Intraoperative Recording of the His Bundle Electrogram in Man

An Assessment of Its Precision

MACDONALD DICK, II, M.D., EHUD KRONGRAD, M.D., ROBERT E. ANTAR, M.D., SAM ROSS, B.E.E.,
FREDERICK O. BOWMAN, JR., M.D., JAMES R. MALM, M.D., AND BRIAN F. HOFFMAN, M.D.

SUMMARY To estimate the effect of distance between the electrode and the signal source on the amplitude of the His bundle electrogram (HBE) recorded during open heart surgery, a specially designed probe, containing six pairs of closely spaced (1 mm) electrodes was placed on the endocardial surface of the right atrium such that each electrode pair was parallel to the course of the His bundle. The amplitude of the HBE recorded through electrodes closest to the His bundle ranged from 0.76 to 3.44 mV, at 1 mm from 0.38 to 1.13 mV, at 2 mm from 0.27 to 0.86 mV, and at 3 mm from 0.2 to 0.44 mV. Maximal amplitude of HBE decreased by 57% at 1 mm, 73% at 2 mm, and 82% at 3 mm. The percent decrease was initially rapid, then declined more slowly at distances greater than 1 mm, resembling in form data obtained previously in animal studies by different techniques.

Since the maximum HBE was greater than 1.0 mV in nine of 11 patients, and equal to or greater than 1.0 mV in only two of 11 patients at 1 mm, and less than 1.0 mV in all patients 2.0 mm from the maximal HBE, the anatomic location of the His bundle can be estimated from HBE amplitude. Intracardiac electrograms, recorded through closely spaced bipolar electrodes during open heart surgery, afford clinically useful precision in locating the specialized conduction tissue of the heart.

THE INTRAOPERATIVE RECORDING of electrograms from the cardiac specialized atroventricular conduction system through closely spaced bipolar electrodes has become a useful adjunct to the cardiac surgeons in delineating the location and course of these structures. This method has been found to be particularly useful in repair of endocardial cushion defects, common atrium, transposition of the great arteries (Mustard procedure), and complex congenital heart lesions, including single ventricle and L-transposition of the great arteries. Although characteristic electrograms of the underlying specialized tissue are readily identified, no data are available concerning the relationship between the amplitude of the signal and the distance between the electrode and the signal source. The purpose of this study is to provide these data for the His bundle electrogram (HBE) recorded during open heart surgery in man. In addition, the study provides an estimate of the distance from the His bundle at which electrograms fail to be recorded in man using our technique, and thereby defines for the cardiac surgeon areas free of specialized conduction tissue.

Materials and Methods

Eleven patients ranging in age from 2 to 48 years, median age 11, were studied during open heart surgery for a variety of cardiac lesions (table 1). Core (R) temperature was 37°C in eight patients and 32°C in three. Serum potassium concentration just prior to surgery was greater than 4.0 mEq/L (4.0–5.1 mEq/L) in ten patients; and in another, 2.8 mEq/L. At the time of data collection all patients had normal atrioventricular conduction initiated by either normal sinus rhythm or atrial pacing.

In order to study the effect of distance on the recorded HBE amplitude, a specially designed electrode probe containing six pairs of parallel electrodes was used to record bipolar electrograms from the His bundle. The distance between the two electrodes in each pair, and the distance between adjacent pairs of electrodes, was 1 mm. After institution of cardiopulmonary bypass, the electrode probe was placed on the right atrial endocardial surface between the coronary sinus ostium and the anterior-superior commissure of the tricuspid valve (fig. 1), so that the pairs of the recording electrodes were parallel to the underlying course of the His bundle. The parallel alignment between electrode pairs and the His bundle was assessed from the characteristic falloff in amplitude of the six simultaneously recorded HBEs (fig. 2). Electrograms showing little or no difference in amplitude between several electrode pairs indicated an oblique orientation of the electrode probe with respect to the course of the His bundle; such complexes were not included in analysis. For the purpose of this study, only complexes showing a marked falloff in HBE amplitude were considered to be parallel to the His bundle course.

Six bipolar His bundle electrograms, an atrial electrogram or a stimulus artifact, and a scalar electrocardiographic lead were recorded from each patient on an Electronics for Medicine DR-10 switched-beam oscilloscope and recorded simultaneously on photographic paper moving at 100 mm per second. Electrocardiograms were recorded at frequencies from 0.1 to 500 Hz, and electrograms from 12 to 500 Hz. All leads in contact with the heart were isolated both from ground and the recording instruments by isolation transformers.

For analysis of the data, four to five electrograms recorded from each patient were selected and subjected to the following criteria: 1) normal and unvarying H-V (His bundle to QRS complex) interval; 2) approximate equiphasic decay of HBE lateral to a maximal electrogram (ME, fig. 2); and 3) normal atroventricular conduction se-
sequence by means of normal sinus rhythm or atrial pacing. Eleven of 21 patients met all criteria and are included in this study. The peak-to-peak amplitude of the HBE was measured in mm at the ME and at sites 1, 2, and 3 mm from the ME and converted to millivolts (10 mm = 1 mV, table 2). Because of variability among individual patients in the magnitude of the potentials observed, the electrograms recorded at sites lateral to the ME were normalized (His bundle amplitude divided by the amplitude of the ME, table 2).

Results
In the recordings from each patient, there was a maximal electrogram (ME) which was considered to originate through the electrode pair nearest to the voltage source, i.e., the His bundle. In addition, there was a clear and almost symmetrical decay in amplitude to both sides of ME (fig. 2, 3).

Table 2 and figure 3 present the range and mean amplitude of His bundle electrograms obtained at the site of ME and sites 1, 2, and 3 mm away from ME: the values ranged from 0.76 to 3.44 mV, 0.38 to 1.13 mV, 0.27 to 0.86 mV and 0.2 to 0.44 mV, respectively. Figure 4 displays the range and mean values of the normalized amplitude of the His bundle electrogram at each site and illustrates the fall-off (in percent) of amplitude lateral to the ME. A 57% decrease in HBE amplitude was observed 1 mm from the ME, 73% at 2 mm, and 82% at 3 mm.

Although considerable overlap existed between the maximal amplitude of the HBE (ME) and the amplitude of electrograms recorded 1 mm from this site, HBE amplitude at 2 mm was lower than ME. In only one of the 11 patients did the His bundle amplitude at 2 mm away from ME fall within the range of His bundle amplitude recorded at ME (fig. 3). No overlap of His bundle amplitude existed between the ME and HBE recorded at 3 mm from the ME. Further, disappearance or near extinction of the recorded HBE occurred on each side of ME at a distance of 2 to 3 mm in almost all of the 11 patients studied (fig. 3). Indeed, His bundle amplitude at site 3 exceeded 0.40 mV in only one of 11 patients. Recognition of the HBE at a site 3 mm from ME in this study was based primarily on the timing with electrograms recorded at adjacent sites (1 and 2) closer to the His bundle. Without simultaneous timing, electrograms recorded 3 mm from ME could not be reliably differentiated from base line undulation (figs. 2 and 5). It is therefore clear

### TABLE 1. Vital, Diagnostic, and Intraoperative Data of the 11 Patients Studied

<table>
<thead>
<tr>
<th>Name</th>
<th>Diagnosis</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>K mEq/L</th>
<th>Temp (°C)</th>
<th>Pre-anesthesia</th>
<th>Anesthesia (mg)</th>
<th>Induction</th>
<th>Maintenance</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. C.D.</td>
<td>VSD</td>
<td>3½</td>
<td>M</td>
<td>5.1</td>
<td>37</td>
<td>MS</td>
<td>2 DTC</td>
<td>DTC</td>
<td>30 DTC</td>
</tr>
<tr>
<td>2. C.S.</td>
<td>VSD</td>
<td>11</td>
<td>F</td>
<td>4.3</td>
<td>37</td>
<td>MS</td>
<td>3.5 SEC</td>
<td>H N20</td>
<td>N20 H</td>
</tr>
<tr>
<td>3. C.F.</td>
<td>VSD, PS</td>
<td>5½</td>
<td>M</td>
<td>4.0</td>
<td>32</td>
<td>MS</td>
<td>2 SC</td>
<td>H N20</td>
<td>N20 H</td>
</tr>
<tr>
<td>4. J.M.</td>
<td>ASD</td>
<td>8</td>
<td>M</td>
<td>4.2</td>
<td>37</td>
<td>MS</td>
<td>2 SEC</td>
<td>H N20</td>
<td>N20 H</td>
</tr>
<tr>
<td>5. M.R.</td>
<td>ASD</td>
<td>13</td>
<td>F</td>
<td>4.2</td>
<td>37</td>
<td>MS</td>
<td>3 SC</td>
<td>80 H</td>
<td>150 N20</td>
</tr>
<tr>
<td>6. H.O.</td>
<td>VSD, TI</td>
<td>12</td>
<td>M</td>
<td>4.2</td>
<td>37</td>
<td>—</td>
<td>4 SCOP</td>
<td>5 H N20</td>
<td>N20 H</td>
</tr>
<tr>
<td>7. M.J.</td>
<td>T/F</td>
<td>8</td>
<td>F</td>
<td>5.0</td>
<td>37</td>
<td>—</td>
<td>12 C</td>
<td>39 C</td>
<td>125 MS</td>
</tr>
<tr>
<td>8. D.V.</td>
<td>T/F</td>
<td>7</td>
<td>M</td>
<td>4.7</td>
<td>32</td>
<td>—</td>
<td>40 KET</td>
<td>25 H N20</td>
<td>C 25 V</td>
</tr>
<tr>
<td>9. S.R.</td>
<td>T/F</td>
<td>5</td>
<td>F</td>
<td>4.5</td>
<td>32</td>
<td>—</td>
<td>150 KET</td>
<td>10 MS</td>
<td>14 V 7.5</td>
</tr>
<tr>
<td>10. T.M.</td>
<td>ASD, VSD</td>
<td>1½</td>
<td>F</td>
<td>5.0</td>
<td>37</td>
<td>—</td>
<td>5 DTC</td>
<td>10.5 H N20</td>
<td>N20 H</td>
</tr>
<tr>
<td>11. T.M.</td>
<td>ASD</td>
<td>47</td>
<td>F</td>
<td>2.8</td>
<td>37</td>
<td>MS</td>
<td>6 MS</td>
<td>40 C</td>
<td>54 V 17.5</td>
</tr>
</tbody>
</table>

Abbreviations: M = male; F = female; ASD = atrial septal defect; VSD = ventricular septal defect; AS = aortic stenosis; PS = pulmonic stenosis; TI = tricuspid insufficiency; T/F = tetralogy of Fallot; C = curare; DTC = tubocurarine; H = halothane; R = serum potassium; KET = ketamine; MS = morphine sulfate; PENT = pentothal; SEC = seconal; SCOP = scopolamine; PAN = panceuronium; VIST = vistaril.
that when His bundle electrograms are recorded with this technique, the distance of the recording electrodes from the His bundle can be estimated from analysis of the recorded His bundle electrogram amplitude. Figure 6 relates percent decrease to distance from voltage source (His bundle) and illustrates an early rapid decline, followed by a more gradual one.

**Table 2.** Mean Voltage at Maximal Electrogram and Sites 1, 2, and 3 of the Patients Studied

<table>
<thead>
<tr>
<th>Patient</th>
<th>ME mV</th>
<th>Site 1 mV</th>
<th>Site 2 mV</th>
<th>Site 3 mV</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>%</td>
<td>%</td>
<td>%</td>
<td>%</td>
</tr>
<tr>
<td>1</td>
<td>2.00</td>
<td>100</td>
<td>0.82</td>
<td>40</td>
</tr>
<tr>
<td>2</td>
<td>2.20</td>
<td>100</td>
<td>1.00</td>
<td>45</td>
</tr>
<tr>
<td>3</td>
<td>1.10</td>
<td>100</td>
<td>0.51</td>
<td>45</td>
</tr>
<tr>
<td>4</td>
<td>1.50</td>
<td>100</td>
<td>0.75</td>
<td>50</td>
</tr>
<tr>
<td>5</td>
<td>1.30</td>
<td>100</td>
<td>0.56</td>
<td>44</td>
</tr>
<tr>
<td>6</td>
<td>1.90</td>
<td>100</td>
<td>0.96</td>
<td>50</td>
</tr>
<tr>
<td>7</td>
<td>0.91</td>
<td>100</td>
<td>0.47</td>
<td>52</td>
</tr>
<tr>
<td>8</td>
<td>2.22</td>
<td>100</td>
<td>1.13</td>
<td>51</td>
</tr>
<tr>
<td>9</td>
<td>0.76</td>
<td>100</td>
<td>0.38</td>
<td>50</td>
</tr>
<tr>
<td>10</td>
<td>3.44</td>
<td>100</td>
<td>0.94</td>
<td>27</td>
</tr>
<tr>
<td>11</td>
<td>1.45</td>
<td>100</td>
<td>0.20</td>
<td>27</td>
</tr>
<tr>
<td>Mean</td>
<td>1.71</td>
<td>100</td>
<td>0.72</td>
<td>43</td>
</tr>
</tbody>
</table>

Voltage in mV and in percent (voltage at site divided by voltage of maximal electrogram, ME, $\times 100$) at the ME and at lateral sites 1, 2, and 3, in each patient. The mean voltage of each patient was obtained from 4–5 beats, and at each lateral site was derived from the combined data recorded from both sides of ME (see fig. 2). Data were not recorded from site 3 in patient 9 because of technical failure.

**Discussion**

The problem of delineating the specialized conduction system of the heart has been a dilemma for the cardiac surgeon since the inception of heart surgery. In 1960, intraoperative recordings of His bundle and right bundle branch electrograms were described in patients undergoing cardiac surgery for closure of a ventricular septal defect. More recently, intra-atrial and intraventricular delineation of the specialized conduction system has been applied in certain complex lesions. Although electrograms generated by the specialized conduction system are readily recognized and recorded during surgery, it is important to evaluate the precision of this technique in man, and specifically to determine at what distance from the His bundle the His bundle electrogram cannot be detected.

**Electrophysiological Considerations**

The study of the electrophysiology of excitable tissue is dependent, in large part, on the method. To draw parallels between events of single fiber preparations and those of surface recordings is difficult and complex. Our data were obtained during open heart surgery, during cardiopulmonary bypass, and under general anesthesia; under these circumstances minor variations in the symmetrical falloff could not be completely avoided (fig. 2). Also, variations in size and course of the underlying His bundle, its depth under the endocardial surface, alterations in conduction velocities of single fibers, and the possibility of pre-existing disease within the His bundle can alter the amplitude of the recorded His bundle electrogram. Further, local metabolic effects of hypothermia, hypokalemia, or pressure from the electrode probe may alter resting and threshold potential, and thereby decrease the generated action potential. Interelectrode distance may also affect the amplitude, shape, and duration of the recorded electrograms. Attempts to ac-
count for metabolic factors revealed only minor variation both in the preoperative serum potassium and in the patient’s temperature and anesthesia during the electrophysiologic study (table 1). Our observations also show that in spite of the marked variability in the absolute potential generated by each patient, the amplitude of the His bundle electrograms decays with distance in a similar manner in all patients (fig. 3). Further, our data on the amplitude-distance relationship of human His bundle potential resemble in form previously described observations obtained by other techniques (unipolar, microelectrode) and in other excitable tissues.10, 11, 15, 18

Alanis et al.,17 in identifying the HBE in dogs through plunge electrodes, observed a biphasic deflection along the entire anatomic course of the His bundle; the amplitude varied from 0.8 to 2.0 mV. He also noted that if a bipolar microelectrode pair was placed such that each single microelectrode was 1 to 2 mm to either side of the His bundle, the HBE disappeared. Our data indicated that at 1 mm from ME there was a 50% decrease in the potential recorded, and at 3 mm from ME the His bundle electrogram usually became indiscernible. Given the 1-2 mm width of the His bundle in the hearts of persons beyond infancy,19, 18 and the disappearance of the His bundle electrogram at distances of 2-3 mm from the ME, extinction of the His bundle potential in man appears to occur at approximately 2-3 mm to either side of the His bundle (fig. 5) when recording with closely spaced bipolar electrodes.

Similarly in studies of isolated frog muscle fibers and isolated strands of canine ventricular conduction system, the amplitude of the electrogram varied as a function of the distance from the surface of the tissue studied. Hakanson and associates,18 studying isolated frog muscle preparations with unipolar extracellular electrodes, found that the amplitude of an electrogram decreased markedly with increasing distance from the fiber, the decrease being linear with the logarithm of the distance. This relationship held true at small distances (less than 0.15 mm); at distances greater than 0.15 mm, the amplitude decreased as a function of distance (d^{-1.9}). The relative decrease in amplitude was identical for fibers of different circumferences. Further, studies in

![Figure 3: Amplitude in mV (potential) of His bundle electrograms plotted against recording site in the 11 patients. Note the mean equiphasic decay to either side, indicating localization of the His bundle at ME.](image)

![Figure 4: Percent decrease in His bundle amplitude at sites lateral to ME. There is a 57% drop at 1 mm from the ME, 73% drop at 2 mm, and 82% at 3 mm.](image)

![Figure 5: His bundle electrograms (HBE) recorded in three patients at the ME, 2 mm and 3 mm from ME, and illustrating the near extinction of the His bundle electrogram 3 mm from the ME.](image)
isolated Purkinje fibers and free-running false tendons of the canine left ventricle have also shown an amplitude-distance relationship. Myerberg et al.,

11 using closely spaced bipolar electrodes, observed an exponential decrease in the electrogram voltage with increasing distance. The voltage fell off from 7.5 mV when the electrodes were touching the surface to less than 1 mV at a distance of 0.48 mm from the tissue. A predictable rate of decay, however, could not be demonstrated. Spach et al.

16 showed with unipolar extracellular electrodes a marked decrease in the amplitude of the electrogram as the extracellular electrode was withdrawn from the surface of isolated canine Purkinje fibers; the decline was steep initially, thereafter decreasing more gradually, and could be described at all distances by a single function. This same function continued to predict the fall-off in the presence of interventions such as hypothermia and hypokalemia which alter intracellular action potential characteristics and conduction velocity.

The difference between recording techniques and preparations used in the above cited studies and in our study does not permit a close comparison. Nonetheless, in our study there was a clear demonstration in each case of His bundle localization by a large maximal electrogram (fig. 2). The falloff of the amplitude of the lateral electrograms indicated that there is a loss of potential in the electrograms recorded as one moves from the His bundle. This decrease in HBE amplitude, furthermore, was initially rapid, then more gradual, a form common to voltage-distance relationships described by investigators employing different methods and material.11, 16 17 We were unable, however, to predict with precision the distance the electrodes were from the voltage source. Nonetheless, as is discussed below, clinical usefulness of the method was, we believe, demonstrated.

**Clinical Considerations**

The data obtained in this study have important clinical applicability. No data were previously known as to the distance from the His bundle at which human HBE could be recorded using the technique of closely spaced bipolar electrodes. Previous studies have suggested that electrophysiological delineation of the cardiac specialized conduction system can be performed with closely (1 mm) spaced bipolar electrodes.2 3 In these previous studies, a 5 mm movement of the recording electrode probe resulted in the disappearance of the specialized conduction system electrograms. The interelectrode distance in the electrode probe used in the current study is identical to that used clinically and in the previously described studies. It is therefore clear that, with parallel alignment, an electrode probe with an interelectrode interval of 1 mm is sufficiently sensitive to delineate the course of the specialized conduction system with an accuracy of about 5 mm, as previously suggested (i.e., 2–3 mm on each side of a maximally recorded electrogram).

No conclusions can be inferred from the data obtained in the current study as to the distance from the His bundle at which His bundle electrograms can be recorded with more widely spaced electrodes such as are commonly used with the electrode catheter technique.16 It is expected, however, that such widely spaced (10 mm apart) electrodes will be less precise in locating the specific underlying specialized conduction system but still useful in studies on the timing and ordering of activation sequences.10, 18

Although His bundle electrograms can be recorded up to 2–3 mm from the ME, the data provided in this study suggest that the His bundle can be localized within 1–2 mm by analysis of His bundle electrogram amplitude. Since HBE amplitude was equal to or greater than one mV in nine of 11 patients at ME, and less than 1 mV in 9 of 11 patients at 1 mm, and in all patients at 2 mm, it is reasonable to conclude that when a recorded His bundle electrogram is smaller than 1 mV, the electrode probe is 2–3 mm away from the His bundle and a fine adjustment in the localization of the electrode probe will place it within 1 mm of the His bundle. The data obtained in this study as to the precision of the method and the spatial behavior of the recorded His bundle electrogram amplitude are therefore helpful in the clinical application of the technique.

**Acknowledgments**

We are indebted to Mr. Tony Howard, Misses Eleanor Monkowski and Pamela Stover for their excellent technical assistance.

**References**

6. Kupersmith J, Krongrad E, Gersony WM, Bowman FO Jr: Electrophysiologic identification of the specialized conduction system in cor-

**Figure 6** Percent falloff in amplitude (potential) of His bundle at sites lateral to ME. This single-arm curve was derived from combined data from both sides of ME. The initial rapid decline is followed by a more gradual one, resembling curves obtained using different techniques. X ± 1 SD = mean ± one standard deviation.
Vectorcardiographic Criteria for the Diagnosis of Anterior Myocardial Infarction

JOHN W. STARR, M.D., GALEN S. WAGNER, M.D., RICHARD M. DRAFFIN, M.D., JOHN B. REED, M.D., ABE WALSTON, II, M.D., AND VICTOR S. BEHAR, M.D.

SUMMARY  Frank lead vectorcardiograms (VCG) from four carefully selected patient subgroups (226 patients) were analyzed to develop optimal criteria for the diagnosis of anterior myocardial infarction. Specificity was evaluated using 100 healthy volunteers under age 30 and 80 patients with normal left ventriculogram and normal coronary arteriograms. Sensitivity was determined using 25 patients with evolutionary ST-T wave changes (V1-6), and LDH and CPK isoenzyme evidence of acute myocardial infarction; and 21 patients with anterior wall akinesia or dyskinesia and >70% occlusion of the left anterior descending coronary artery. Patients with VCG evidence of bundle branch block, left or right ventricular hypertrophy were excluded. The criteria for the diagnosis of anterior myocardial infarction which was found to give the highest sensitivity with >95% specificity was: initial anterior QRS forces must not exceed 0.1 mV in maximal amplitude and also must not exceed 24 msec in duration. The performance of this proposed criterion was then tested using four similarly defined patient subgroups consisting of a total of 222 patients. The incidence of false positive diagnosis in these test subgroups was <1% with a sensitivity of >95%. The overall performance of the proposed criterion was found to be significantly superior to both the widely accepted VCG and ECG criteria for anterior myocardial infarction. Thus, this quantitative criterion using both time and duration of initial anterior forces is both a highly specific and a sensitive indicator of anterior myocardial infarction.

VCG criteria for diagnosis of inferior myocardial infarction which were significantly more specific than previously published criteria.

Methods

Patient Selection

Our purpose was to evaluate the quantitative descriptors of the QRS complex in living patients who had strong evidence for AMI and in subjects who had a normal myocardium by clinical criteria. Patients were divided into two groups. Data from the first were used to develop the criteria (criteria group) and from the second to test these criteria (test group).

The criteria group (226 patients) included two subgroups (A and B) in which the presence of AMI was most unlikely and two subgroups (C and D) in which the diagnosis of AMI was established without regard to descriptors of the QRS. Subgroup A was composed of 100 healthy volunteers aged 20-29, without history of heart disease or hypertension. Subgroup B was composed of 80 patients who had undergone coronary arteriography because of chest pain...
M Dick, 2nd, E Krongrad, R E Antar, S Ross, F O Bowman, Jr, J R Malm and B F Hoffman

Circulation. 1976;53:224-229
doi: 10.1161/01.CIR.53.2.224
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/53/2/224

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/