Noninvasive Recording of His-Purkinje Activity in Patients with Complete Atrioventricular Block
Clinical Application of an “Automated Discrimination Circuit”

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SUMMARY  In seven patients with complete atrioventricular (AV) block, His bundle electrograms (HBEs), standard ECG recordings, bipolar esophageal ECGs and highly amplified, filtered, bipolar chest lead ECGs were simultaneously recorded. The filtered chest lead ECG was averaged to determine His-Purkinje activity (HPA). A simplified device, the “automated discrimination circuit,” was used to selectively eliminate the superimposed atrial and ventricular potentials that are characteristic of complete AV block and unsuitable for signal averaging. The processed chest lead ECG was amenable to conventional techniques of signal averaging. In four patients with block proximal to the AV node diagnosed by HBE, there was no activity after the P wave in the surface-averaged ECGs. HPA was consistently recorded before the QRS in the surface-averaged ECG. The measurements of the HV and HPA-V intervals were very close, with a difference of <2 msec. Three patients with block distal to the His bundle by HBE showed a loss of electrical potential before the QRS in the surface-averaged ECG, but had a consistent HPA after the P waves. The P-HPA intervals coincided well with PH intervals, with a maximal difference of 5 msec.

DIAGNOSIS AND EVALUATION of disorders in atrioventricular (AV) conduction are the major purposes of His bundle electrograms (HBEs). The recording of His bundle potentials, however, usually requires cardiac catheterization. Working independently and using similar but not identical techniques of signal averaging, Berbari et al.1 and Flowers et al.2 showed that His-Purkinje activity (HPA) could be recorded from the body surface in dogs. Recently several reports have been published on initial application of this technique to man.3-5 Berbari and co-workers extended their studies to include extracardiac recordings of HPA during induced conduction disorders and junctional rhythm in open-chest dogs.6 In complete AV block, in which atrial (P wave) and ventricular (QRS complex and T wave) potentials superimpose, His bundle activity is buried in the large amplitudes of atrial or ventricular potentials and cannot be extracted with the conventional techniques of signal averaging. The greatest problem in such superimposition was resolved by the appropriate compilation of ECGs by use of a simplified device, the “automated discrimination circuit.” The present study is an additional contribution in the search for an effective method for identifying and recording electrical activity from the surface that represents His-Purkinje excitation in patients with complete AV block.

Subjects and Methods

The subjects of this study included seven patients, four men and three women, with complete AV block. Their clinical data are given in table 1. After the patient's consent was obtained, cardiac catheterization was performed and HBEs were recorded by a modified technique of Scherlag et al. The bipolar surface ECGs were taken by applying a cathode to the midsternal line at the fourth intercostal space and an anode to the posterior midline at the same level. The signals were amplified by a direct-current amplifier (Nihon Koden Co, RB-5) and filtered through a variable band pass. The band-pass filter was designed so that a high-frequency cut-off was variable between 220–300 Hz (24 db/octave) and a low-frequency cut-off between 50–80 Hz (24 db/octave). The band width between 50–300 Hz was usually used with satisfactory results.

The bipolar esophageal lead ECG was recorded with bipolar electrodes 2 cm apart to obtain larger P waves, together with a standard lead (usually lead II) or an unipolar limb lead. The esophageal ECG was recorded with a time constant of 0.03 second to ensure a stable baseline and sharply defined higher amplitude of P waves. These three sets of ECGs were simultaneously recorded on a data recorder (Nihon Koden Co, RMB 5104).

The crucial aspect of this study was selective elimination of atrial (P wave) and ventricular (QRS complex and T wave) potentials from the surface ECG to avoid the superimposition that is characteristic of
high-degree AV block. The filtered surface electrocardiographic signals were fed into the elimination circuit of the automated discrimination circuit. This circuit was designed to nullify the filtered surface ECG for a pre-set interval when triggered by an esophageal P wave or the R wave of a standard lead. The elimination intervals could be set in 10-msec increments from 10-1000 msec. Before deciding on which intervals are adequate to avoid atrial and ventricular superimpositions, it is essential to measure accurately the P and QRS intervals in the high-speed, magnified ECG of standard leads. Figure 1 illustrates the actual procedure of nullifying ventricular potentials from the surface ECG. The surface signals were abolished for QT intervals plus the preceding 100 msec to assure perfect elimination of the initial portion of the R-wave contents when triggered by the ascending limb of the R waves of the standard ECG. The postatrial segment superimposed by ventricular potentials is abolished (the segment after the P indicated by the arrow in figure 1A), while the segment without ventricular superimposition remains (the segment after the P indicated by the arrow in figure 1B). Figure 2 illustrates the actual elimination of P waves from the surface ECG. The initiation of P waves in the esophageal ECG is delayed in comparison with those in the surface ECG because the former depicts left atrial activity and, in addition, because a short time constant shortens the duration of the P waves. Therefore, it is important to start elimination at the beginning of the P waves in the surface ECG preceding the triggering point in the esophageal ECG. P waves and the ensuing 400-msec intervals were eliminated from the surface ECG. The pre-ventricular segment superimposed by the P wave is abolished (the segment before the R indicated by the arrow in figure 2A), while the segment without a P-wave superimposition remains (the segment before the R indicated by the arrow in figure 2B).

After these elimination procedures, two sets of surface ECGs are available for averaging — one with P waves and ensuing intervals, the other with QRS complexes and preceding intervals. The former was passed through a signal averager (Nihon Koden Co, ATAC 350) that was triggered by P waves amplified from the esophageal ECG. It was usually possible to find a position where the amplitude of the P wave clearly exceeded that of the R wave. Even in a few patients in whom the R wave was always taller than the P wave in the esophageal ECG, the averager could be triggered by a smaller P wave by use of a window comparator, as long as there was a distinctive difference in the amplitude of P and R waves. The latter was directed to the averager triggered by the R wave of a standard lead when the recorded signals were replayed in reverse. Since level detection was used as a means of triggering, it was crucial to ascertain the fiducial point where the trigger is activated. The fiducial point was

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**Table 1. Clinical Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex</th>
<th>Clinical diagnosis</th>
<th>Atrial rate (beats/min)</th>
<th>Ventricular rate (beats/min)</th>
<th>QRS duration (sec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>75/M</td>
<td>SHD</td>
<td>68</td>
<td>27</td>
<td>0.06</td>
</tr>
<tr>
<td>2</td>
<td>70/M</td>
<td>SHD</td>
<td>74</td>
<td>36</td>
<td>0.16</td>
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<tr>
<td>3</td>
<td>67/M</td>
<td>SHD</td>
<td>90</td>
<td>35</td>
<td>0.10</td>
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<tr>
<td>4</td>
<td>77/F</td>
<td>SHD</td>
<td>65</td>
<td>34</td>
<td>0.06</td>
</tr>
<tr>
<td>5</td>
<td>30/F</td>
<td>CHB</td>
<td>79</td>
<td>41</td>
<td>0.08</td>
</tr>
<tr>
<td>6</td>
<td>80/M</td>
<td>SHD</td>
<td>68</td>
<td>39</td>
<td>0.14</td>
</tr>
<tr>
<td>7</td>
<td>78/F</td>
<td>SHD</td>
<td>66</td>
<td>40</td>
<td>0.17</td>
</tr>
</tbody>
</table>

Abbreviations: CHB = congenital heart block; SHD = sclerotic heart disease.

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**Figure 1.** Simultaneous recording of bipolar chest lead (BCL), bipolar esophageal lead (BEL) and standard lead II ECGs. The BCL signals were eliminated for the QT interval plus the preceding 100 msec to assure perfect elimination of the initial portion of the R-wave contents. The postatrial segment superimposed by ventricular potentials is eliminated (the segment following the P indicated by the arrow in panel A), while the segment without ventricular superimposition remains (the segment following the P indicated by the arrow in panel B).
examined in 100 consecutive cardiac cycles on magnified high-speed (1000 mm/sec) strip recording. The differences of timing all fell within 2 msec. The total amplification was about $1 \times 10^6$ times. A block diagram of the system is shown in figure 3. The automated discrimination circuit is shown in figure 4 in detail. The timings of HPA or electrical potentials thus extracted in relation to the P wave or the QRS complex were compared with those of His bundle potentials recorded by intracardiac catheterization. It is prudent to exclude the possibility of cross talk between the amplified recordings from the internally detected HBE when the surface ECG was simultaneously recorded. Therefore, the surface ECG alone was recorded immediately after the intracardiac electrode for the HBE was removed. The elimination and averaging procedures were repeated twice and identical signals were reproduced.

**Results**

The actual procedures in extracting His bundle potentials in complete AV block are best shown in two representative patients, one with block proximal to the AV node and the other with block distal to the His bundle.

**Figure 2.** By complete elimination of P waves in bipolar chest lead (BCL) ECG, the preventricular segment superimposed by the P wave is eliminated (the segment before the R indicated by the arrow in panel A), while the segment without the P wave superimposition remains (the segment before the R indicated by the arrow in panel B). BEL = bipolar esophageal lead ECG.

**Figure 3.** Block diagram of the system for recording and processing signals. Bipolar chest lead (BCL), bipolar esophageal lead (BEL) and standard ECGs were simultaneously recorded. The automated discrimination circuit and Schmitt trigger circuit were activated by the P waves of BEL and R waves of the standard ECG lead. Diff Amp = differential amplifier.

**Figure 4.** Block diagram of the “automated discrimination circuit.” The standard ECG and bipolar esophageal lead (BEL) ECG were fed into the window comparator, which produces square-wave electrical impulse. The pulse triggers the discrimination circuit, which blocks the filtered bipolar chest lead (BCL) ECG for a desirable, pre-set interval (block time). In case R waves are always and clearly higher than P waves in the BEL, two thresholds, upper (U) and lower (L), are necessary to form an electrical impulse synchronous with the P wave.
Case 1

The patient was a 75-year-old man with repeated episodes of dyspnea and an oppressive sensation from the epigastrium to chest on mild exertion for 2 months before admission. He was found to have complete AV block with a slow ventricular rate. The electrophysiologic studies included HBE and surface-averaged ECG (SAE). His symptoms disappeared with temporary transvenous cardiac pacing. Figure 5A shows the standard 12-lead ECG and rhythm strip of lead II. The atrial rate was regular at 68 beats/min, while the ventricular complex of normal configuration and width (0.06 sec) occurred regularly at 27 beats/min. Figure 5B shows the HBE with a simultaneous lead II ECG. A His deflection consistently preceded the QRS complexes at an interval of 36 msec. The atrial waves, independent of ventricular deflections, were not followed by a His deflection. The site of block was determined to be proximal to the AV node. The SAE and a lead II ECG for the reference of timing are shown in figure 6. No electrical potential was found in the segment after the P wave in the surface ECG after averaging 200 cycles (fig. 6A). Meanwhile, reproducible HPA emerged in the segment preceding the QRS complex when averaged for 200 cycles by triggering it from the QRS (fig. 6B). The HPA-V interval was 38 msec, which was very close to the HV interval of 36 msec on the HBE.

Case 2

A 70-year-old man with a documented history of repeated syncopal episodes was referred to our laboratory for electrophysiologic studies. The standard 12-lead ECG and a continuous strip of lead V1 showed complete AV block. The atrial complex occurred regularly at a rate of 74 beats/min, while the ventricular rate was 36 beats/min. The ventricular complex was abnormally wide, with a duration of 0.16 second, and morphologically showed a complete right bundle branch block pattern (fig. 7A). The intracardiac His bundle recording revealed that each of the atrial complexes was followed by a His deflection. The PH and AH intervals measured 172 msec and 91 msec, respectively. The site of the block was presumably distal to the His bundle (fig. 7B). Figure 8 shows the SAEs, with the lead II ECGs as references. When the band-passed surface ECG, from which ventricular potentials had been eliminated, was averaged 800 cycles using P waves of the esophageal ECG as a trigger, a reproducible HPA was shown after the P wave (fig. 8A). On the other hand, no HPA was found when the surface ECG was averaged for 800 cycles by starting the trigger from the QRS complex of the recorded surface ECG played back in reverse (fig. 8B). This indicated that there was no fiducial electrical

Figure 5. The standard 12-lead ECG and lead II rhythm strip showed a complete atrioventricular (AV) block, with a ventricular rate of 27 beats/min. A) The QRS complex had a normal configuration and duration. B) The intracardiac His bundle electrogram (HBE) with a simultaneously recorded lead II ECG showed a block proximal to the AV node. The HV interval was 36 msec.

Figure 6. The surface-averaged ECG together with lead II ECG for reference of timing. No electrical potential was found in the segment after the P waves after averaging 200 cycles (panel A). The reproducible His-Purkinje activity (HPA) emerged in the segment preceding the QRS complex after averaging 200 cycles (panel B). $V_{sa}$ = atrial potential, surface averaged; $V_{sa}$ = ventricular potential, surface averaged.
The standard ECG and V1 rhythm strip showed complete atrioventricular block, with a ventricular rate of 36 beats/min. The ventricular complex was abnormally wide (0.16 second) and morphologically showed a complete right bundle branch block pattern (panel A). The His bundle electrogram (HBE) with simultaneously recorded lead II ECG showed a block distal to the His bundle. The AH interval was 91 msec and the PH interval (from the beginning of P wave to the His potential) was 172 msec (panel B).

potential before the QRS complex. In this case, it was necessary to average the signals for 800 cycles to improve the signal-to-noise ratio. The P-HPA interval was calculated by adding the P duration measured separately in a high-speed magnified ECG and the interval between termination of the P wave and HPA. The P-HPA interval thus calculated was 176 msec and was very close to the PH interval of 172 msec on the HBE. The comparative data of the HBE and the SAE in all patients are presented in table 2. Of seven patients, four had block proximal to the AV node or proximal to the HPA and three had block distal to the His bundle or distal to the HPA. The HV and HPA-V intervals were almost identical, with a maximal difference of 2 msec. The differences between the PH and P-HPA intervals were slightly larger, but still within 5 msec.

TABLE 2. Comparative Data on Diagnoses and Measurements by His Bundle Electrogram and Surface-averaged Electrocardiogram

<table>
<thead>
<tr>
<th>Patient</th>
<th>Site of block by HBE</th>
<th>PH interval (msec)</th>
<th>HV interval (msec)</th>
<th>Site of block by SAE</th>
<th>P-HPA interval (msec)</th>
<th>HPA-V interval (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Proximal to the AV node</td>
<td>—</td>
<td>36</td>
<td>Proximal to the HPA</td>
<td>—</td>
<td>38</td>
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<td>2</td>
<td>Distal to the His bundle</td>
<td>172</td>
<td>—</td>
<td>Distal to the HPA</td>
<td>176</td>
<td>—</td>
</tr>
<tr>
<td>3</td>
<td>Distal to the His bundle</td>
<td>165</td>
<td>—</td>
<td>Distal to the HPA</td>
<td>162</td>
<td>—</td>
</tr>
<tr>
<td>4</td>
<td>Proximal to the AV node</td>
<td>—</td>
<td>40</td>
<td>Proximal to the HPA</td>
<td>—</td>
<td>40</td>
</tr>
<tr>
<td>5</td>
<td>Proximal to the AV node</td>
<td>—</td>
<td>35</td>
<td>Proximal to the HPA</td>
<td>—</td>
<td>33</td>
</tr>
<tr>
<td>6</td>
<td>Proximal to the AV node</td>
<td>—</td>
<td>45</td>
<td>Proximal to the HPA</td>
<td>—</td>
<td>44</td>
</tr>
<tr>
<td>7</td>
<td>Distal to the His bundle</td>
<td>155</td>
<td>—</td>
<td>Distal to the HPA</td>
<td>150</td>
<td>—</td>
</tr>
</tbody>
</table>

Abbreviations: HBE = His bundle electrogram; SAE = surface-averaged electrocardiogram; HPA = His-Purkinje activity; AV = atrioventricular.
Discussion

Methodological Considerations

We previously evaluated seven kinds of lead axes, including bipolar and unipolar chest leads. The bipolar chest lead used in this study had the highest detection rate of HPA.

Assessment of the bandwidth was done to obtain the most satisfactory results. A bandwidth between 50–300 Hz (24 db/octave) gave the most suitable delineation for timing of extracted potentials in this study. Berbari and co-workers also evaluated the bandwidth and stated that the His-Purkinje waveform recorded in all previously published studies must have been attenuated and distorted by the 70–80-Hz low-frequency cut-off.8

When a prolonged electrocardiographic strip is recorded in a patient with complete AV block, some of the atrial and ventricular potentials are superimposed, but others are independent and completely free from superimposition. We first averaged the postatrial or preventricular segment, disregarding these superimpositions. However, HPA could not be isolated because of sizable contamination by superimposing atrial or ventricular potentials. By critically analyzing the rhythm strip of complete AV block, we found that only atrial potentials and postatrial segments not superimposed by the ventricular potentials remain if the QT interval is electrically nullified. Atrial potentials and postatrial segments superimposed by the ventricular potentials are included in the QT interval and eliminated together with ventricular potentials. Similarly, by eliminating atrial potentials, pre-ventricular segments not contaminated by atrial potentials remain.

The discrimination circuit is triggered either by the P wave of the esophageal ECG or by the R wave of the standard ECG. The elimination intervals should be specifically determined for each patient because the durations of the P wave and the QT interval vary from person to person. Once the elimination interval is decided in any patient and set on the automated discrimination circuit, it is unnecessary to change it during the entire elimination process.

The esophageal ECG used in this study must ensure a stable baseline for precise triggering of the averager. A time constant of 0.03 second was usually sufficient, but 0.01 second was sometimes required for a stable baseline. The initiation of the P wave was delayed because the esophageal lead recorded the left atrial component of the P wave. A short time constant also made a narrower P-wave width. However, the bipolar esophageal lead gave a more stable, consistent ECG and a higher amplitude of P wave than that obtainable by a unipolar esophageal lead; both are needed to trigger the averager. It was essential, therefore, to measure the P-wave duration and the QT interval precisely in high-speed, magnified ECGs before setting the elimination intervals on the automated discrimination circuit.

Another possibility of removing atrial and ventricular superimposition would be the use of a pattern recognition method, because superimposed waves are morphologically different from nonsuperimposed ones.

Clinical Application

Complete AV block may result from lesions in any of these three sites: proximal to the AV node, within the His bundle, and distal to the His bundle.8

In block proximal to the AV node, His bundle recordings usually show that each ventricular complex is preceded by a His deflection (fig. 5B; table 2). In patients with block proximal to the AV node in the present study, no electrical activity was recorded after the P wave by the averaging method. When a surface potential preceding the ventricular complex was averaged, a reproducible signal was obtained (fig. 6; table 2). The HPA-V intervals thus measured were almost identical to the HV intervals in intracardiac recordings. However, it was impossible to measure the AH interval noninvasively, because the A wave was buried in the averaged P waves. The block proximal to the AV node by HBE was equivalent to the block proximal to the HPA by SAE.

Three patients with block distal to the His bundle were available for comparison (table 2). The site of block by SAE was verified as a block distal to the HPA. There were small differences between the P-HPA and PH intervals because the initiation of the P wave was difficult to measure precisely. The largest difference was 5 msec and was considered acceptable for clinical purposes. We conclude that the diagnosis of the block site and the measurement of the P-HPA or HPA-V intervals in complete AV block by a noninvasive and simplified method are clinically applicable.

References

Noninvasive recording of His-Purkinje activity in patients with complete atrioventricular block. Clinical application of an "automated discrimination circuit".
H Takeda, K Kitamura, T Takanashi, T Tokuoka, H Hamamoto, T Katoh, I Niki and Y Hishimoto

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