Functional 2:1 A-V Block within the His-Purkinje System
Simulation of Type II Second-Degree A-V Block

By Anthony N. Damato, M.D., P. Jacob Varghese, M.D., Anthony R. Caracta, M.D., Masood Akhtar, M.D., and Sun H. Lau, M.D.

SUMMARY
In eight subjects in whom the effective refractory period of the His-Purkinje system was determined to be longer than that of the A-V node or atrium, abrupt acceleration of the atrial rate resulted in either 2:1 block within the His-Purkinje system or 1:1 A-V conduction. The former conduction pattern simulated a type II second-degree A-V block. The occurrence of either 2:1 block within the His-Purkinje system or 1:1 A-V conduction was determined primarily by (1) an effective refractory period of the His-Purkinje system which exceeded that of the A-V node, (2) the coupling interval of the first atrial capture beat and its effect or lack thereof on the refractory period of His-Purkinje system as it relates to changes in ventricular cycle length, and (3) the relative speed of A-V nodal conduction time. The distinction between this functional 2:1 block within the His-Purkinje system and a true type II second-degree block is discussed from both the clinical and electrophysiological points of view. Functional 2:1 block within the His-Purkinje can also result when sudden acceleration of atrial rate occurs spontaneously such as in atrial flutter or A-V nodal reentrant atrial tachycardias.

Additional Indexing Words:
Atrial pacing  Atrial tachycardias  Premature atrial stimulation  A-V node  His bundle recording  Refractory periods

Generally, A-V block occurs in that portion of the A-V conducting system which has the longest effective refractory period. In most subjects, the effective refractory period of the A-V node exceeds that of the His-Purkinje system, and therefore 2:1 block at the A-V nodal level results whenever a sufficiently high rate of stimulation is applied to the atria.1,2 In such patients, A-V nodal block occurs irrespective of whether the atria are stimulated at a high rate abruptly or by increments. The specific rate at which 2:1 block occurs varies in different individuals but, in general, in normal subjects it is greater than 120 beats/min.

This report deals with the findings in eight subjects in whom abrupt acceleration of the atrial rate resulted in either 1:1 A-V conduction or 2:1 A-V block within the His-Purkinje system. The latter simulated a type II (Mobitz) second-degree A-V block. Those factors which are responsible for this “functional” form of block within the His-Purkinje will be analyzed.

Methods
Right heart catheterization was performed with the patient in the postabsorptive, nonsedated state. Informed consent was obtained. The patients were not taking any cardiovascular drugs. Using local anesthesia, a tripolar electrode catheter was percutaneously inserted into a femoral vein and fluoroscopically positioned in the region of the tricuspid valve in order to record bundle of His electrograms as previously described.3 A quadripolar electrode catheter was similarly inserted into an antecubital vein and fluoroscopically positioned against the lateral wall of the right atrium at its junction with the superior vena cava. The proximal pair of electrodes was used to record a high right atrial electrogram, and the distal pair used to stimulate the right atrium. Three standard electrocardiographic leads were simultaneously recorded. Atrial stimulation was performed using a programmed digital stimulator which delivered rectangular pulses of 2-msec duration at twice diastolic threshold through an isolation unit. In each patient two modes of atrial stimulation were performed. The extrastimulus method was used to determine the refractory periods of

From the Cardiopulmonary Laboratory, U. S. Public Health Service Hospital, Staten Island, New York.
Supported in part by the Federal Health Program Service, U. S. Public Health Service Project Py 72-1 and National Heart and Lung Institute, Projects HE 11829 and HE 12536.
Address for reprints: Anthony N. Damato, M. D., Cardiopulmonary Laboratory, U.S.P.H.S. Hospital, Staten Island, New York 10304.
Received September 28, 1972; revision accepted for publication November 12, 1972.
the A-V conduction system during sinus rhythm, and this involved the introduction of a single atrial premature depolarization at progressively shorter coupling intervals within the cardiac cycle. In the second method of stimulation the atrial rate was abruptly accelerated. Careful attention was paid to the grounding of all equipment.

The A-H interval was used as an approximation of A-V nodal conduction time. Normal values in our laboratory are 60–140 msec. The H-V interval measures the effective refractory period (ERP) of the His-Purkinje conduction system, and our normal values are 30–55 msec. The His-Purkinje system includes the bundle of His, right and left bundle branches, and peripheral Purkinje network.

Refractory Period Determinations

A single atrial premature depolarization (A₂) was introduced after every eighth sinus beat (A₁) at progressively shorter coupling intervals (A₁-A₂ interval) up to the point of atrial refractoriness.

The effective refractory period (ERP) of the atrium was defined as longest interval between the last atrial sinus beat and the delivered stimulus (S) at which S failed to capture the atrium. Effective refractory period (ERP) of the His-Purkinje system—the longest H₁-H₂ interval at which H₂ fails to propagate to the ventricles. Effective refractory period (ERP) of the A-V node—the longest A₁-A₂ interval at which A₁ fails to conduct to the bundle of His.

Relative refractory period (RRP) of the His-Purkinje system is determined by the H₁-H₂ interval at which H₂ conducts with an H₂-V₂ interval which is longer than the sinus beat (i.e., H₂-V₂ > H₁-H₂) or H₂ results in aberrant ventricular activation.

All tracings were taken on a seven-channel tape recorder using a multichannel oscillographic photographic recorder and later transferred to photographic recording paper at a speed of 150 mm/sec. Time lines were inscribed at 10- and 100-sec intervals.

Results

Table 1 lists the essential clinical data of the eight patients of this report. QRS duration and axis were normal in all patients except one who had a RBBB pattern. For each patient the electrocardiogram at the time of study was similar to that recorded 1–5 years earlier. Similarly, the electrocardiograms have not changed over a 1–2-year period following study.

Table 2 lists the electrophysiologic data obtained during sinus rhythm. In all patients A-V nodal conduction time, as measured by the A-H interval, was normal during sinus rhythm. His-Purkinje conduction time (H-V interval) was also normal. The A-H interval demonstrated the normal progressive increase as the atrial rate was gradually increased (by increments of 10 beats/min) to the maximum rate which allowed 1:1 A-V conduction. The H-V interval remained constant over this same range of paced heart rates. At sinus rhythm, the effective refractory period of the His-Purkinje system was greater than that of the A-V node in all patients. In five of the eight patients the effective refractory period of the right atrium was reached before that of the A-V node.

In every patient, sudden acceleration of the atrial rate at cycle lengths which ranged between 380 and 415 msec resulted in either 2:1 A-V block within the

<table>
<thead>
<tr>
<th>Pt</th>
<th>Age (yr), sex</th>
<th>Diagnosis</th>
<th>Symptoms</th>
<th>P-R (sec)</th>
<th>QRS complex</th>
<th>Sinus CL (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>15 F</td>
<td>NHD; frequent PVBs</td>
<td>None</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 820–960</td>
</tr>
<tr>
<td>2</td>
<td>56 M</td>
<td>ASHD</td>
<td>AP</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 810–920</td>
</tr>
<tr>
<td>3</td>
<td>75 M</td>
<td>Occasional PVBs</td>
<td>None</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 1130–1400</td>
</tr>
<tr>
<td>4</td>
<td>63 M</td>
<td>Occasional PVBs; old inferior MI</td>
<td>None</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 780–945</td>
</tr>
<tr>
<td>5</td>
<td>65 M</td>
<td>Diabetes; old anteroseptal MI</td>
<td>None</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 800–920</td>
</tr>
<tr>
<td>6</td>
<td>35 F</td>
<td>PAT due to A-V nodal reentry</td>
<td>Palpitations</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 850–1000</td>
</tr>
<tr>
<td>7</td>
<td>65 M</td>
<td>PABs; PVBs</td>
<td>None</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 1000–1350</td>
</tr>
<tr>
<td>8</td>
<td>60 M</td>
<td>Old inferior MI</td>
<td>None</td>
<td>&lt; 0.20</td>
<td>Normal duration</td>
<td>Normal axis; 1000–1350</td>
</tr>
</tbody>
</table>

Abbreviations: NHD = no heart disease; ASHD = arteriosclerotic heart disease; MI = myocardial infarction; PVB = premature ventricular beat; PAB = premature atrial beat; PAT = proxysmal atrial tachycardia; AP = angina pectoris; CL = cycle length.

Circulation, Volume XLVII, March 1973
His-Purkinje system or 1:1 A-V conduction with normal ventricular activation. In three patients abrupt acceleration of the atrial rate resulted in 1:1 A-V conduction with aberrant ventricular activation.

Figure 1 depicts the refractory period determination during sinus rhythm for patient 2. Figure 1C demonstrates that the effective refractory period of the His-Purkinje system begins at an H1-H2 interval of 440 msec. Figure 2A and B demonstrates that in this patient both a 1:1 and 2:1 A-V conduction response could be produced when the atrial rate was suddenly accelerated to a cycle length of 380 msec. The sinus cycle lengths preceding the onset of abrupt atrial stimulation vary by only 20 msec. The A-H interval of the sinus beats measures 70 msec. In figure 2A, the first atrial capture beat (A2) occurs at an A1-A2 interval of 470 msec with an A2-H2 interval of 110 msec. The corresponding H1-H2 interval is 510 msec and, since this value is greater than the determined ERP of the His-Purkinje system, conduction to the ventricles occurs. The decrease in ventricular cycle length from 870 to 510 msec results in a corresponding decrease in ERP of the His-Purkinje system to a value of less than 380 msec. This is reflected in the fact that all subsequent atrial impulses are conducted at H-H interval of 380 msec. In figure 2B, the first atrial capture beat occurs at an A1-A2 interval of 720 msec. The corresponding H-H interval measures 750 msec. A decrease in the ventricular cycle length from 890 to 750 msec does not significantly alter ERP of the His-Purkinje system from its determined value of 440 msec (fig. 1). This is reflected in the fact that the next atrial capture beat is blocked within the His-Purkinje system at an H-H interval of 400 msec. Thereafter, 2:1 A-V block is maintained at H-H intervals of 380 msec.

Patient 3 demonstrated three types of A-V conduction patterns at the same paced atrial cycle length of 380 msec. Using the extrastimulus method (as shown in fig. 1), the ERP of the His-Purkinje system at sinus cycle lengths, which ranged from 800 to 950 msec, occurred at an H1-H2 interval of 440 msec. Figure 3 (continuous trace) demonstrates that the first atrial capture beat results in an H1-H2 interval which is the same as the ERP of the His-Purkinje system (440 msec) and consequently block within this system occurs. Thereafter, 2:1 A-V block within the His-Purkinje system occurs at H-H intervals of 380 msec.

Figure 4A illustrates that, at the same paced cycle length of 380 msec, 1:1 A-V conduction resulted by the same mechanism as in figure 2A. The first atrial capture beat results in a shortening of the ventricular cycle length from 800 to 590 msec. A significant decrease in ERP of the His-Purkinje system follows so that 1:1 A-V conduction was maintained at H-H intervals of 435 msec to 380 msec.

Figure 4B demonstrates 1:1 A-V conduction with aberration at a paced cycle length of 380 msec. The first atrial capture beat shortens the ventricular cycle length from 880 to 760 msec which in turn shortens the refractory period of the His-Purkinje system. However, the decrease in the refractory period is less than that which occurs in figure 4A. Consequently, the second atrial capture beat which results in an H-H interval of 440 msec falls within the relative refractory period of the His-Purkinje system and 1:1 conduction with LBBB aberration results.

The functional nature of this type of His-Purkinje block is further exemplified by the findings shown in figures 5 and 6. Patient 4 (fig. 5) demonstrated either 1:1 or 2:1 A-V conduction when the atria
Figure 1
Refractory period determination in patient 2 during sinus rhythm. The sinus cycle lengths (CL) vary between 810 and 870 msec. In each panel, the tracings from top to bottom are electrocardiographic leads I, II, III, a high right atrial bipolar electrogram (HRA), and His bundle electrogram (HBE). T denotes time lines at 10 and 100 msec. A premature atrial beat (A2) is introduced after every eighth sinus beat (A1). (A–C) The A1-A2 coupling interval is progressively shortened from 600 to 420 msec. (C) The A2 depolarizes the bundle of His (H2) but fails to activate the ventricles. The resultant H1-H2 of 440 msec defines the effective refractory period of the His-Purkinje system. Similar abbreviations will be used for other figures.

were suddenly accelerated at a cycle length of 430 msec. The initial portion of figure 5A shows a period of 2:1 A-V block within the His-Purkinje system. The third ventricular complex is a spontaneous premature ventricular beat followed by 1:1 A-V conduction. The premature ventricular beat retrogradely depolarizes the bundle of His as evidenced by the fact that the fifth atrial beat is not.
followed by a His deflection. This results in a shortening of the cycle length of the His-Purkinje system and consequently a decrease in its effective refractory period so that 1:1 A-V conduction follows at the same H-H intervals that previously produced 2:1 A-V block. In figure 5B a spontaneous ventricular beat occurring at a slightly more premature coupling interval is retrogradely concealed within the His-Purkinje system. Note that the fifth atrial impulse is followed by a His deflection. Since the cycle length of the His-Purkinje system was not altered by the premature ventricular beat refractoriness of this system remains the same and 2:1 A-V conduction persist.

Figure 6 demonstrates that during 1:1 A-V conduction at a paced atrial cycle length of 400 msec failure to capture the atrium for one beat (third stimulus) causes a sudden increase in the ventricular cycle length which in turn produces an increase in effective refractory period of the His-Purkinje system. Following this latter change 2:1 block within the His-Purkinje system occurs.

Discussion

In the present study, as contrasted with most previous studies using His bundle recordings, atrial acceleration was initiated abruptly rather than incrementally. In addition, the resultant conduction patterns (2:1 A-V block within the His-Purkinje system, 1:1 A-V conduction with normal ventricular activation, and 1:1 A-V conduction with aberrant ventricular activation) were correlated with refractory period studies using the extrastimulus method.

The occurrence of any one of the three A-V conduction patterns was determined primarily by (1) an effective refractory period in the His-Purkinje system which exceeded that of the A-V node, (2) the coupling interval of the first atrial capture beat and its effect or lack thereof on the refractory period of the His-Purkinje system as it relates to changes in ventricular cycle length, and (3) the relative speed of A-V nodal conduction time.

The fact that both the effective and relative refractory periods of the His-Purkinje system can be altered by changes in ventricular cycle length explains why at the same paced rate, and with identical H-H intervals, 2:1 A-V block or 1:1 A-V conduction (with or without aberration) can result. The effective and relative refractory periods of the

**Figure 2**

Results of abrupt atrial pacing at a cycle length (CL) of 380 msec in patient 2. (A) Sudden acceleration of the atrial rate results in 1:1 A-V conduction, while in B 2:1 block within the His-Purkinje system occurs.
His-Purkinje system are greater at longer ventricular cycle lengths and decrease when the ventricular cycle length is shortened.5,9,10 This same relationship between ventricular cycle length and refractoriness also explains the perpetuation of either the 2:1 or 1:1 A-V conduction patterns throughout the pacing procedure.

During 2:1 A-V block, the nonconducted atrial impulses encounter an area of maximum refractoriness within the His-Purkinje system. The exact area of maximum refractoriness could not be determined in this study but may be within the common bundle itself, the bundle branches, or peripheral Purkinje network. Since the area of maximum refractoriness and the conducting tissues distal to it are not depolarized, they effectively maintain a long cycle length which is equivalent to that of the conducted atrial impulses. The longer ventricular cycle length is associated with a longer effective refractory period and, consequently, 2:1 A-V block is maintained. However, if during 2:1 A-V block, the distal area of maximum refractoriness is prematurely depolarized, as can occur retrogradely by a premature ventricular beat (fig. 5), its refractory period is shortened. Thereafter, the shorter refractory period permits 1:1 A-V conduction at the same H-H interval which previously caused 2:1 A-V block. This same relationship between cycle length and refractoriness can be invoked to explain 1:1 A-V conduction when the cycle length is sufficiently shortened by the first atrial capture beat (figs. 2A and 4A).

The relative speed of A-V nodal conduction time during atrial pacing also contributed to the development of 2:1 A-V block within the His-Purkinje system. The A-H intervals during abrupt acceleration of the atrial rate were not markedly prolonged relative to the A-H interval during sinus rhythm. This lack of significant A-V nodal conduction delay permitted the paced atrial impulses to be delivered to the His-Purkinje system within its effective refractory period. Had A-V nodal conduction time been more prolonged, the H-H intervals

---

Figure 3

Continuous record of patient 3 in whom 2:1 block within the His-Purkinje system resulted when the atrial rate was suddenly accelerated to a CL of 380 msec. The first atrial capture results in an H-H interval of 440 msec which is within the effective refractory period of the His-Purkinje system.
would have been greater than the ERP of the His-Purkinje system and 1:1 conduction would have always resulted. Alternatively, a greater delay in A-V nodal conduction time may have only resulted in A-V nodal Wenckebach type of conduction or 2:1 A-V nodal block during abrupt atrial pacing.

The occurrence of a functional 2:1 A-V block within the His-Purkinje system during abrupt atrial pacing simulates a type II second-degree A-V block. As seen clinically, patients with type II block are generally in sinus rhythm. However, patients who have type II second-degree A-V block during sinus rhythm almost always show 2:1 A-V block within the His-Purkinje system when the atrial rate is incrementally increased. In both groups of patients, the His-Purkinje system has the longest effective refractory period of the A-V conducting system. Despite these electrophysiologic similarities, the two groups can and should be distinguished. The more frequent clinical use of His bundle recordings and atrial pacing as a stress test makes this distinction of more than academic interest.

Patients who have a spontaneous or true type II second-degree A-V block generally have symptoms which can be attributed to the A-V block and invariably develop complete heart block for which permanent ventricular pacemaker therapy is indicated. On the other hand, the eight patients who had functional block with the His-Purkinje system did not have symptoms which could be related to A-V block and never demonstrated A-V block during sinus rhythm prior to or following this study. A majority of patients with type II A-V block have an associated right or left bundle-branch block pattern while most patients in this study did not. Also, the H-V interval during sinus rhythm is almost invariably prolonged (>55 msec) in the former group, while it was found to be within normal limits in the latter group.

A functional 2:1 block within the His-Purkinje system can also occur during abrupt spontaneous increases in atrial rate. This has been documented to occur in a case of paroxysmal A-V nodal reentrant tachycardia in an otherwise healthy...
Patient 4. (A) A 2:1 block within the His-Purkinje system is converted to 1:1 conduction following a spontaneous premature ventricular beat (PVB) which retrogradely depolarizes the bundle of His and shortens the His-Purkinje cycle length to <390 msec. The arrow indicates the point at which the antegrade His deflection would have appeared had it not been retrogradely depolarized by the PVB. (B) A PVB occurs slightly earlier in the cardiac cycle and fails to retrogradely depolarize the bundle of His. The cycle length of the His-Purkinje system was effectively not changed and 2:1 A-V block continues.

Functional nature of 2:1 His-Purkinje block. Atrial pacing at a constant cycle length of 400 msec resulted in continuous 1:1 A-V conduction as shown by the first two beats. The third stimulus fails to capture the atria, and the ventricular cycle length is suddenly increased from 400 to 755 msec. The associated increase in the refractory period of the His-Purkinje system results in 2:1 His-Purkinje block during continuous pacing.

Circulation, Volume XLVII, March 1973
Finally, figure 6 demonstrates that when 1:1 A-V conduction suddenly reverts to 2:1 His-Purkinje block an erroneous diagnosis of a true type II block can be avoided by carefully monitoring those events which immediately precede the period of block.

References
Functional 2:1 A-V Block within the His-Purkinje System: Simulation of Type II Second-Degree A-V Block

ANTHONY N. DAMATO, P. JACOB VARGHESE, ANTHONY R. CARACTA, MASOOD AKHTAR and SUN H. LAU

_Circulation_. 1973;47:534-542
doi: 10.1161/01.CIR.47.3.534

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1973 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/47/3/534