.7

The ERP of the A-H tissues was the longest A1-A2 interval at which A2 was not followed by an H deflection.7 According to Denes et al. the normal duration of this interval for the driving cycle length used in our patients (600 msec to 850 msec) ranges between 250 and 365 msec.8

The functional refractory period (FRP) of the A-H tissues was the shortest H1-H2 interval.7 The normal range of the FRP according to Denes et al. for the basic drive rates used in our four patients varies between 350 msec and 485 msec.6

The ERP of the most proximal part of the His-Purkinje system (at the site from which the H deflection was recorded) was given by the longest H1-H2 interval at which H2 was not followed by a QRS complex.7

Two different types of graphs were constructed. In the first type, cycle lengths were plotted against the A-H intervals. According to Caracta et al. the average increase in A-H intervals that occurs in 120 msec before second degree A-V block appears at cycle lengths of about 300 msec.6 Development of A-H Wenckebach at cycle lengths shorter than 300 msec coincided with abnormally short A-H intervals. The second type of graph was constructed by plotting A1-A2 intervals against H1-H2 intervals.7

A dual pathway response was considered to have been present if as A1-A2 intervals became progressively shorter an abrupt increase in H1-H2 intervals occurred.10

Type 1 gap is a descriptive term indicating that A2 was blocked at the A-H tissues at a given coupling interval but that conduction was resumed at even shorter coupling intervals with longer H1-H2 intervals than those at which block had occurred.11-13 These impulses were sufficiently delayed at the A-V node so as to reach the infra-A-V nodal tissues after the end of their effective refractory period.

In type 2 gap A2 was blocked at a given coupling interval and conduction resumed at even shorter coupling intervals but with H1-H2 intervals shorter than those at which block had occurred. The H2-V2 interval was longer than those occurring with test response occurring late in the cycle. In this type of gap early impulses were conducted because they encountered enough delay within the proximal His-Purkinje system (at the site from which the His bundle deflection was recorded) to give the previously refractory distal areas more time to recover.11-13

### Case Reports

**Case 1**

Pertinent information regarding this patient is shown in tables 1 and 2. The A-H interval was short and the H-V

### TABLE 1. Clinical and Electrophysiological Information on Patients During Sinus Rhythm

<table>
<thead>
<tr>
<th>Case</th>
<th>Age</th>
<th>Diagnosis</th>
<th>Arrhythmias Documented</th>
<th>ECG Diagnosis</th>
<th>P-R (120°)</th>
<th>P-A (10-40°)</th>
<th>A-H (55-120°)</th>
<th>H-V (33-55°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>48</td>
<td>HCVD</td>
<td>A fib</td>
<td>LVH?</td>
<td>115</td>
<td>30</td>
<td>40</td>
<td>45</td>
</tr>
<tr>
<td>2</td>
<td>50</td>
<td>C</td>
<td>A fib</td>
<td>LVH</td>
<td>110</td>
<td>25</td>
<td>50</td>
<td>35</td>
</tr>
<tr>
<td>3</td>
<td>31</td>
<td>C</td>
<td>AT</td>
<td>LVH</td>
<td>110</td>
<td>25</td>
<td>45</td>
<td>35</td>
</tr>
<tr>
<td>4</td>
<td>52</td>
<td>N</td>
<td>AT, AF</td>
<td>Normal</td>
<td>115</td>
<td>30</td>
<td>45</td>
<td>40</td>
</tr>
</tbody>
</table>

*All values in msec.*

**Abbreviations:** HCVD = hypertensive cardiovascular disease; A fib = atrial fibrillation; C = cardiomyopathy; AT = atrial tachycardia; N = normal heart; AF = atrial flutter; LVH = left ventricular hypertrophy.

### TABLE 2. Dynamic Electrophysiological Information (values in msec)

<table>
<thead>
<tr>
<th>Case</th>
<th>Pacing</th>
<th>A-H Increment at CL of 300</th>
<th>CL at which W occurred (&lt;300)</th>
<th>Driving CL</th>
<th>ERP (250-355°)</th>
<th>FRP (350-485°)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>HRA</td>
<td>40</td>
<td>275</td>
<td>650</td>
<td>300</td>
<td>405</td>
</tr>
<tr>
<td></td>
<td>CS</td>
<td>10</td>
<td>235</td>
<td>650</td>
<td>300</td>
<td>390</td>
</tr>
<tr>
<td>2</td>
<td>HRA</td>
<td>60</td>
<td>290</td>
<td>700</td>
<td>280</td>
<td>325</td>
</tr>
<tr>
<td></td>
<td>CS</td>
<td>45</td>
<td>275</td>
<td>700</td>
<td>280</td>
<td>300</td>
</tr>
<tr>
<td>3</td>
<td>HRA</td>
<td>70</td>
<td>290</td>
<td>670</td>
<td>&lt;290</td>
<td>380</td>
</tr>
<tr>
<td></td>
<td>CS</td>
<td>45</td>
<td>270</td>
<td>670</td>
<td>&lt;270</td>
<td>337</td>
</tr>
<tr>
<td></td>
<td>LRA</td>
<td>--</td>
<td>--</td>
<td>670</td>
<td>&lt;265</td>
<td>345</td>
</tr>
<tr>
<td>4</td>
<td>HRA</td>
<td>--</td>
<td>465</td>
<td>800</td>
<td>370,300</td>
<td>400,580</td>
</tr>
<tr>
<td></td>
<td>CS</td>
<td>--</td>
<td>380</td>
<td>800</td>
<td>365,300</td>
<td>405,580</td>
</tr>
<tr>
<td></td>
<td>MRA</td>
<td>--</td>
<td>--</td>
<td>800</td>
<td>370, ---</td>
<td>390, ---</td>
</tr>
</tbody>
</table>

*Normal values according to Caracta et al.*

†Normal values according to Denes et al.

**Abbreviations:** CL = cycle length; W = Wenckebach.
interval normal. According to some investigators these electrophysiological findings suggest the existence of accessory pathway bypass over all or a great part of the A-V node. The response at increasing rates was abnormal in view of the cycle length (< 300 msec) at which A-H Wenckebach was seen and of the magnitude of the A-H interval measured at a cycle length of 300 msec (table 2). Pacing from HRA and CS produced an increment of 40 and 10 msec, respectively (table 2). Wenckebach phenomenon occurred at a shorter cycle length (higher rate) when pacing from CS than when pacing from HRA.

The graph in figure 1 shows no appreciable differences in the ERPs of the A-H tissues during HRA and CS pacing. However, the FRP was 15 msec shorter when measured from the latter site. In addition, the CS (H1-H2) curve showed that the H2 intervals were only 5 msec apart from the oblique line of identity at coupling intervals between 690 msec and 410 msec. It thereafter shifted away from the line of identity but always closer to the latter than the HRA curve.

**Case 2**

Pertinent information from this patient is given in tables 1 and 2. Whereas the A-H interval was short, the H-V interval was within normal limits. Increments in A-H interval were less than normal during rapid atrial stimulation from both paced sites (table 2). A-H Wenckebach occurred at a shorter-than-normal cycle length, namely, 290 msec from HRA and at 275 msec from CS.

Figure 2 was similar to figure 1 in that the ERP of A-H tissues had approximately the same value from HRA and CS (table 2). The FRP was 25 msec shorter when pacing from CS than from HRA. Moreover, the CS (H1-H2) curve was so close to the line of identity that it could have been considered an example of almost pure bypass tract conduction. That is, when the CS was paced, A2 was only slightly delayed when compared with A1. The FRP was shorter (from both sites) than the values given by Denes et al. for similar driving rates in patients with normal P-R intervals.

**Case 3**

Information regarding this patient can be found in tables 1 and 2. The A-H interval was short and the H-V interval normal. A-H Wenckebach occurred at cycle lengths of 290 msec with HRA pacing and 270 msec with CS pacing, the A-H increments being 70 and 45 msec, respectively, at a cycle length of 300 msec.

Pacing with the extrastimulus technique showed that the HRA curve, as in cases 1 and 2, was located to the left of the

---

**Figure 1** (Case 1) Graph relating the A1-A2 intervals to the H1-H2 intervals during HRA and CS premature stimulation. DCL = driving cycle length. In this and all graphs values are expressed in msec. HRA = high right atrium; CS = coronary sinus; ERP = effective refractory period.

**Figure 2** (Case 2) Graph relating the A1-A2 intervals to the H1-H2 intervals during HRA and CS premature stimulation.

**Figure 3** (Case 3) Graph relating the A1-A2 intervals to the H1-H2 intervals during HRA and CS pacing. X indicates H1-H2 intervals at which H2 deflections were not followed by QRS complexes.
By guest on July 26, 2017 http://circ.ahajournals.org/ Downloaded from

Stimulation, suggesting were A-V node. The ERP exceeded 400 msec. fig. 4, left-sided panel. However, at A1-A2 interval of 310 msec, H2-H2 decreased to 390 msec and A2 was followed by an H2 deflection which failed to excite the ventricles (fig. 3 and fig. 4, second panel from left). Thus, the ERP of that part of the His-Purkinje system located immediately at the site from which the H deflection was recorded measured 390 msec. This response persisted until the A1-A2 interval was reduced to 280 msec. At 270 msec and 260 msec conduction to the ventricles again occurred (gap phenomenon) because A2 was delayed within the A-H tissues (presumably at the A-V node). In consequence, the H2-H2 intervals again exceeded 390 msec (fig. 3 and fig. 4, second panel from left). This response resembles that seen in the so-called type I gap because during the part of the cycle preceding the gap the duration of the H2-H2 intervals exceeded that seen while conduction to the ventricles was failing. Moreover, the H2-V2 intervals were not longer than those seen late in the cycle.11-13 The ERP of the A-H tissues was within normal range (table 1) suggesting that A2 might have been conducted through the A-V node.

The CS curve was closer to the line of identity than the HRA curve but when the A1-A2 intervals were reduced between 370 msec to 320 msec, H2 was unable to stimulate the ventricles while the H2-H2 values ranged between 390 and 350 msec (fig. 3). Yet, at shorter A1-A2 interval (between 310 and 260 msec) conduction to the ventricles was again possible (fig. 3 and fig. 4, right-sided panel). When this occurred the H2-H2 intervals were shorter than when block had occurred. In addition, the H2-V2 intervals were longer than during the pre-gap part of the cycle. This response is characteristic of the so-called type II gap.11-13 The FRP of the A-H tissues was shorter than normal during CS premature stimulation, suggesting that A2 could have been, at least partially, conducted through the accessory pathway.

Because of the marked differences in the HRA and CS curves the decision was made to pace the low right atrium (LRA) in the vicinity of the inferior vena cava. The corresponding LRA curve shifted to the left of the CS curve (fig. 5). Conduction to the ventricles was possible at A1-A2 intervals greater than 370 msec because the H2-H2 intervals exceeded 390 msec. In contrast, when the A1-A2 intervals fell between 360 and 320 msec, H2 was not followed by V2 because the corresponding H2-H2 intervals measured 390 msec or less (fig. 6, left panel). However, at A1-A2 intervals between 315 msec and 275 msec conduction to the ventricles was again possible with shorter H2-H2 intervals than those at


![Figure 5](case-3-graph-relating-the-A1-A2-intervals-to-the-H1-H2-intervals-during-CS-and-low-right-atrial-LRA-stimulation-the-CS-curve-shown-in-the-graph-is-the-same-as-that-present-in-figure-3-it-is-depicted-for-comparison-with-the-LRA-curve)
which block had occurred (fig. 5 and fig. 6, middle panel). A type 2 gap was present because the $H_2$-$V_2$ intervals were longer than during the pre-gap part of the cycle. At still shorter $A_1$-$A_2$ intervals (270 to 255 msec) conduction to the ventricles again occurred with longer $H_1$-$H_2$ intervals than when block had occurred (fig. 5 and fig. 6, right panel). In addition, the $H_2$-$V_2$ intervals had the same duration as late in the cycle. Thus the type 2 gap was transformed into a type 1 gap because of additional delay of $A_1$ (presumably within the A-V node) at the shortest coupling intervals.

In this patient the ERP of the A-H tissues could not be measured from any paced site because the atria became effectively refractory before $A_2$ failed to produce an $H_2$ deflection.

**Case 4**

Pertinent information regarding this patient is presented in tables 1 and 2. The A-H interval was short and the H-V interval normal. Although A-H Wenckebach phenomenon occurred at a shorter driving cycle length when the atria was paced from the CS (380 msec) than from the HRA (465 msec), these values were significantly longer than in the first three cases (table 2). Therefore, the response to atrial pacing at increasing rates was not abnormal, as in cases 1, 2, and 3. In this patient pacing was also performed from the mid-right atrium (MRA). Figure 7 shows that the HRA and CS curves had similar characteristics. Both descended very close to the line of identity at $A_1$-$A_2$ intervals ranging from 780 and 360 msec (first part of both panels in fig. 8). But when the $A_1$-$A_2$ interval was reduced to 360 msec, the $H_1$-$H_2$ values suddenly increased to 560 msec (fig. 7, and second part of both panels in fig. 8). This (dual pathway) response suggests that the ERP of the A-V node was shorter than that of the accessory pathway.

On the other hand the MRA curve descended parallel to the HRA and CS curves, but only until $A_1$-$A_2$ measured 360 msec (fig. 7 and fig. 9, left side). Thereafter, at $A_1$-$A_2$ intervals ranging from 350 to 300 msec, $A_2$ was no longer followed by $H_1$ (fig. 9, right side). Hence, the upward shift seen during HRA and CS stimulation did not occur when the MRA was paced (fig. 7). This graph suggests that, from the MRA, the A-V node (that is, the pathway with the shorter ERP) was not penetrated.

**Discussion**

The four patients studied had short P-R intervals, narrow QRS complexes, and spontaneous (and iatrogenic) supraventricular tachyarrhythmias (Lown-Ganong-Levine syndrome). During sinus rhythm the A-H interval was short and the H-V interval normal (table 1). Although several hypotheses have been proposed, most investigators believe that the shortened A-V conduction intervals result from the presence of an accessory pathway bypassing all, or a great part, of the A-V node.

The first three patients had an abnormal response to atrial pacing at increasing rates manifested by occurrence of A-H Wenckebach at cycle lengths of less than 300 msec (rates greater than 200 beats/min) (fig. 1, table 2). Second degree
A-V block invariably occurred at faster rates when paced from CS than when paced from HRA (fig. 1). A-H increments were also less from CS (table 1).

In cases 1 and 2 testing with the extrastimulus method showed that the FRPs were shorter from CS than from the HRA but only in case 2 was this value shorter than normal (according to the figures given by Denes et al.).

In case 3, where the normal FRP during HRA stimulation suggests that A₂ reached the His bundle mainly through the A-V node, the H₁-H₂ curve (and shorter-than-normal FRP) seen during CS pacing suggested that A₂ was conducted entirely through an A-V nodal bypass (with some delay in the bypass itself) or through a partial bypass (the delay occurring in the part of the A-V node that A₂ had to traverse). A type 1 gap occurred from HRA and a type 2 gap from CS.³⁵ ³⁶

Because of this paradox, pacing was also performed from the LRA in the vicinity of the inferior vena cava. From this site, A₂ followed the same route as it had when the CS was stimulated, except at shorter coupling intervals when it propagated almost exclusively through the A-V node. Both type 1 and type 2 gaps were seen from the LRA.

Case 4 had dual pathways.³⁵ At long coupling intervals conduction occurred mainly through the bypass tract. At a shorter coupling interval the impulses propagated through the A-V node only during HRA and CS pacing since they

---

**Figure 8** (Case 4) Dual pathway response during HRA and CS premature stimulation. At the shorter A₁-A₂ intervals (second part of each panel), the H₁-H₂ intervals were considerably longer than at shorter A₁-A₂ intervals (first part of each panel).

**Table**

<table>
<thead>
<tr>
<th></th>
<th>HRA</th>
<th>CS</th>
</tr>
</thead>
<tbody>
<tr>
<td>A₁ − A₂</td>
<td>380</td>
<td>350</td>
</tr>
<tr>
<td>H₁ − H₂</td>
<td>395</td>
<td>580</td>
</tr>
<tr>
<td></td>
<td>H₁ − H₂</td>
<td></td>
</tr>
<tr>
<td>I</td>
<td></td>
<td></td>
</tr>
<tr>
<td>II</td>
<td></td>
<td></td>
</tr>
<tr>
<td>III</td>
<td></td>
<td></td>
</tr>
<tr>
<td>VI</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>HRA</td>
<td>CS</td>
</tr>
<tr>
<td>H₁</td>
<td></td>
<td></td>
</tr>
<tr>
<td>H₂</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Figure 9** (Case 4) During mid-right atrial (MRA) stimulation conduction to the His bundle was possible at an A₁-A₂ interval of 360 msec. However, A₂ was not followed by an H deflection at similar A₁-A₂ intervals (350 msec) at which, during HRA and CS stimulation (last part of each panel in figure 8), conduction had been possible to the His bundle. This indicates that A₂ did not penetrate the A-V node from MRA.
could not penetrate the A-V node where the MRA was stimulated.

The present study shows that changing the pacing site can influence A-V conduction in the Lown-Ganong-Levine syndrome.

Our findings are in keeping with the studies by Batsford et al., who observed that, in 50% of their cases with normal P-R intervals, pacing from the CS produced a shortening of A-H conduction time and refractoriness. These authors postulated that the impulse delivered to the CS was preferentially conducted through an internodal tract, thus altering the site of its entry into the A-V node. This, in turn, could account for the shortened A-H interval and A-H refractory periods. However, as suggested by Janse, a change in the site and/or mode of entry into the A-V node could also influence A-H transmission without direct stimulation of an internodal tract.

In addition to the factors operating in patients with normal P-R intervals, A-H transmission in the Lown-Ganong-Levine syndrome can be influenced by: a) the type (total or partial) of A-V nodal bypass; b) the anatomical location of the bypass; c) the conduction time between the area of stimulation and bypass; d) the site and/or mode of entry into the bypass.

Moreover, in our cases, the place of origin of the atrial impulses had definite and varying (although indirect) effects on impulse propagation through the His-Purkinje system, possibly by altering the moment of arrival of excitation at the His bundle.

Unfortunately, the information obtained from the patients discussed in this report does not allow us to clearly establish the mechanisms for the differences in A-H conduction and refractoriness seen during atrial pacing from different areas. Further studies are required.

References

Effects of the pacing site on A-H conduction and refractoriness in patients with short P-R intervals.

J Aranda, A Castellanos, F Moleiro and B Befeler

doi: 10.1161/01.CIR.53.1.33

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1976 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/53/1/33

Permissions: Requests for permissions to reproduce figures, tables, or portions of articles originally published in Circulation can be obtained via RightsLink, a service of the Copyright Clearance Center, not the Editorial Office. Once the online version of the published article for which permission is being requested is located, click Request Permissions in the middle column of the Web page under Services. Further information about this process is available in the Permissions and Rights Question and Answer document.

Reprints: Information about reprints can be found online at:
http://www.lww.com/reprints

Subscriptions: Information about subscribing to Circulation is online at:
http://circ.ahajournals.org//subscriptions/