Normalization of Bundle Branch Block Patterns by Distal His Bundle Pacing

Clinical and Experimental Evidence of Longitudinal Dissociation in the Pathologic His Bundle

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SUMMARY Clinical and experimental observations in which bundle branch block patterns (BBBP) in ECG leads were normalized by distal His bundle (H) pacing are reported. The clinical material includes four patients with acute right BBBP secondary to anterior wall myocardial infarction and three patients with chronic left BBBP. Six of the seven patients had a prolonged H-V interval (60–85 msec) including three who showed evidence of an intra-H conduction delay (IHCD) with split H (H and H'). Distal H pacing from a right-sided electrode catheter normalized the BBBP with a stimulus-to-QRS (P-Q interval 20–35 msec shorter than the H-V interval and almost identical to the H'-V interval in the three patients with documented IHCD. In 18 dogs ligation of the anterior septal artery resulted in IHCD with split H associated with right or left BBBP. Distal H pacing from catheter and/or plunge wire electrodes normalized the BBBP in 12 experiments (67%) with a P-Q interval identical to the H'-V interval. H pacing was selective and direct stimulation of myocardium was excluded by monitoring the high ventricular septal electrogram. The clinical and experimental observations are discussed as evidence that functional longitudinal dissociation is probably only operative in the pathologic H due to selectively greater depression of conduction in the transverse interconnections.

CONVENTIONALLY, bundle branch block patterns in electrocardiographic leads including the hemiblocks are considered to represent conduction delay and/or block in the corresponding bundle branch or fascicle.1–2 The possibility that bundle branch block patterns in the electrogram may be caused by more distal conduction delays in the Purkinje network, Purkinje-muscle junctions and the working myocardium (so called peripheral or parietal blocks) has been entertained for years.3 Probably for as many years the alternative hypothesis that bundle branch block patterns can occur secondary to a more proximal lesion in the A-V junction was suggested.4 Several experimental studies5–11 and few clinical observations12–13 examined the concept that functional longitudinal dissociation in the A-V node and/or the His bundle can result in significant alteration in the ventricular activation pattern. Some of these studies presented controversial evidence especially in regard to the functional significance of the transverse interconnections in the His bundle which could mitigate against longitudinal dissociation of conduction under normal physiologic conditions.

In this report both experimental and clinical observations in which bundle branch block patterns were normalized by distal His bundle pacing are presented. These observations are discussed as evidence of functional longitudinal dissociation of conduction in the pathologic His bundle. This concept can have significant clinical implications if it is proved that bundle branch block patterns secondary to His bundle lesions are more likely to progress to complete A-V dissociation. Part of the present material has been reported previously in abstract form.14

Experimental Observations-Material and Method

Experiments were performed in adult mongrel dogs weighing 10–20 kg and anesthetized with intravenous sodium pentobarbital (20 mg/kg). A Harvard respirator using room air provided mechanical ventilation through a cuffed endotracheal tube. Blood pressure in the femoral artery was monitored through a polyethylene catheter connected to a Statham transducer. A thoracotomy incision was made through the left fourth intercostal space. The bifurcation of the left coronary artery was exposed by retracting the tip of the left atrial appendage and incising the epicardium overlying the proximal portions of the anterior descending and left circumflex arteries. The anterior septal artery was exposed by blunt dissection of the bifurcation and branches of the left coronary artery and a silk ligature was placed around the vessel to be occluded after taking control records.

The left thoracotomy was then closed and the animal turned to expose the heart through a right thoracotomy. One to three pairs of plunge wire electrodes were placed in the His bundle area through a 22 gauge needle 1½ inches in length containing two teflon-coated stainless steel wires (0.007 inches coated diameter).16 The cut ends of the wires served as close bipolar pairs. The position of the recording wires was considered satisfactory when late septal depolarization dominated the ventricular deflection and was inscribed coincident with the terminal part of the surface QRS and/or slightly afterward. Invariably, a His bundle deflection and low amplitude atrial activity were also seen on these recordings. Recordings of the His bundle and regular ventricular muscle were also obtained by electrode catheters (5 French with ring electrodes 10 mm apart).17 The catheters were either inserted into a common carotid artery and advanced to the aortic root or into a femoral vein and positioned in the right side of the heart across the tricuspid valve. Validation of the catheter electrode and plunge wire recordings were made by pacing from the various sites, together with analysis of the QRS configuration of the paced beats.

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impulse (PI) and the relationship of the paced impulse to the QRS complex (V).

In some experiments two reference electrodes were placed in the lateral free walls of the right and left ventricles at sites approximately equidistant from the ventricular septum. These recordings were achieved by inserting two fine teflon-coated stainless steel wires (0.003 inches diameter) into the epicardium through a 25 gauge needle. In addition to the electrograms, two or more standard electrocardiographic leads were recorded, specifically leads II and aVR. All records were obtained on a multichannel oscilloscopic photographic recorder (E for M, DR-8) at paper speeds of 25–200 mm/sec with the filter frequencies of 0.1–200 Hz for ECG leads and 40-200 Hz for electrogram recordings. Some of the recordings were stored on a magnetic tape recorder (Honeywell 5600) and replayed so that selected sections could be transferred to photographic paper for detailed analysis. Measurements were accurate up to ± 3 msec at a paper speed of 200 mm/sec.

Atrial pacing (2 msec duration, 180–200 pulses per minute and 2–10 volts) was achieved via two fine stainless steel wires (0.003 inches diameter) inserted by a 25 gauge hypodermic needle into the right atrial appendage. Pacing was performed with a Grass S-88 stimulator and SIU 5 isolation unit. For slowing of the heart rate, two silver wires (0.012 inches diameter) were inserted into the distal portion of the right or left vagosympathetic trunk. Vagal stimulation was accomplished by delivery of 0.05 msec square wave pulse of 1–10 V intensity at a frequency of 20 Hz. In all experiments postmortem dissection was performed to see that the anterior septal artery had been completely occluded, as well as to verify the position of the plunge wires which were used to record His bundle electrical activity. In addition, measurements of interelectrode distance of the plunge wires as well as the distance between each pair of wires were done in each experiment in which plunge wire recordings were obtained. The spatial alignment of the wires to each other as judged at postmortem was always correlated with the temporal relationship of the rapid activation spike of the His bundle potential recorded by each pair of wires.

**Procedures**

Control recordings during sinus rhythm, vagal-induced cardiac slowing, and atrial pacing up to rates that produced atioventricular conduction of the Wenckebach type were obtained in each experiment before the anterior septal artery was ligated. Abrupt one-stage ligation of the anterior septal artery was performed. After subsidence of the initial arrhythmic period, the recorded electrical activity was continuously monitored for the onset of intra-His bundle conduction delays and/or bundle branch block patterns. In experiments showing first degree intra-His bundle block (split His bundle potential) associated with a bundle branch block pattern in ECG leads, His bundle pacing at different rates was performed from the plunge wire electrodes and/or electrode catheters recording the split His bundle potential.

**Results**

Anterior septal artery ligation was performed in 40 dogs. Thirty-eight dogs that survived the initial arrhythmic period developed various combinations of intra-His bundle block and bundle branch block patterns within 30–120 min following ligation (table 1). Eighteen dogs showed an intra-His bundle conduction delay in the form of a split His bundle potential closely associated with the appearance of a bundle branch block pattern (BBBP) in ECG leads (right BBBP in 12, left BBBP in four and alternating RBBB and LBBB in two). The diagnosis of incomplete or complete BBBP was made from QRS configurations in leads II and aVR as previously described. In these 18 experiments the effect of pacing from the proximal and distal His bundle on the QRS configuration of BBBP in ECG leads was studied. Pacing was obtained from plunge wire electrodes and/or electrode catheters recording the split His bundle potential.

**Table 1. Localization of Conduction Disorders in the Canine Proximal His-Purkinje System (HPS) following Ligation of the Anterior Septal Artery in 38 Experiments**

<table>
<thead>
<tr>
<th>Group</th>
<th>Number of experiments</th>
<th>Site of conduction disorder</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>His bundle</td>
</tr>
<tr>
<td>Group I</td>
<td>2</td>
<td>-</td>
</tr>
<tr>
<td>Group II</td>
<td>10</td>
<td>+</td>
</tr>
<tr>
<td>Group III</td>
<td>12</td>
<td>+</td>
</tr>
<tr>
<td>Group IV</td>
<td>8</td>
<td>-</td>
</tr>
<tr>
<td>Group V</td>
<td>2</td>
<td>+</td>
</tr>
</tbody>
</table>

and bundle branch block patterns within 30–120 min following ligation (table 1). Eighteen dogs showed an intra-His bundle conduction delay in the form of a split His bundle potential closely associated with the appearance of a bundle branch block pattern (BBBP) in ECG leads (right BBBP in 12, left BBBP in four and alternating RBBB and LBBB in two). The diagnosis of incomplete or complete BBBP was made from QRS configurations in leads II and aVR as previously described. In these 18 experiments the effect of pacing from the proximal and distal His bundle on the QRS configuration of BBBP in ECG leads was studied. Pacing was obtained from plunge wire electrodes and/or electrode catheters recording the split His bundle potential.

Figures 1 and 2 illustrate an experiment in which an intra-His bundle conduction delay developed associated with a right BBBP in ECG leads following ligation of the anterior septal artery as well as the results of pacing the proximal and distal His bundle from plunge wire electrodes. In figure 1, tracings from top to bottom are ECG leads 2 and aVR, electrode catheter recording of the His bundle from the aortic root (Hb-LV) and two plunge wire recordings from proximal and distal His bundle sites (Hb1 and Hb2, respectively). Note the presence of sharp His bundle spikes in the control recording (panel A). The control H-V interval measured from both the Hb-LV and Hb2 recordings was 34 msec while the intra-His bundle conduction time between the proximal and distal plunge wire recordings was 6 msec. Control pacing from the electrode catheter and plunge wire electrodes resulted in a supraventricular QRS and a stimulus-to-QRS (PI-V) interval equal to the H-V interval obtained by each recording. Panel B was obtained 60 min following ligation of the anterior septal artery. The ECG leads show the development of an incomplete RBBB. The Hb (LV) recording shows slurring of the terminal S wave of the His bundle potential and an increased duration of both the His bundle potential and H-V interval reflecting the occurrence of an intra-His bundle conduction delay. There was relatively little change in the His bundle potential recorded by the proximal plunge wire electrode (Hb2) but the H-V interval lengthened from 34 to 45 msec. On the other hand, the distal plunge wire recording (Hb1) showed a decrease in the amplitude and widening of the distal His bundle potential which was synchronously recorded with the terminal S wave of the His bundle potential in the electrode catheter recording. A split His bundle potential (H1 and H2) in the Hb (LV) recording developed 90 min following ligation at a basic cardiac cycle of 405 msec. This was associated with further
lengthening of the H1-V interval to 56 msec. The intra-His bundle conduction delay was also displayed in the plunge wire recordings. Concomitant with the increase in intra-His bundle conduction delay, the ECG leads showed the development of a complete right BBBP. Panel C illustrated that conduction in the His bundle was tachycardia dependent with the occurrence of 2:1 intra-His bundle block during atrial pacing at a cycle length of 200 msec.

Figure 2 was obtained immediately following the recording of panel C in figure 1 and illustrates the results of His bundle pacing at different heart rates from both the proximal and distal plunge wire electrodes. Panel A shows pacing from the proximal plunge wire electrode at a cycle length of 270 msec. There was a 1:1 response to Hb pacing with a PI-V interval equal to the H1-V interval and a QRS configuration of complete right BBBP similar to conducted supraventricular beats. Panel B shows that increasing the pacing cycle length to 260 msec resulted in a 2:1 response. This was similar to the response to atrial pacing at the same cycle length and similarly reflected the occurrence of a 2:1 intra-Hb block. Panel C illustrates the results of pacing from the distal plunge wire electrode utilizing the same stimulus strength and polarity. Distal Hb pacing at a cycle length of 370 msec resulted in a PI-V interval of 32 msec which was approximately equal to the H2-V interval of 34 msec. Pacing also resulted in normalization of the complete right BBBP. The QRS configuration in surface leads was remarkably similar to the control recording in figure 1, panel A. The ST-T alteration (fig. 2, panel C) compared to control (fig. 1, panel A) reflects myocardial ischemia secondary to the anterior septal artery ligation. In contrast to the occurrence of a 2:1 block during proximal Hb pacing at a cycle length of 260 msec, distal Hb pacing at the same cycle resulted in 1:1 response with a PI-V interval and a QRS configuration similar to the one in figure 2, panel C. Figure 2, panel D shows that distal Hb pacing at a shorter cycle of 200 msec was still associated with a 1:1 response. The slight increase in the PI-V interval and the occurrence of an incomplete left

Figure 1. Recordings obtained from a canine experiment in which an intra-His bundle conduction delay developed associated with a right bundle branch block pattern following ligation of the anterior septal artery. Panel A shows control recording. Panels B and C were obtained 60 and 90 min following ligation and illustrate the gradual occurrence of an intra-His bundle conduction delay and a split His bundle potential (H1 and H2) associated with an incomplete block. Panel C also illustrates the effect of rapid atrial pacing at an atrial cycle length (PI-PI) of 200 msec. This resulted in a 2:1 intra-His bundle block.

Figure 2. Recordings obtained from the same experiment shown in figure 1 to illustrate the effect of His bundle (Hb) pacing at different rates from both the proximal and distal plunge wire electrodes. Distal Hb pacing as opposed to proximal Hb pacing resulted in normalization of the right bundle branch block pattern (panel C) as well as a 1:1 response at fast pacing rates (panel D).
BBBP at this fast pacing rate probably reflects a tachycardia-dependent conduction delay in the left bundle.

Figures 3–5 illustrate another experiment in which an intra-Hb conduction delay developed in association with a right BBBP in ECG leads following ligation of the anterior septal artery. The results of Hb pacing applied through the electrode catheter recording the split Hb potential was studied. In figure 3, tracings from top to bottom are ECG leads 2 and aVR, an electrode recording from the aortic root (Hb eg), a plunge wire recording from the His bundle area (HVS eg) and two bipolar epicardial recordings from the lateral free wall of the right and left ventricles at sites approximately equidistant from the ventricular septum (RV eg and LV eg, respectively). In experiments in which Hb pacing was applied through the electrode catheter recording the split Hb potential, the gain on the plunge wire Hb recording was decreased to emphasize the ventricular deflection which represents high ventricular septal activation (HVS eg). Control recordings in figure 3, panel A show that the HVS electrogram is inscribed coincident with the terminal part of the surface QRS and later than the synchronously recorded right and left ventricular electrograms. This illustrates that the upper part of the ventricular septum is activated later than the epicardial surface of the right and left ventricles.

Figure 3, panel B shows control Hb pacing from the electrode catheter. The Hb was selectively stimulated as revealed by: 1) a QRS configuration similar to conducted supraventricular beats, 2) a PI-V interval equal to the H-V interval, 3) absence of simultaneous atrial stimulation and 4) no apparent change in the timing of the RV eg and LV eg and in particular the HVS eg. The persistence of late depolarization of the high ventricular septum during Hb pacing excluded the possibility of simultaneous pacing of the septal myocardium and emphasized the selective Hb pacing. Figure 3, panel C was obtained 75 min following ligation of the anterior septal artery. The record illustrates the development of an intra-Hb conduction delay (a split Hb potential and lengthening of the H-V interval from 35 to 60 msec) associated with a complete right BBBP in ECG leads. Note the delayed inscription of the RV eg which is now being recorded later than the HVS eg denoting late activation of the epicardial surface of the right ventricle.

Figure 4 was obtained shortly following the recording in figure 3, panel C and illustrates Hb pacing from the electrode catheter. In panels A and B, the stimulus strength was kept constant at 15 V but the polarity was changed. This resulted in a proximal Hb pacing in panel A and a distal Hb pacing in panel B. As would be anticipated, the structures directly stimulated at relatively low stimulus intensity by a bipolar electrode in opposition to A-V junctional tissues would depend upon which pole was selected as the cathodal stimulating site. 21 In panel A, proximal Hb stimulation was not selective since the atria were simultaneously activated. The PI-V interval was equal to the H-V interval and the tim-

**FIGURE 3.** Recordings obtained from another canine experiment in which an intra-His bundle conduction delay developed in association with a right bundle branch block pattern following ligation of the anterior septal artery. Panel A shows control recordings while panel B illustrates the control His bundle pacing from the electrode catheter. Panel C was obtained 75 min following occlusion and illustrates the development of a split His bundle potential (H and H') and a right bundle branch block pattern.

**FIGURE 4.** Recordings obtained from the same experiments shown in figure 3 that illustrate both proximal (panel A) and distal (panel B) His bundle pacing. Both tracings were obtained by pacing from the electrode catheter recording the split His bundle potential by reversing the stimulus polarity. Note a shorter PI-V interval during distal His bundle pacing and persistence of the right bundle branch block pattern during distal His bundle pacing at the relatively low stimulus intensity of 15 V.
and distal Hb pacing at a relatively low stimulus intensity of 15 V resulted in a QRS configuration of complete right BBBP similar to conducted supraventricular beat. Figure 5, obtained from the same experiment, illustrates the effect of slight increase of the stimulus strength during distal Hb pacing on the QRS configuration of paced beats. The upper and lower tracings in figure 5 represent a continuous recording. The first three beats in the upper recording show distal Hb pacing with a stimulus intensity of 15 V and a QRS configuration of right BBBP. Slight increase of the stimulus intensity from 15 to 22 V resulted in normalization of the QRS configuration of right BBBP (the last two beats in the upper recording). The lower recording shows that a gradual decrease of the stimulus intensity resulted in the reappearance of the right BBBP. The PI-V interval remained the same during distal Hb pacing with both normal QRS configuration and right BBBP. As expected, the RV eg was only delayed during paced beats with right BBBP. However, the timing of the HVS eg in relation to the pacing stimulus as well as to the onset of the QRS complex remained the same during paced beats with both normal QRS configuration and right BBBP. This provides evidence that selective Hb pacing was maintained despite the slight increase of the stimulus intensity and excludes direct stimulation of the upper part of the ventricular septum during increased stimulus intensity.

The observation that distal Hb pacing could normalize the BBB pattern only at a relatively high stimulus intensity was demonstrated in four other experiments. In each of these experiments monitoring of the HVS eg was obtained to exclude direct septal activation at the higher pacing strength. Table 2 summarizes the results of distal Hb pacing in 18 experiments in which an intra-Hb conduction delay was associated with a BBBP in ECG leads. Selective distal Hb pacing normalized the BBBP in 12 of the 18 experiments (67%). The intra-Hb conduction delay (H1-H2 interval) associated with a BBBP in ECG leads ranged from 16-35 msec. The PI-V interval during distal Hb pacing was always

Table 2. Results of Distal His Bundle Pacing in 18 Experiments in Which an Intra-His Bundle Conduction Delay Was Associated with a Bundle Branch Block Pattern

<table>
<thead>
<tr>
<th>No of experiment</th>
<th>1st IHBB (H-V interval with normal QRS in msec)</th>
<th>RBBBP</th>
<th>LBBBP</th>
<th>Distal His pacing with normal QRS</th>
<th>PI-V interval during distal His pacing with normal QRS (msec)</th>
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<tbody>
<tr>
<td>1</td>
<td>56-34</td>
<td>+</td>
<td>−</td>
<td>+</td>
<td>32</td>
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<td>−</td>
<td>−</td>
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<td>−</td>
<td>+</td>
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<td>9</td>
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<td>46-30</td>
<td>+</td>
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<td>+</td>
<td>30</td>
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</table>

Abbreviations: 1st IHBB: first degree intra-His bundle block; RBBBP: right bundle branch block pattern; LBBBP: left bundle branch block pattern.
closely similar to the H₂-V interval. In experiments in which
distal Hb pacing was applied through the electrode catheter,
right BBBP could be normalized by pacing from a catheter
in the aortic root while LBBB was normalized by pacing
from a right sided electrode catheter.

Clinical Observations-
Material and Methods

Seven patients with BBBP in ECG leads and in whom dis-
tal Hb pacing normalized the QRS were analyzed. The
patients fall in two groups: I) four patients who developed
acute right BBBP in the course of an acute anterior wall
myocardial infarction, II) three patients with chronic left
BBBP. In the first group of patients the His bundle electro-
gram was recorded either during the insertion or removal of
a temporary ventricular pacemaker after obtaining the
patient’s informed consent. Recordings were obtained using
a no. 5 bipolar electrode catheter (1 cm interpolar distance)
passed percutaneously via the femoral vein and positioned
across the tricuspid valve.2 Simultaneous recording of ECG
leads I, aVF and V₁ or I, II, III and V₁ were obtained. In the
Hb electrogram, the A-H interval was measured from the
first rapid deflection of the A wave to the first rapid de-
fection of the Hb potential and was taken to represent A-V
nodal conduction time (normal 50–120 msec).23 The H-V in-
terval was measured from the Hb deflection to the earliest
ventricular activation recorded in either the intracardiac
electrogram or surface leads and reflected His-Purkinje con-
duction time (normal 35–50 msec).22 His bundle pacing24
was performed using a programmed digital stimulator that
delivered rectangular impulses of 1.5 msec duration using
the lowest milliamperage that permitted stable His bundle
stimulation. Pacing from slightly different catheter positions
as well as reversing stimulus polarity were tried to insure
proximal and/or distal His bundle pacing. Attempts were
made in all patients to separately stimulate the proximal and
distal Hb as well as the proximal right bundle region.

Results

Table 3 summarizes the results of the electrophysiologic
studies including Hb pacing in all seven patients. Group I
(patients 1–4) had acute right BBBP with normal frontal plane
axis in three and left axis deviation in one. All patients
had acute anterior wall myocardial infarction and one
patient had an old inferior wall infarction. All four patients
had a prolonged H-V interval of 60–85 msec. In patients 1
and 2 an intra-Hb conduction delay was illustrated by the
recording of proximal and distal Hb deflections (H and H'),
respectively from two different electrode catheter positions.
The H-H' interval was 20–25 msec. In these two patients
pacing from the proximal electrode catheter position resulted
in a QRS configuration of right BBB identical to
conducted supraventricular beats and a PI-V interval similar
to the H-V interval. On the other hand, pacing from the dis-
tal electrode catheter position normalized the BBBP with a
PI-V interval equal to 5 msec shorter than the H'-V interval.
Proximal and distal Hb pacing was obtained utilizing the
same stimulus strength and polarity. In patients 3 and 4, an
intra-Hb conduction delay could not be documented because
of failure to record a split Hb deflection from the same elec-
"TABLE 3. Clinical, Electrophysiologic Data in Seven Patients with Bundle Branch Block Pattern Normalized by Distal His Bundle Pacing"

<table>
<thead>
<tr>
<th>Pt/Age/Sex</th>
<th>Clinical diagnosis</th>
<th>Control QRS Duration (sec)</th>
<th>BBBP Frontal axis</th>
<th>Hb Study P-A (msec) A-H (msec) H-V (msec)</th>
<th>Normalized QRS Duration (sec)</th>
<th>Frontal axis PI-V (msec)</th>
<th>Follow-up</th>
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<tbody>
<tr>
<td>1/65/M AAWMI Old IWI</td>
<td>0.14</td>
<td>RBBBP +10</td>
<td>35</td>
<td>H-V = 70</td>
<td>+20</td>
<td>45</td>
<td>Permanent pacemaker, 1:1 A-V conduction and LBBBP 6 month later</td>
</tr>
<tr>
<td>2/58/M AASMI</td>
<td>0.14</td>
<td>RBBBP +35</td>
<td>38</td>
<td>H-V = 65</td>
<td>+35</td>
<td>40</td>
<td>Permanent pacemaker, 2:1 A-V block 9 months later</td>
</tr>
<tr>
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<td>0.12</td>
<td>RBBBP -45</td>
<td>42</td>
<td>H-V = 40</td>
<td>-35</td>
<td>50</td>
<td>Died from pump failure on the fifth hospital day</td>
</tr>
<tr>
<td>4/55/F AASMI</td>
<td>0.14</td>
<td>RBBBP -10</td>
<td>30</td>
<td>H-V = 45</td>
<td>+5</td>
<td>45</td>
<td>Permanent pacemaker, 2:1 A-V block 6 months later</td>
</tr>
<tr>
<td>5/60/M HCVD</td>
<td>0.14</td>
<td>LBBBP -35</td>
<td>30</td>
<td>H-V = 65</td>
<td>+15</td>
<td>45</td>
<td>1:1 A-V conduction and LBBBP 12 months later</td>
</tr>
<tr>
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<td>0.15</td>
<td>LBBBP +20</td>
<td>43</td>
<td>H-V = 45</td>
<td>+30</td>
<td>40</td>
<td>1:1 A-V conduction and LBBBP 9 months later</td>
</tr>
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<td>7/68/F HCVD</td>
<td>0.13</td>
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<td>41</td>
<td>H-V = 45</td>
<td>+20</td>
<td>39</td>
<td>1:1 A-V conduction and LBBBP 12 months later</td>
</tr>
</tbody>
</table>

Abbreviations: AAWMI = acute anterior wall myocardial infarction; AASMI = acute anteo septal myocardial infarction; IWI = inferior wall myocardi-
mal infarction; HCVD = hypertensive cardiovascular disease; ASHD = arteriosclerotic heart disease; RBBBP = right bundle branch block pattern; LBBBP = left bundle branch block pattern; Hb = His bundle.
associated with only slight changes in the frontal plane axis. This includes patient 4 who had a left axis deviation.

One patient in group I died in the first week following infarction from pump failure while the three other patients left the hospital with a permanent cardiac pacemaker. Follow-up observation 6-12 months later revealed 1:1 A-V conduction with persistence of the right BBBP in one patient and a 2:1 A-V block in two.

Group II (patients 5-7) had chronic left BBBP with left axis deviation in one patient and normal frontal plane axis in two. Two patients had prolonged H-V interval of 60-65 msec and one had an H-V interval of 46 msec. In patient 5 an intra-Hb conduction delay was illustrated by recording H and H' deflections from proximal and distal electrode catheter positions, respectively, with an H-H' interval of 20 msec. In this patient pacing from the proximal electrode position resulted in a QRS configuration of left BBBP identical to conducted supraventricular beats and a PI-V interval similar to the H-V interval. On the other hand, pacing from the distal electrode position normalized the BBBP with a PI-V interval equal to the H'-V interval. Normalization of the BBBP was also associated with a shift of the left axis deviation to a normal frontal plane axis. In patients 6 and 7, Hb pacing resulted in normalization of the left BBBP with only a slight change in the frontal plane axis and a PI-V interval which was 20 and 7 msec shorter than the H-V interval in patients 6 and 7, respectively.

Illustrative examples of groups I and II patients are shown in figures 6 and 7, respectively. Figure 6 shows Hb electrograms from patient 1 who had an old inferior wall infarction, a recent anterior infarction (pathologic Q waves in V2-V4) and an acute RBBBP. Panels A and B illustrate the proximal and distal Hb potentials (H and H', respectively). Hb pacing from the proximal electrode position gave rise to a right BBBP identical to conducted supraventricular beats and a PI-V equal to the H-V interval (not shown in the figure). Panel C illustrates distal Hb pacing. His bundle stimulation was selective since the atria were not simultaneously activated as seen in the right atrial electrogram (RA eg). Distal Hb stimulation resulted in disappearance of the right BBBP with decrease of the QRS duration from 0.14 to 0.08 sec. The record illustrates the disappearance of terminal QRS conduction delay of the right BBBP with no change in the initial QRS vectors including the pathologic Q wave of inferior wall infarction in lead aVF. The PI-V interval was 5 msec shorter than the H'-V interval. Slight advancement of the electrode catheter resulted in right bundle and/or right ventricular septal stimulation (fig. 6, panel D). The paced QRS showed a left BBBP with marked change of the initial QRS vectors.

Figure 7 illustrates Hb electrograms from patient 5 who had hypertensive cardiovascular disease and chronic left BBBP. Panels A, C and E were obtained from different electrode catheter positions and illustrate, respectively, the proximal Hb potential, distal Hb potential and the right bundle spike. Panels B, D and F illustrate pacing from the three different sites utilizing the same stimulus strength and polarity. Proximal Hb pacing in panel B gave rise to a QRS configuration identical to conducted supraventricular beats and a PI-V interval similar to the H-V interval. On the other hand, distal Hb pacing in panel D resulted in disappearance of the left BBBP with decrease of the QRS duration from 0.14 to 0.08 sec. The frontal plane QRS axis also changed from -35° during left BBBP to +15° during the normalized

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

**Figure 6.** His bundle recordings obtained from patient 1 to illustrate normalization of acute right bundle branch block pattern (BBBP) by distal His bundle pacing. The patient had an old inferior and a recent anterior wall infarction. Hb eg represents the His bundle electrogram obtained from a right sided electrode catheter while RA eg illustrates the high right atrial electrogram. Panels A and B illustrate recordings from, respectively, the proximal (H) and the distal (H') His bundle obtained from two different electrode positions. Panel C illustrates normalization of the right BBBP during distal His bundle pacing. Panel D shows right bundle and/or right ventricular septal stimulation resulting in a left BBBP.
QRS pattern. The PI-V interval of 45 msec was identical to the H'-V interval. Right bundle pacing in panel F was obtained from an electrode position few mm distal to that during distal Hb pacing. This resulted in paced beats with left BBBP similar to but not identical to conducted supraventricular beats. The difference was in the frontal plane QRS axis which measured +15° during right bundle pacing (compare lead 2 during both patterns).

Discussion

Our experimental observations strongly suggest that acute ischemic injury of the His bundle can result in asynchronous conduction delay and functional longitudinal dissociation giving rise to significant alteration in the sequence of ventricular activation including typical bundle branch block patterns. Utilizing the same experimental model we have previously reported other in vivo (reference 20-figures 9 and 10) and in vitro (reference 25-figure 9) evidence of functional longitudinal dissociation of conduction in the ischemic His bundle. Because of limitations of the recording technique, the clinical observations in this report can be only considered as suggestive of functional dissociation of conduction in the distal His bundle. Group I patients may represent the clinical correlate of the experimental observations. However, since in these patients normalization of the acute ischemic right BBBP was obtained by electrode catheter pacing from the right side of the heart the possibility of pacing the proximal right bundle cannot be excluded. In this case, the right BBBP may have been due to a conduction delay in the very proximal right bundle region rather than in the distal His bundle. However, the fact that in two patients an intra-Hb conduction delay was demonstrated and the PI-V interval during distal Hb pacing with normal QRS configuration was almost equal to the H'-V interval suggests a distal Hb pacing rather than a proximal right bundle pacing. Further,

in the two other patients with prolonged H-V intervals but no demonstrable intra-Hb conduction delay, the PI-V interval associated with a normal QRS configuration was 40–50 msec. This value is longer than would be expected from simple pacing of the proximal right bundle unless pacing was applied in an area with depressed conduction. In group II patients with chronic left BBBP, the possibility of normalizing the BBBP by pacing the proximal bundle branch rather than the distal Hb was excluded since pacing was applied through a right-sided electrode catheter. These patients represent a stronger evidence of longitudinal dissociation in the distal Hb. However, since direct stimulation of the ventricular septum cannot be excluded as was done in the canine experiments (fig. 5), the selectivity of distal Hb pacing24 cannot be reliably established.

Preliminary clinical observations remarkably similar to those in groups I and II have been reported recently and were explained along similar lines. Schuilenburg et al.39 normalized acute right BBBP in patients with acute anterior wall infarction by pacing from a right-sided electrode catheter. They suggested that the conduction delay was localized in the proximal part of the right bundle branch and that it has a short length. However, the possibility of a distal Hb lesion cannot be excluded in their cases. On the other hand Narula and Linhart37 reported normalization of chronic left BBBP by distal Hb pacing from a right-sided electrode catheter and suggested longitudinal dissociation in the distal Hb.

The concept of functional longitudinal dissociation of conduction in the His bundle is by no means new. It was initially suggested by Kaufman and Rotherberger in 1919 and later emphasized by the experiments of Condorelli6 and Sciacca and Sangiorgi6 in which traumatic lesions in the His bundle caused BBBP in ECG leads. Histologic studies of the His bundle of canine and human hearts by James and Sherf28 suggested an anatomic basis for the concept of
longitudinal dissociation of conduction in the His bundle. These studies showed the canine and human His bundle to be composed of longitudinally oriented Purkinje strands separated by collagen and joined by transverse crossover connections at various intervals along their longitudinal axes. Several other observations by Sherf and James based primarily on deductive analysis of the ECG suggested that functional longitudinal dissociation of conduction may be operative in the normal A-V junction.\textsuperscript{13, 14} This suggestion would seem to deemphasize the role of normal transverse interconnections in synchronizing impulse conduction along the longitudinal Purkinje strands. On the other hand, some recent in vitro studies have emphasized the functional significance of the transverse conducting pathways in the His bundle and hence questioned the possibility of functional longitudinal dissociation of conduction in the normal His bundle.\textsuperscript{8, 9} However, a recent in vitro study by Fabregas et al.\textsuperscript{11} suggested that tachycardia-dependent functional failure of the transverse conducting pathways could result in longitudinal dissociation of conduction that would explain a BBBP secondary to a localized lesion in the nonbranching portion of the His bundle.

The site of lesion in the His bundle that would result in significant alteration of the sequence of ventricular activation has also raised some controversy. Thus, while the studies of Watt and Pruitt\textsuperscript{7} suggested that a lesion in the branching portion of the His bundle is required to produce changes in the form of the QRS complex, the work of Fabregas et al.\textsuperscript{11} demonstrated that a BBBP can result from a lesion in the nonbranching portion of the His bundle.

The controversy over the functional significance of the transverse interconnections and the site of lesion in the His bundle that can result in BBBP is closely related. Thus, if the transverse interconnections are functionally operative in the normal His bundle, a localized lesion in the nonbranching portion of the bundle will not produce significant changes in the QRS configuration since conduction in the transverse pathways distal to the site of lesion will tend to mitigate the effect of the proximal asynchrony of conduction. On the other hand, if we postulate that conduction across the transverse interconnections suffers a more selective depression in the pathologic His bundle (whether the pathology is secondary to acute ischemic or chronic sclerodegenerative lesion), then longitudinal dissociation in the pathologic His bundle can have both functional and anatomic basis. This is schematically illustrated in figure 8 which depicts functional longitudinal dissociation in the ischemic His bundle. The figure suggests that acute ischemia results in non-homogeneous depression of conduction in the His bundle and proximal bundle branches. A larger area of slightly depressed conduction is represented by the dotted area while islands of severely depressed conduction are depicted by the hatched areas. The main area of severely depressed conduction is localized in the right side of the nonbranching portion of the His bundle. Impulse conduction (represented by straight arrows) proceeds along the left side of the His bundle but is blocked on the right side. If both the longitudinal and transverse pathways in the dotted area below the area of block in the His bundle had an equal albeit slightly depressed conduction velocity, then impulse propagation from the left side of the His to the right side distal to the area of block could still result in fairly synchronous conduction in the three major fascicles. The figure, however, illustrates that transverse conduction below the site of block in the His bundle is relatively more depressed (represented by an undulating line). This would have the effect of a faster conduction to the left bundle branch system relative to the right bundle resulting in a right BBBP in ECG leads. The diagram also illustrates that pacing from a plunge wire electrode inserted in the His bundle distal to the site of severely depressed conduction (E<sub>2</sub>) can result in synchronous activation of the bundle branch system as opposed to pacing from the proximal electrode (E<sub>1</sub>). The same could be achieved by pacing from the distal rather than the proximal pole of an electrode catheter.

In some experiments in the present study we have shown that a slight increase in the stimulus strength during distal Hb pacing can result in normalization of the BBBP (fig. 5). Close monitoring of the high septal electrode excluded the possibility of direct muscle activation at the higher stimulus strength. This observation probably can be explained by assuming pacing close to the distal border of the severely depressed zone in the His bundle (i.e., the hatched island in the His bundle in fig. 8). Minor increment in the stimulus strength can result in recruitment of fibers in the closely bordering less depressed zone and hence a faster and more synchronous conduction to the right bundle. This resembles, in part, the experience during A-V nodal stimulation.\textsuperscript{29} This observation helps to emphasize the functional nature of conduction delay and block in the ischemic His bundle. Thus a relatively slow activation wave front in the ischemic His bundle may succeed in conducting along one pathway but fail to conduct in another pathway due to only minor differences in refractoriness and/or excitability.

Discussion of possible clinical implications of the concept of BBBP secondary to functional longitudinal dissociation in the pathologic His bundle raises at least two pertinent matters: first, the ability to discern that a BBBP is due to asynchronous conduction in the His bundle rather than a more peripheral lesion in the bundle branch system; and second, the prognostic difference between a BBBP due to a
proximal versus a distal lesion. In answer to the first question, our experimental observations have consistently demonstrated the presence of significant intra-Hb conduction delay manifested as a split Hb potential and an H-V interval of 16–35 msec in experiments in which a BBBP was considered as secondary to the Hb lesion. Similarly, six of our seven patients had a prolonged H-V interval of 60–85 msec and in three of these an intra-His bundle conduction delay of 25–35 msec was demonstrated. With the known limitations of current recording techniques in demonstrating intra-Hb conduction delay in clinical cases, the possibility exists that in the other three patients in this series the H-V prolongation was in fact due to an intra-Hb delay rather than a delay in the bundle branch system. This is substantiated by a PI-V interval during distal Hb pacing with normal QRS configuration that was 20–35 msec shorter than the H-V interval. On the other hand, the possibility of failure to record a proximal Hb potential in patient 7 who had a normal H-V interval and only showed a slight decrease of the PI-V interval during distal Hb pacing with normal QRS configuration cannot be excluded. Our observations suggest that in the majority of patients with possible BBBP secondary to longitudinal dissociation in the Hb, a prolonged H-V and/or a split Hb potential could be demonstrated. Normalization of the BBBP in those patients with distal Hb pacing associated with significant shortening of the PI-V interval compared to the H-V interval would provide suggestive evidence of longitudinal dissociation in the His bundle.

The prognostic significance of BBBP secondary to an intra-Hb conduction delay in the experimental studies is primarily related to the presence of the Hb lesion. In this experimental model an acute ischemic intra-Hb conduction delay is frequently the harbinger of paroxysmal complete A-V block. However, the significance of similar lesions in the acute clinical situation (group I patients) or in the chronic clinical situation (group II patients) requires a close follow-up of a larger number of patients and comparison with a matched group of patients with BBBP not due to a His bundle lesion. Our clinical observations suggest that in patients with BBBP and a prolonged H-V interval and/or a split Hb potential, the possibility of longitudinal dissociation in the His bundle cannot be excluded. Although the prognostic significance of chronic BBBP and prolonged H-V interval is still controversial, it seems that in acute myocardial infarction a BBBP with prolonged H-V interval carries a higher risk of A-V block. It is possible that the prolonged H-V interval in some of these patients may reflect an intra-Hb conduction delay and that the BBBP may have been due to functional longitudinal dissociation in the ischemic His bundle.

There is another clinical situation in which a BBBP due to a His bundle lesion may carry a higher risk of complete A-V block. These are cases of postoperative left anterior hemiblock and right bundle branch block following repair of tetralogy of Fallot. Two distinct groups of patients have been recognized: one group in which the ECG pattern is secondary to a Hb lesion and a second group in which the pattern is caused by lesions in the peripheral conduction system. The first group of patients has a higher risk of complete A-V block. Also, in these patients, a prolonged H-V interval was suggested as a useful index to identify those at risk.

In summary, the present study has presented suggestive experimental and clinical evidence of BBBP secondary to functional longitudinal dissociation in the pathologic His bundle. The diagnostic and prognostic implications of this concept require further observations.

References

The Noninvasive Diagnosis of Right Ventricular Infarction


SUMMARY We evaluated scintigraphy and echocardiography for the diagnosis of right ventricular (RV) infarction. Of 26 patients with acute transmural myocardial infarction (MI), six with inferior MI had abnormal radionuclide uptake localized to the RV free wall on infarct scintigraphy or segmental akinesis of the RV free wall on gated radioangiography or both. These six patients with RV involvement (group I) were compared with the remaining nine with inferior MI (group II) and 11 with anterior MI (group III). RV/LV area ratios determined radioangiographically were significantly greater in group I than group II in diastole and systole. Echocardiographic RV end-diastolic dimension and RV/LV end-diastolic dimension ratio were significantly greater in group I than group II. Mean RV filling pressure was significantly greater and RV stroke work index was significantly lower in group I than in group II. Predominant RV involvement in inferior MI may occur commonly. Anatomic and functional evidence of this diagnosis can be obtained noninvasively.

We sought a direct noninvasive indicator of RV infarction. To this end we evaluated the techniques of radionuclide scintigraphy and echocardiography for their ability to diagnose RV infarction and dysfunction.

Materials and Methods

Studies were performed on 26 consecutive patients admitted to our Coronary Care Unit with acute transmural myocardial infarction. The diagnosis of myocardial infarction was based on the clinical history, serial electrocardiographic abnormalities with the development of Q waves > 0.04 sec, and serum CPK-MB enzyme elevation. Patients were classified as having anterior (Q waves in leads I, aVL, V₅, V₆) or inferior (Q waves in leads II, III, aVF) infarction. Historical, electrocardiographic and enzymatic evidence of prior infarction was similarly sought. All patients were examined clinically for evidence of valvular heart disease, left to right shunting, chronic lung disease or acute pulmonary embolism. Signs of elevated systemic and pulmonary venous pressure were sought on physical examination and chest X-ray. The occurrence of arrhythmias, hypotension or other complications was noted in all cases. Informed consent was obtained and scintigrams, echocardiograms and hemodynamic measurements were carried out within 72 hours of admission. Myocardial infarct scintigraphy was performed 24–48 hours after admission, following the intravenous administration of 15 mCi of 99m Technetium (stannous) pyrophosphate (Tc-PYP) manufactured according to the method of Huberty and coworkers. Anterior, 45° left anterior oblique and left lateral projections, each taken to 300,000 counts, were obtained at least...
Normalization of bundle branch block patterns by distal His bundle pacing. Clinical and experimental evidence of longitudinal dissociation in the pathologic his bundle.
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