Surface Recording of His-Purkinje Activity on an Every-beat Basis Without Digital Averaging

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SUMMARY Efforts to record evidence of electrical activity from the body surface originating in the His bundle or bundle branches have been reported since 1973. Almost exclusively, these techniques have required digital averaging of 50–100 sequential cardiac cycles.

For immediate diagnostic, therapeutic and prognostic application, recording on an every-beat basis is highly desirable. This is especially important in instances of changing atrioventricular conduction, arrhythmias or less-than-constant RR intervals.

Our object has been to develop a system for more nearly optimal noise reduction, to avoid the disadvantages of serial signal averaging, and to be able to record His-Purkinje activity in man on an every-beat basis. Using multiple parallel inputs with linear amplification, additional logarithmic amplification, some bandpass filtering, and a logic circuit that ultimately examines and accepts or rejects a deflection as "true" signal, we can record, in most instances, on a beat-by-beat basis, this very valuable component of the cardiac electrical cycle.

IN 1973, Berbari et al. and our group, working independently with only slightly different approaches to digital averaging, showed that the electrical activity arising from the His-Purkinje system could be recorded from the body surface. Other reports have followed, describing further limited success in obtaining such recordings in the dog and in man.

The ability to record and to verify electrical activity in the PR segment as having arisen from the His-Purkinje system has been exciting and full of promise, yet the necessity of noise reduction and enhancement of the very low level signal with digital averaging has major shortcomings, and distinctly limits clinical usefulness of the approach. First, the averaging process excludes the ability to detect moment-to-moment changes in the true signal, and introduces errors in amplitude, duration and morphology. Averaging blunts deflections that are individually discrete and sharp. If the deflection of interest, for example, has 100-Hz components and the interval from the trigger signal (usually the upstroke of the R wave) varies ± 0.45 msec, the resultant amplitude of the digitally averaged signals will be roughly one-half of the amplitude of the original signal, even if we can assume no contamination with noise. More important, digital averaging systems cannot generate on-line information on a beat-by-beat basis, which is usually necessary for meaningful diagnostic and therapeutic applications.

The following report concerns a different approach in which our object has been to develop a system for optimal noise reduction that avoids the disadvantages of serial signal-averaging techniques, correlation techniques, and peak-level-detection techniques. Our goal has been to record His-Purkinje activity in man on an every-beat basis as a part of the surface ECG.

Methods

Our initial concern was to exclude as much as possible both electrical and magnetically generated noise. We first built a 64-cubic-foot, highly shielded room composed of a wood frame, an external layer of copper screen and a silicon-iron layer of high-saturation, middle-permeability (500–7000 μ) shielding.

Our approach to further noise reduction and low-level signal enhancement is diagrammed in figure 1. Multiple parallel input signals were recovered from a predetermined number of closely spaced, though isolated from each other, surface electrodes using the same predetermined number (n) of optical amplifiers. The multiple parallel inputs are connected to an equivalent number of amplifiers in an analog circuit (block 1) that are combined so that noise is increased by a factor of the √n, while the cardiac signal is increased by a factor of n. After initial linear amplification of 10,000 times or more, the signal may, if desired, be passed through a bandpass filter. The low end of the frequency band may be limited up to about 35 Hz if desired before amplification of another 200 times takes place (block 2). Using logarithmic amplifiers at this stage, the low-level signals, such as the His-Purkinje signals, are amplified to a much greater degree than are the already relatively large P, QRS and T, and may be added (block 3) for further specific elimination of 60-Hz noise (block 5). The output of the logarithmic amplifiers are then inputs to a digital logic circuit (block 4). This digital logic circuit, again, includes a number of amplifiers equivalent to the original number of inputs from the surface. The digital logic circuit examines the instantaneous polarity from each of the parallel input signals and passes this information to gates. When identical polarities are present from all of the closely spaced inputs, the signal will be regarded as the "true" signal, and a coincidence condition will be fulfilled. At instants in which the signal from all inputs does not have...
identical polarity, the signal is rejected as probable instrument (amplifier) noise.

When a "true" signal is identified through the logic circuit, this signal enables a circuit that adjusts the gain of a variable-gain amplifier, so that the portion of the signal in which His-Purkinje activity is expected is magnified (block 6). In this maneuver the variable-gain amplifier circuit provides moment-to-moment amplification of the "true," low-level input signal originally received from the surface.

Results

In figure 2A, a simultaneous surface lead and catheter recording of His-Purkinje activity from a dog are shown. In figure 2B, from the same dog, three surface electrocardiographic leads at conventional gain recorded simultaneously with a surface recording of His-Purkinje activity are shown. Note the correspondence of the onsets of His-Purkinje activity in internal and surface records and their relationship to the onset of ventricular activity. These records, although not obtained simultaneously, were obtained within a few minutes of each other, the time necessary to move the dog from an area where fluoroscopy is available into the shielded room for the surface His-Purkinje recording.

Figure 3 is a record from a dog given 2 mg of digoxin i.v. over an 80-minute period. The record above is a recording from parallel inputs of the surface His-Purkinje lead, while the bottom record is a transverse lead of an approximate lead I position inverted to avoid crossover. Compare the baseline values for the atrial to His-Purkinje activity, and the His-Purkinje deflection to the ventricular deflection with the subsequent records, as progressive digitalis toxicity was achieved. The atrioventricular (AV) nodal conduction delay (atrial to HP interval) becomes progressively greater until the development of AV dissociation with preterminal idioventricular rhythm. In panel 4, the His-Purkinje deflection preceding the narrow dissociated idioventricular QRS complex is absent.

Figure 4 is a record from a normal 26-year-old woman in which leads I, aVF, and V1 were recorded simultaneously with a His-Purkinje recording from the surface of the chest. The calibration signal of 10 μV only refers to the logarithmically amplified low-level signals such as those in the PR segment. The onsets of atrial, His-Purkinje and ventricular activity are indicated by the vertical lines. These signals were consistently repeated in cycle after cycle during the entire recording period.

Figure 5 is a record of leads I, aVF, and V1 recorded simultaneously with the surface His-Purkinje recording from a normal 27-year-old man. Note the repeatability of the deflection in the PR segment, which we believe represents His-Purkinje activity occurring 45 msec before onset of the QRS. In each instance, the calibration is a standard calibration for the extremity leads, but because of the nature of the logarithmic records, the 10 μV/cm calibration refers to the logarithmically amplified low-level signals only.

Discussion

Using needle electrode recordings from the isolated perfused heart of both the dog and the cat, Alanis and
co-workers described the physiologic significance of the common bundle of His. This was some 65 years after His and Kent described the histologic discovery of the bundle. Hoffman and co-workers described further successes in directly recording electrical activity during the PR segment from animal hearts, and much in the way of our physiologic understanding derives from their work. The descriptions of Scherlag renewed interest in electrophysiology, because His bundle activity could be recorded locally from an endocardial catheter site in intact dog and man. We considered that the next obvious step was to capitalize on the important work of predecessors and to attempt to transfer from the catheterization laboratory the capability of recording His-Purkinje activity noninvasively from the body surface.

In the present study we tried to focus on problems that were identified in recording His-Purkinje activity from the surface in the earlier reported work, from our laboratory and that of others. First, the problem of signal-to-noise ratio by itself has proved difficult. The level of the His-Purkinje signal as detected on the surface is 3-10 μV. In the usual circumstance of recording in hospital settings, these levels may be well below the noise level. Various categories of interference, or electromagnetic noise, obscure the effect of these low-level signals and make them very difficult to appreciate. Some interference is from 60-Hz sources, or harmonics of 60 Hz. Some noise is myotonic in origin from skeletal muscles in the patient. Much of the interference may come from the amplification system itself.

Digital averaging techniques, while extremely valuable as a means of enhancing signals for analytic purposes and for fundamental understanding, was not immediately helpful in situations of changing degrees of AV block or intermittent arrhythmias, or when premature complexes or any dynamic alterations in
AV conduction were present. These are, however, the exact clinical circumstances in which such information could be of the most value. For surface detection of the His-Purkinje signal to have an impact on diagnostic and therapeutic decision-making, the physician needs on-line records of the effects of structural or functional disease, or the effects of biochemical derangements on the conduction system.

Therefore, we used a combination of noise reduction techniques, including an electrically quiet environment, multiple, very closely spaced analog inputs, logarithmic enhancement of low-level signal (with consequent relative attenuation of interfering larger signals from the atrium and the ventricle), and a logic system that allows further differentiation of real signal from noise.

We attacked the problem of alternating line current interference, specifically 60-Hz interference and its harmonics at 120 and 240 Hz, with very precise notch filters that remove only the frequencies of concern but do not attenuate the other frequencies that might be of interest.

By using the newly developed, closely spaced electrodes and parallel analog inputs for a type of analog averaging, we minimized interference from respiratory movement and from atrial activity. Further minimization occurred with appropriate low-frequency constraints. Input into the variable gain amplifier circuitry offered the final means for emphasis of the signals of interest in the PR segment.

The present system has several limitations. First is the problem of the lack of portability of the system for

![Figure 4](http://circ.ahajournals.org/)

**Figure 4.** Simultaneous recording of the relatively orthogonal leads I, aVF, and V1 from a 26-year-old woman with the simultaneous acquisition of surface potential recordings from His-Purkinje lead system (HP). The vertical lines indicate the earliest evidence of surface atrial activity, His-Purkinje activity, and ventricular activity recorded in any lead. Our measurements of the intervals in msec are indicated in the lower left. The 10-μV calibration refers only to the surface His-Purkinje lead, and specifically refers to the low-level logarithmically amplified signals. The deflection in the PR segment is probably from the His-bundle proper, judging from its temporal relationship to the PR segment of the surface leads and the measurement of the interval between it and the onset of the QRS deflection.

![Figure 5](http://circ.ahajournals.org/)

**Figure 5.** Surface leads I, aVF, and V1 recorded simultaneously with the surface His-Purkinje record. The repeatable activity in the PR segment, which we believe represents His-Purkinje activity, is indicated (HP). The vertical lines indicate our points of measurement of the onset of atrial activity (P), His-Purkinje activity (HP) and ventricular activity (V). The surface leads are calibrated as indicated in the conventional fashion, while the calibration of His-Purkinje record refers to the low-level aspects of the logarithmic record only.
determining His-Purkinje activity in a setting outside the shielded room. This is an important limitation, for although many people may be easily moved into the shielded room in order to obtain recordings, portability is necessary to record from settings such as the coronary care unit, other critical-care areas or the emergency room. We have had only limited success in recording in these areas, with later confirmation of His-Purkinje activity in the electrically quiet setting.

Another problem has been identification of exactly which part of the His bundle or the bundle branch system produced the recorded deflection. We believe that the late preventricular, discrete activity may represent a coalescence of early apicoseptal Purkinje activity. Occasionally, 10-25 msec before obvious ventricular activation begins, we have recorded discrete deflections that may represent bundle branch activity. We believe, however, that most relatively early deflections (35-45 msec before ventricular activity) arise from the nonbranching portion of the His bundle. Such a working hypothesis of the origin of these various deflections arises in part as a result of considering the relative anatomic compactness of the penetrating portion of the bundle of His, the probable resulting electrical field, and the probable current densities. Further, the timing of subintervals of the PR segment and durations of deflections from the catheterization data of others would correspond to our own experience. Further clarifying data of bundle branch vs His bundle origin should be forthcoming from experimental animal protocols, and clinical opportunity of recording in specific and varied electrophysiologic circumstances. The latter opportunity will be enhanced as we solve the portability problem.

In conclusion, we believe this work is another step toward making His-Purkinje recording available as an expected part of the ECG.

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