The His-Purkinje Electrocardiogram in Man
An Initial Assessment of its Uses and Limitations

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SUMMARY A methodology is described for noninvasive recording of the electrical activity generated by the His-Purkinje system of man utilizing filtering, high amplification, and signal averaging. A waveform ranging between 1 and 10 μV was observed during the P-R segment. In many individuals, there was temporal overlap between the terminal P wave and the initial portion of the His-Purkinje waveform. In ten patients with long P-R intervals there was a strong correlation (r > 0.95) between the H-V time measured by electrode catheter and the duration of the His-Purkinje waveform. In two patients with atrial fibrillation the resultant His-Purkinje waveform was similar in morphology and duration to those observed in the ten patients. In each group H-V time was determined noninvasively and a waveform associated with electrical activation of the major portions of the His-Purkinje system was obtained.

RECENT REPORTS have described the use of signal averaging to obtain recordings from the His-Purkinje system on the body surface and the epicardial surface of dogs. Several experimental means, including atrial pacing, administration of procainamide and ischemic and traumatic injury to the His-Purkinje system were used to substantiate the conclusion that these potentials represented activation of the His bundle and bundle branches.

Several reports have described initial applications of this technique to man. This study was done to further outline the methodology for human application and to point out some of the potential uses and present limitations of the signal averaging technique which have become apparent during the early applications.

Methods

Patients were asked to lie on a comfortable bed in a small acoustically quiet room (originally designed for phonocardiographic studies). This room is conducive to a restful state and its metal walls are grounded to insure an electrically shielded environment. All sources of 60 Hz power to the room were eliminated.

A bipolar lead was attached across the patient's chest. Various lead axes were studied, including an anterior (+ pole) to posterior (− pole) lead at the nipple level; a diagonal lead between the apex (+ pole) and right pectoral area (− pole); and a lateral to posterior lead between the ECG positions of V4 (+ pole) and VR (− pole). Most data were obtained with the last configuration. The bipolar lead was amplified with a battery powered low noise (< 1 μV volt) differential preamplifier (gain = 1000) placed next to the patient's bed. The output of this amplifier was directed through a small hole in the wall to an adjacent room housing the rest of the equipment. After this initial stage of amplification the signal was filtered with a variable bandpass (Krohn-Hite, Model 3750). The low frequency cutoff was varied between 2 and 80 Hz (24 dB/octave) and the high frequency cutoff was set at 300 Hz (24 dB/octave). Usually the bandwidth was 10–300 Hz or 20–300 Hz. The signal was also amplified by a factor of ten by these filters. The final stage of analog processing was a variable gain, high voltage amplifier (Analog Devices, Model 171) adjustable between gains of 1 to 500. The total gain of the system was on the order of 5 × 106.

This conditioned signal was then converted to a digital format (Hewlett Packard Analog to Digital Converter, Model 5610, 10 bits of resolution, sampling rate = 2.5 KHz per channel) for processing by a minicomputer (Hewlett Packard Model 2100A). The relatively high sampling rate for ECG signals enhances the visual resolution of the signal thus decreasing the digitized (discontinuous) appearance of the waveforms. A magnetic tape system stores the averaged waveforms for future processing and analysis. The signals were averaged for 50 to 200 cardiac cycles, most commonly 100 cycles. The square wave response of the system shows no ringing and the response to sine waves of frequencies of 15, 50, and 100 Hz shows slight diminution of amplitude and moderate phase shift in the passband of 10–300 Hz.

To determine the onset of ventricular activation three roughly orthogonal bipolar chest leads were examined at the recording speed (1000 mm/sec) of the digitized display, but with intermediate gains, i.e., between standard ECG recordings and the highly amplified ECG. The earliest departure of the QRS complex from baseline on any of the three leads was chosen as the time of earliest ventricular activation. All measurements were made with this time reference which is marked in the figures with an arrow and labeled QRS. The lead marked ECG in each figure represents the output of the preamplifier and is comparable in gain to standard ECG leads. It is not necessarily the lead that showed earliest ventricular activation. In only a few cases was there a discrepancy between the leads of greater than 5 msec in measuring the onset of ventricular activity, and in no case was this observed to be greater than 10 msec.

In order to select a precise time window of the cardiac cycle for averaging, the computer must be triggered at a precisely fixed instant within the cycle. This can be done by

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means of an atrial pacer spike or by conditioning the QRS complex to derive a brief pulse at a fixed time (fiducial point). In order to obviate atrial pacing, the second scheme was used. This necessitated the use of a QRS detector and a more complex computer program. A QRS detector similar in principle to one described by Brandon and Brody was used to derive the fiducial point and the computer program stored the signals so that information preceding the QRS could be averaged. To test the stability of this derived fiducial point with the cardiac cycle, a special monitor scheme was used. Since the QRS was detected sometime during the R wave, the remaining portion of the QRS could be observed on a triggered sweep oscilloscope (Hewlett Packard Visoscope). Thus the QRS detector would trigger both the computer (to average the selected time interval prior to the fiducial point) and monitor the oscilloscope (to record activity after the fiducial point). By displaying some feature of the QRS, usually the S wave, it was possible to observe the exact superimposition of this wave at fast sweep speeds (1000 mm/sec) on the monitor oscilloscope. Temporal displacements greater than ±2 msec were not tolerated. Such variation occurred in only two patients. Figure 1 shows a block diagram of the system and a representation of the ECG with various timing marks and viewing windows.

His bundle electrograms were obtained from ten patients. Each patient was referred to the Cardiovascular Laboratory for a conduction system evaluation prior to possible pacemaker implantation. Standard electrode catheter techniques, including validation by atrial and His bundle pacing, were utilized.

**Results**

Reproducibility of recorded signals with repeated averages was a prerequisite for acceptance of the cardiac origin of the signals. In this study, a complete record usually consisted of an ECG lead (see Methods), and at least three serially obtained recordings from the same highly amplified surface averaged lead. Figure 2 is an example of data obtained from a normal 20-year-old male volunteer. The onset of the QRS of the ECG is indicated on the right of the top trace. The “isoelectric” interval precedes the QRS complex. The three surface averaged leads (SAL) shown below the ECG were obtained serially in order to observe the reproducibility of the waveforms in the P-R segment. Reproducibility in this context does not necessarily mean exactness, but similarity. The proximity of the high level atrial activity (P) prevents unequivocal demarcation of the end of atrial activity and the onset of the His-Purkinje system activity. The magnitude of the waveforms in the P-R segment range from 2 to 10 μV.

In those subjects where the duration of atrial activity overlapped with His-Purkinje system activity, we attempted to selectively attenuate terminal atrial potentials by high pass filtering. The results of stepwise increases in the low frequency cutoff are shown in figure 3 which contains recordings from the normal subject of figure 2. Each trace was obtained with the same lateral-lateral lead axis and gain setting, but the low frequency cutoff was increased from 10 to 60 Hz in 10 Hz increments. Note the emergence of several prominent waveforms. Each trace was aligned with the onset of the QRS marked by the heavy vertical line. There is a slight phase shift to the left due to the interaction of high and low pass filters. However, atrial and His-Purkinje waveforms remain continuous.

To eliminate the ambiguity involved with selecting the onset of His-Purkinje activity, patients with long P-R intervals were studied. With sufficient A-V nodal delay, atrial ac-
**Figure 3.** Changes in the His-Purkinje system waveforms produced by variations of the low frequency cut-off. These data are from the normal subject shown in figure 2. Each trace is aligned with the QRS onset occurring at the solid vertical line on the right. The low frequency cut-off for panels A–F is 10, 20, 30, 40, 50, and 60 Hertz, respectively. There is a slight phase shift of the waveforms in the P-R segment produced by the filters. This phase shift at a midband frequency (125 Hz) ranges between 0.8 msec (A) and 2.0 msec (F). The voltage scale applies to all traces.

Activity ends prior to the onset of His-Purkinje activity even in the highly amplified lead. The recordings in figure 4 were obtained from a patient with a P-R interval greater than 0.40 seconds. A short rhythm strip is shown in panel A. Panel B was obtained in the same manner as that in figure 2. Note the large reproducible biphasic waveform preceding the QRS. The onset of the His-Purkinje system waveform is not obscured by overlapping atrial activity, but the onset of the His-Purkinje waveform is not definite on any single recording. Comparison of the three SAL recordings allows a determination of the point at which reproducibility of the His-Purkinje waveform begins. From this point the duration of the His-Purkinje waveform is about 55 msec as measured to the onset of the QRS. The magnitude of this waveform is about 5 microvolts.

In atrial fibrillation atrial potentials are not synchronous with ventricular activity. Thus atrial activity would act as random noise and average toward zero allowing the emergence of His-Purkinje activity. Figure 5 consists of recordings taken from a patient with atrial fibrillation (panel A). Panel B shows the ECG and three serially obtained SAL recordings. The reproducible portion of the P-R segment begins about 40 msec prior to the QRS.

With a bandwidth between 10 and 300 Hz the His-
Purkinje system waveform preceding the QRS was consistently seen in patients with long P-R segments or fine atrial fibrillation. Figure 6 shows five SAL recordings (A-E). Each trace in this figure was obtained after averaging 300 cardiac cycles. On the right, a solid vertical line used for alignment signifies the onset of the QRS of each of the respective ECGs of the subjects. The duration of the waveforms range between 39 and 72 msec. Trace B was obtained from the patient with atrial fibrillation described in figure 5 and trace C is from the patient described in figure 4. Panels A, D and E were obtained from other patients with first degree block. There is a gross morphological similarity of these waveforms. Each was obtained using the lateral-lateral lead axis and was filtered between 10–300 Hz.

In ten patients, His bundle electrograms were clinically indicated. These patients had the surface recording study performed either immediately before or after the catheter procedure. Table 1 lists the H-V times and the duration of the His-Purkinje waveform (HPS-V time). There is a strong correlation between these two indices of His-Purkinje conduction time (r > 0.95). The H-V measurements were arrived at independently from the HPS-V measurements. Each measurement was made by several observers and where disagreement occurred a mean value was derived.

Direct comparisons between the His-Purkinje waveforms and the His bundle electrograms of two subjects are shown in figures 7 and 8. Figure 7A shows two ECG leads (II and V1) and the His bundle electrogram from a subject with a

![Figure 6](http://circ.ahajournals.org/)

**Figure 6.** SAL recordings from five different patients. Each trace is aligned so that the onset of the QRS occurs at the solid vertical line on the right. Each recording is the result of averaging 300 cardiac cycles. The arrow points to a more clearly defined onset of the His-Purkinje system (HPS) waveform. The duration of the HPS waveform in traces A-E is 39, 43, 55, 60 and 72 msec, respectively. Panel B was obtained from a patient with atrial fibrillation, while the others are all from patients with first degree heart block. The voltage scale for each trace is shown opposite itself.

![Table 1](http://circ.ahajournals.org/)

**Table 1.** His Electrocardiogram Intervals and SAL Interval

<table>
<thead>
<tr>
<th>Patient</th>
<th>H-V (msec)</th>
<th>HPS-V (msec)</th>
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<tbody>
<tr>
<td>1</td>
<td>60</td>
<td>62</td>
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<td>10</td>
<td>55</td>
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r > 0.95

![Figure 7](http://circ.ahajournals.org/)

**Figure 7.** Patient with a normal H-V and HPS-V time. The recordings in panel A are from the standard His bundle catheterization procedure. The timelines are 1 sec apart and the H-V time is 40 msec. Panel B shows the ECG and three serial SAL recordings. The onset of the HPS waveform was chosen by selecting the point at which reproducibility begins. The duration of the His-Purkinje system waveform (HPS-V) is 42 msec.
normal H-V time (40 msec). Panel B shows the ECG and three surface averaged lead recordings. The duration of the His-Purkinje waveform is 42 msec. The data in figure 8 is from a patient with a prolonged H-V time of 60 msec (panel A). The surface averaged leads (panel B) from this patient also indicate a His-Purkinje waveform duration of 60 msec.

Discussion

Methodological Considerations

Two criteria must be met before a signal averaged recording can be considered valid. First, the averaging window, in this case the P-R segment, must maintain a constant time relationship with a predetermined fiducial point, in this case, an instant within the QRS complex. Absence of this temporal stability is referred to as "jitter" and results in a smoothing and eventual deterioration of the averaged waveform. The repeated superimposition of specific inflections of the ECG such as the nadir of the S wave on a high speed oscilloscope triggered by the QRS detector indicates a fixed time relationship between ventricular activity and the trigger, i.e., the absence of jitter.

A second criterion for validation of a signal averaged record is the degree of reproducibility of serially obtained averages. As alluded to in the results, reproducibility of the His-Purkinje waveform refers to the gross similarity of the waveforms versus a strict point by point exactness. The reason for these differences is due to the statistical nature of signal averaging and the variability of the interfering noise. This reproducibility of repeated averages indirectly assures that the interfering noise has been reduced to a significantly smaller amplitude than the true signal. In cases of random noise, it is well known that the signal-to-noise ratio increases by the square root of the number of cycles averaged. Random physiological and electronic noise are in this category. Careful attention to the reduction of 60 Hz interference was effective in limiting this type of noise to only a few microvolts. Signal averaging further reduces this type of noise.

The low level of the His-Purkinje system potentials at the body surface (1–10 μV) posed a formidable recording problem. Occasionally, satisfactory signal-to-noise ratios were not achieved even after signal averaging. Although the recording environment is conducive to a restful state, overly alert or anxious subjects produced skeletal muscle potentials and motion artifacts resulting in high levels of unresolved noise. Nevertheless, in only three of 40 subjects did we fail to record reproducible waveforms coincident with activation of the His-Purkinje system. In the study of Furness et al., it was noted that activity corresponding to the His-Purkinje system was recorded in only 30% of the patients studied. The authors point out that more stable QRS detection and greater reduction of AC interference would aid these recordings. These authors used only an amplitude scheme of QRS detection for triggering and their records were taped in the catheterization laboratory for subsequent signal averaging. Recording on magnetic tape provided another source of noise.

In normal subjects, overlap of atrial and His-Purkinje activity often prevented a clear demarcation between the two types of activity (fig. 2). In this case it was not possible to equate the onset of His bundle activation with the onset of reproducible waveforms preceding the QRS because the entire averaging window contained reproducible potentials. With the high gains employed, atrial activity extends much further into the P-R segment than indicated on the standard ECG. On the assumption that atrial activity and His-Purkinje activity might have significantly different frequency components, it was anticipated that the problem of temporal overlap could be overcome by high pass filtering. This expectation was not realized because overlap in frequency content prevented elimination of atrial activity without serious degradation of the His-Purkinje waveform.

The possibility remains that high pass filtering may sufficiently attenuate atrial potentials to allow recognition of the higher frequency His-Purkinje waveform within the continuum of activity. The records of figure 3 provide some promise of success. Higher frequency waveforms are recognizable beginning about 40 msec prior to the QRS, a time when His-Purkinje activation should begin in the normal subject. We cannot be certain that these waveforms do not represent higher frequency components of the terminal P wave which fortuitously coincide with the onset of His-Purkinje activation. Data from a large number of normal subjects will provide a firmer basis for deciding whether it is possible to distinguish His-Purkinje waveforms in a continuum of activity after appropriate high pass filtering. If, in many records, there is consistently a distinguishable higher frequency waveform beginning 40–50 msec prior to the QRS...
Despite differing P-R intervals among subjects, it will be reasonable to conclude that these higher frequency signals are generated by the His-Purkinje system.

The application of high pass filtering has limitations. Analysis of the His-Purkinje waveforms indicates that most of the frequency content is below 60 Hz. Thus severe degradation of the waveform occurs if the low frequency cutoff is much higher than 60 Hz. The His-Purkinje waveform recorded in all previously published studies must have been attenuated and distorted by the 70-80 Hz low frequency cutoff.

**Clinical Correlation**

To bypass the problem of atrial overlap, patients with long P-R segments and atrial fibrillation were studied. This allowed the registration of the unadulterated His-Purkinje waveform. Under these conditions, it may be possible to describe a “normal” His-Purkinje waveform. Of course one could not rule out His-Purkinje disease in patients with long P-R intervals. The present assessment of the His-Purkinje system rests upon normalcy of the QRS and of the H-V time. By these criteria traces A and B of figure 5 are normal; trace C is marginal, and traces D and E are abnormal. The recording of many more His-Purkinje waveforms will allow recognition of common characteristics and a statistical definition of confidence limits for certain parameters of the waveform.

Of the several leads studied, the lateral-lateral provided the most consistently reproducible averages. It is a reflection of a low signal-to-noise ratio, even after averaging, that often only one of several leads yielded reproducible His-Purkinje waveforms. Optimization of the lead axis involves maximizing His-Purkinje potentials and minimizing respiratory muscle and atrial potentials. The frequent failure to achieve high signal-to-noise ratios in all lead axes have so far hindered the spatial vector characterization of the His-Purkinje waveform.

His-Purkinje potentials were recorded throughout the interval from His bundle activation to ventricular activation. An isolated His bundle potential corresponding to the His bundle electrogram recorded with a catheter electrode would not be expected on a surface recording nor would the right bundle branch and left bundle branch potentials be recorded. To the contrary, because of the smaller size of the His bundle, its potentials may be more difficult to record. There was not a definite demarcation between His-Purkinje potentials and ventricular myocardial potentials. Consequently, the determination of the onset of ventricular myocardial activation at very high gains was somewhat arbitrary. The use of three conventionally amplified orthogonal leads to time the onset of the ventricular activation has been accepted as a reasonably accurate procedure in His bundle electrocardiography.14

The correlation of the H-V time with the duration of the His-Purkinje waveform is the most direct test of this technique (see table 1). While the correlation coefficient is quite high, many more studies must be done to further increase the confidence in this statistical measure. It should be noted that the values in table 1 are rounded in accordance with recording limitations of each technique. Both the catheter technique and signal averaging technique introduce certain measurement errors. Specifically, the choice of measurement points, e.g., the onsets of ventricular and His-Purkinje activity, are partially subjective. Once the measurement points are chosen, regardless of their correctness, resolution is limited with both techniques. Measurements from the standard His bundle electrogram recorded at paper speeds of 100 mm/sec will not accurately resolve less than 5 msec. The computer displays equivalent recording speed of 1000 mm/sec and resolution to 0.5 msec is possible. The values used for the H-V intervals were rounded to the nearest 5 msec, and the values for the HPS-V interval were rounded to the nearest 1 msec.

In conclusion we have shown that in human subjects a waveform representing activation of the His-Purkinje system can be recorded from the body surface with the signal averaging technique. In subjects with normal P-R intervals, contamination of the waveform with atrial potentials has so far been a problem which requires further attention but does not appear insoluble. The duration of the waveform correlates well with the H-V interval, but further studies are required to characterize normal and diagnostically abnormal configurations of the waveform. Long term studies are also needed to assess the predictive capabilities of the His-Purkinje system waveform.

**References**

7. Stopczyk MJ, Kopec J, Zochowski RJ, Pieniak M: Surface recording of electrical heart activity during the P-R segment in man by a computer averaging technique. (abstr) Proc World Congress of Cardiol #162, 1974
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