Intracardiac Conduction Intervals in Children with Congenital Heart Disease

Comparison of His Bundle Studies in 41 Normal Children and 307 Patients with Congenital Cardiac Defects

Aaron R. Levin, M.D., Jacob I. Haft, M.D., Mary Allen Engle, M.D., Kathryn H. Ehlers, M.D., and Arthur A. Klein, M.D.

SUMMARY His bundle electrograms were recorded in 348 patients aged 2 months to 24 years following routine diagnostic cardiac catheterization. Among 41 children found to be free of anatomic or hemodynamic abnormality the following mean values were obtained: P-H interval, 93.4 ± 15.3 msec; P-A time, 21.2 ± 7.1 msec; A-H interval, 72.2 ± 15.9 msec; and H-V interval, 39.8 ± 5.2 msec. The remaining 307 patients were analyzed by diagnosis of congenital heart disease. The mean P-H interval was found to be significantly increased in both ostium primum and secundum atrial septal defect (ASD) with A-H prolongation in primum ASD and P-A prolongation in secundum ASD. The mean H-V interval was significantly prolonged in ostium primum ASD and in patients with severe aortic stenosis, aortic insufficiency, and mitral regurgitation. Grouping of the patients physiologically revealed that patients with moderate-to-severe right ventricular volume overload had P-H prolongation, and patients with severe left ventricular volume or pressure overload had H-V prolongation. The clinical implications of these findings are discussed.

DURING THE PAST FIVE YEARS a number of studies of His bundle electrograms in children with congenital heart disease have been reported. In most of the investigations the number of patients included have been relatively small, and when further grouped according to abnormality, became even smaller. Because of their inability to separate out the incidence of electrophysiologic abnormalities associated with each congenital defect, most investigators have grouped all their patients together. These studies also suggested that parameters such as P-H, A-H, and H-V intervals vary directly with age and that in most defects the intervals are essentially normal. Waldo et al. and Anderson and co-workers did find a prolonged P-H interval in patients with atrial septal defect, both ostium primum and secundum. Over the past four years, His bundle electrograms have been performed as a routine part of evaluation of the cardiac status in children who come to cardiac catheterization at this medical center. The purpose of this report is to present our findings in 348 patients who were in normal sinus rhythm at the time of the study.

Method and Materials

All children undergoing routine cardiac catheterization at The New York Hospital-Cornell Medical Center from July 1972 to May 1976 were subjected to His bundle electrophography following completion of the diagnostic studies, after parental informed consent had been obtained, and if they appeared able to tolerate an additional 10 min procedure. Three hundred forty-eight patients with technically adequate records were included in this study.

His bundle electrograms were performed according to a modification of the technique of Scherlag et al. A quadrupolar electrode catheter with interpolar intervals of 1 cm was introduced percutaneously through a #6 Desilets sheath into the right femoral vein. The tip of the catheter was advanced under fluoroscopic control until the catheter just entered the right ventricle and was manipulated until an adequate recording of the His bundle electrogram was obtained on at least two of the electrograms recorded from adjacent pairs of electrodes. The recordings were made at a paper speed of 100 mm/sec using an Electronics-for-Medicine oscillographic photographic recorder with frequency limits set at 40-500 cycles/sec. The His bundle electrograms were recorded simultaneously with two leads of the surface ECG, usually leads II and aVR. The electrogram with earliest onset of the His depolarization was used for all measurements. Measurements were made in the following manner (fig. 1):

P-H interval: from the onset of the earliest deflection of the P wave of either of the surface leads to the earliest onset of the deflection of the His spike.

A-H interval: from the first major rapid deflection of the atrial depolarization as noted on the His bundle electrogram that recorded the earliest His spike to the onset of the His deflection.

H-V interval: from the onset of the earliest His bundle spike to the onset of ventricular depolarization, whether occurring first on any of the intracardiac electrograms or on the surface ECG.

Ten consecutive beats were analyzed and the mean value for each of the parameters for each patient were calculated. From these measurements the mean difference between the P-H and A-H intervals was calculated. This will be referred to as the P-A time. The mean R-R interval was calculated for the same 10 beats analyzed.

None of the patients was in congestive heart failure and none was receiving any cardiac medications, i.e., digitalis, anti-arrhythmic, diuretic, or anti-hypertensive agents during the period of study or in the preceding two weeks. All of the patients, both normal and abnormal, were in normal sinus rhythm at the time of the study. The routine preparation for
catheterization consisted of Demerol (1 mg/kg), Phenergan (0.25 mg/kg), and Thorazine (0.25 mg/kg) administered parenterally one hour prior to the catheterization procedure, and usually three hours prior to His bundle recordings.

Data obtained from His bundle electrograms were correlated with the diagnostic findings at cardiac catheterization. Ages varied from 2 months to 24 years; the mean age was 8.2 years and median age was 8.0 years. The 41 normal subjects included patients with murmurs thought to be organic, but found not to correlate with functional or anatomic abnormalities when studied with both right and left heart catheterizations, multiple oxygen content determinations, and hydrogen studies.14 None of the normal children had a significant gradient across any valve or outflow tract, nor did any have evidence of intracardiac or extracardiac shunting.

In the 307 patients with heart disease, the His bundle electrogram data was analyzed with respect to the specific diagnoses. The patients were also grouped according to the physiological strain on the heart, and the His bundle electrogram measurements of the groups were compared. Group I was composed of those patients with right ventricular volume overload, including ostium primum and ostium secundum atrial septal defect (ASD) and partial anomalous pulmonary venous return (PAPVR). Group II was composed of patients with left ventricular (LV) volume overload (ventricular septal defect, patent ductus arteriosus, mitral regurgitation, and aortic regurgitation). Patients with right ventricular (RV) pressure overload lesions (pulmonary stenosis and tetralogy of Fallot), made up group III and those with LV pressure overload, namely valvular aortic stenosis (AS), idiopathic hypertrophic subaortic stenosis (IHSS) and coarctation of the aorta, group IV.

For the purpose of data analysis, each of these physiological groups, as well as each specific diagnostic category, was further subdivided into hemodynamic divisions. In group I with RV volume overload, analyses were undertaken on the basis of shunt size within each diagnostic category and in the group as a whole; arbitrary limits utilized in these patients were pulmonary-to-systemic flow ratios (Qp/Qs) of less than 2.0, 2.0–3.0, and greater than 3.0. Patients in group II with ventricular septal defect (VSD) and patent ductus arteriosus (PDA) were similarly subdivided; in addition, analyses were also undertaken following categorization by the magnitude of the right ventricular systolic pressure elevation (<50 mm Hg, 50–70 mm Hg, and >70 mm Hg). Patients in group II with aortic regurgitation (AI) and mitral regurgitation (MI) were ranked according to an angiographic estimate of the degree of insufficiency as greater than +++ vs less than +++. Patients in group III with RV pressure load were analyzed in a similar fashion: those with pulmonic stenosis (PS) were categorized according to the magnitude of RV pressure, while those with tetralogy of Fallot (TF) were not subdivided further since all had RV pressure at a systemic level. In addition, values of patients exhibiting PS with right ventricular pressures in excess of 70 mm Hg were grouped with TF and compared to normal values. All group IV patients as well as those in individual diagnostic groups were analyzed according to the magnitude of gradient across the aortic valve in patients with aortic stenosis (AS) or peak pressure differences between the transverse and descending aortic arches in patients with coarctation of the aorta (<50 mm Hg, 50–70

**Figure 1.** Method of measuring the His bundle electrographic time intervals. Three His electrographic leads were recorded simultaneously with two surface electrocardiographic tracings (usually lead II and aVL) at a paper speed of 100 mm/sec.
mm Hg, and >70 mm Hg). Additional analyses were undertaken based on the height of LV pressure (whether the pressure was <150 mm Hg, 150-200 mm Hg, <175 mm Hg, >175 mm Hg, and >200 mm Hg).

In addition, patients with complete d-transposition of the great arteries, congenital cardiomyopathy, and mitral stenosis were studied. Data for these patients were analyzed separately and were not included in the above four functional groupings. Patients who had undergone surgical repair of ASD, VSD, and TF and surgical relief of valvular PS were not included in the physiologic categories above; but where data on preoperative hemodynamics were available, the groups were compared with the preoperative hemodynamic abnormalities.

The data were calculated using a Monroe Computer and the mean, standard deviation, and standard error of the mean for all parameters were determined for each diagnosis and for each physiologic group. All results according to individual diagnoses were compared to the normal values and to each other, and postoperative cases were compared with preoperative cases with the same diagnosis. Comparisons were also made according to physiologic groups. Tests of significance were determined using the Student’s t-test for independent variables.12

After preliminary analysis, the findings for each of the His bundle electrogram parameters were plotted against age and R-R interval, and correlation coefficients were determined using the standard Monroe program for each diagnosis, each group, and for the entire patient group. Regression equations were determined for each parameter. Correlation coefficients were tested for the hypothesis that \( r \neq 0.\)

Results

The distribution of diagnoses and number of patients with each diagnosis and in each group are presented in Table 1.

P-H Interval

The normal P-H interval (tables 2 and 3) was 93.4 ± 15.3 msec (mean ± standard deviation). The mean value for group I (table 2) differed significantly from normal (104.5 ± 15.0 msec, \( P < 0.01 \)). None of the means of the other physiologic groups were significantly different from the normal values (tables 2 and 3). Patients with ASD (both ostium primum and secundum) had significantly greater P-H intervals than the normal subjects (table 2 and fig. 2). The mean interval in those with ostium primum defect was 108.7 ± 10.9 msec \( (P < 0.05) \) and in ostium secundum defects was 103.1 ± 16.3 msec \( (P < 0.05) \). In both the primum and secundum ASDs the shunt magnitude was not significantly related to the abnormality noted in the P-H interval. Those with postoperative ostium primum ASD (table 4) also had significantly prolonged P-H intervals (164.0 ± 72.7 msec, \( P < 0.001 \)). In patients with VSD and TF, although the preoperative P-H means were not significantly different from normal (tables 2 and 3), postoperative P-H intervals were prolonged when compared to normal (110.4 ± 24.7, \( P < 0.01 \), and 102.3 ± 21.2, \( P < 0.05 \)) respectively (table 4). Postoperative subjects with VSD and with TF had a significantly longer mean P-H interval than did preoperative patients with these disorders (110.4 ± 24.7 msec vs 96.1 ± 15.4 msec, \( P < 0.05 \) [tables 2 and 4] and 102.3 ± 21.2 msec vs 93.6 ± 14.1 msec, \( P < 0.05 \) [tables 3 and 4], respectively). Neither the size of the shunt nor the RV pressure preoperatively correlated with the presence or absence of abnormality in the P-H interval in these postoperative patients.

P-A Time

The calculated mean P-A time was found to be statistically different from normal only in group I with RV volume overload (tables 2 and 3). The P-A time was 25.3 ± 9.5 msec compared to 21.2 ± 7.1 msec in normal patients \( (P < 0.05) \). In this group it was the ostium secundum ASD patients who were abnormal, 25.7 ± 9.2 msec \( (P < 0.05) \) (table 2, fig. 2) whereas the P-A time of the ostium primum patients was not significantly prolonged. Those with secundum ASD who had Qp/Qs > 2.0 had abnormal findings whereas those with lesser shunts had values closer to the normal mean value. Postoperatively, secundum ASD patients had normal P-A times (table 4). Two other diagnostic categories were found to correlate with a prolonged P-A time, namely the small group of PS patients with RV pressure greater than 70 mm Hg (table 3), and the postoperative VSD patients (table 4).

A-H Interval

The A-H interval in normal children was 72.2 ± 15.9 msec (tables 2 and 3). Among the physiologic groupings,
<table>
<thead>
<tr>
<th>Group</th>
<th>RV Volume Overload</th>
<th>LV Volume Overload</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>41</td>
<td>66</td>
</tr>
<tr>
<td>Age (yr)</td>
<td>10.1 ± 4.2</td>
<td>7.3 ± 4.4</td>
</tr>
<tr>
<td>R-R (msec)</td>
<td>629.1 ± 128.6</td>
<td>605.8 ± 135.5</td>
</tr>
<tr>
<td>P-H (msec)</td>
<td>93.4 ± 15.3</td>
<td>94.6 ± 15.3</td>
</tr>
<tr>
<td>P-A (msec)</td>
<td>21.2 ± 7.1</td>
<td>22.7 ± 9.8</td>
</tr>
<tr>
<td>A-H (msec)</td>
<td>72.2 ± 15.9</td>
<td>73.2 ± 13.5</td>
</tr>
<tr>
<td>H-V (msec)</td>
<td>39.8 ± 5.2</td>
<td>41.2 ± 7.2</td>
</tr>
</tbody>
</table>

### Group I: RV Volume Overload

- **Normal**
  - Qp/Qs <2.0: 16
  - Qp/Qs 2.0-3.0: 16
  - Qp/Qs >3.0: 9

- **ASD Primum**
  - Qp/Qs <2.0: 6
  - Qp/Qs 2.0-3.0: 2
  - Qp/Qs >3.0: 2

- **ASD Secundum**
  - Qp/Qs <2.0: 8
  - Qp/Qs 2.0-3.0: 14
  - Qp/Qs >3.0: 7

- **PAPVR**
  - 2

### Group II: LV Volume Overload

- **VSD**
  - Qp/Qs <2.0: 36
  - Qp/Qs 2.0-3.0: 4
  - Qp/Qs >3.0: 3

- **RV mm Hg <50**
  - 30

- **RV mm Hg >70**
  - 5

- **PDA**
  - Qp/Qs <2.0: 11
  - Qp/Qs 2.0-3.0: 3

- **RV mm Hg <50**
  - 9

- **RV mm Hg >70**
  - 1

- **AI**
  - >++++: 6
  - ++++: 2
  - +++: 4

- **MI**
  - >++++: 6
  - ++++: 3
  - +++: 3

**Asterisks indicate significant difference from normal:**
- *P < 0.05; **P < 0.01; ***P < 0.001.

**Abbreviations:**
- AI = aortic insufficiency; ASD = atrial septal defect; RV = right ventricle; LV = left ventricle; PAPVR = partial anomalous pulmonary venous return; VSD = ventricular septal defect; Qp = pulmonary flow; Qs = systemic flow; PDA = patent ductus arteriosus; s.d. = standard deviation; N = number of patients; MI = mitral insufficiency.
there was a significant difference from normal noted in group I with RV volume overload (table 2), with a mean value of 82.8 ± 22.7 (P < 0.05). This increase in A-H interval for the entire group was due to prolongation of the A-H among those with ostium primum ASD (table 2 and fig. 2), for they had significantly prolonged A-H intervals (96.3 ± 37.8 msec, P < 0.01), whereas those with ostium secundum defects did not significantly differ from normal (77.3 ± 13.0 msec). The degree of A-H interval abnormality tended to vary with the magnitude of the shunt, but this correlation was not significant. Postoperatively, the patients with repaired ostium primum ASD (table 4) maintained or further lengthened the prolonged A-H interval (146.8 ± 78.5, P < 0.001). In contrast, pre- and postoperative patients with ostium secundum ASD were normal on both occasions. No other diagnostic categories nor physiologic groups showed significant differences in A-H interval from the normal.

H-V Interval

The mean normal H-V interval was 39.8 ± 5.2 msec (tables 2 and 3). Those with ostium primum ASD (table 2) had a mean H-V interval that was significantly greater than normal, 47.1 ± 14.4 msec (P < 0.05). The H-V interval did not correlate with the magnitude of the shunt in primum ASD patients. Patients with marked hemodynamic effects on the LV (tables 2 and 4 and fig. 3) were also found to have significant prolongation of the H-V interval.
In group 4, with LV pressure overload (table 3) those with pressures of greater than 175 mm Hg had H-V intervals of 44.5 ± 9.4 msec (P < 0.05) and those with pressures greater than 200 mm Hg had H-V intervals of 56.0 ± 4.5 (P < 0.001), while patients exhibiting a pressure gradient greater than 70 mm Hg had H-V intervals of 44.8 ± 9.1 (P < 0.05). The abnormal H-V values in this group were concentrated among patients with valvular AS. More nearly normal values were found in patients with coarctation of the aorta, possibly because none of the latter achieved high LV pressures nor gradients. The group II patients with LV volume overload due to AI or MI (table 2) both had significantly prolonged H-V intervals, and this elevated value for the group was found to be due to abnormalities in those with the more hemodynamically severe AI or MI (fig. 3). In contrast, patients with LV volume overload due to PDA or VSD had normal H-V intervals, as did patients with less severe MI or AI.

Postoperatively, both the repaired primum and secundum ASD patients had H-V intervals significantly different from the normal (table 4); here, the H-V intervals were significantly shorter than normal in the patients with repaired ostium primum ASD who preoperatively had had Qp/Qs > 2.0 and those with ostium secundum ASD who had had Qp/Qs < 2.0.

Correlation with Age and Rate

Analysis of the His bundle electrogram intervals with regard to age was made for each diagnosis and for each physiologic group. No significant correlation was found. Since the intervals did not deviate from normal in the majority of the groups and diagnoses studied, all of the patients were analyzed together with respect to age and R-R interval. P-H, A-H, P-A, and H-V intervals varied with age and P-A time varied with heart rate, but the correlation coefficient was less than 0.23 in all instances and therefore of little importance. There was an association between age and R-R interval with r = 0.50, confirming the well-recognized fact that heart rate falls with growth from infancy through adolescence. The H-V interval varied with age (r = 0.23), but not significantly with rate.

Discussion

The mean P-H interval was increased in both the ostium primum and ostium secundum ASD patients (table 2 and fig. 2). However, the cause for the P-H prolongation was different in these two groups. Among patients with ostium primum ASD the A-H interval was prolonged whereas the P-A time was normal, suggesting that the P-H interval prolongation was due to delay in conduction from the low right atrium across the A-V node to the bundle of His. In contrast, P-H prolongation noted in patients with secundum ASD (table 2, fig. 2) was found to be associated with a prolonged P-A time and a normal A-H interval suggesting that P-H prolongation in secundum ASD patients was due to intra-atrial conduction delay rather than to slowing of conduction across the A-V node. Of interest was the fact that in ostium primum ASD patients, the A-H interval prolongation did not correlate significantly with the magnitude of the left-to-right shunt and postoperatively these patients maintained the A-H abnormality (table 4), whereas among the patients with ostium secundum ASD (table 2 and fig. 2), the P-A time abnormality correlated with the shunt magnitude and only those with Qp/Qs > 2.0 had PA time prolongation. Postoperatively, those patients who had secundum ASD repair (table 4) had normal P-A times and probably as a result, normal P-H intervals.

These findings suggest that the P-R prolongation noted on the surface electrocardiogram in ostium primum ASD patients is due to functional abnormality in conduction across the A-V node area, an abnormality that persists after the repair of the defect. It is likely that because the ostium...
Table 4. Mean Values (± 1 sd) of His Bundle Time Intervals for Miscellaneous and Postoperative Patients

<table>
<thead>
<tr>
<th></th>
<th>N</th>
<th>Age (yr)</th>
<th>R-R (msec)</th>
<th>P-H (msec)</th>
<th>P-A (msec)</th>
<th>A-H (msec)</th>
<th>H-V (msec)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>41</td>
<td>10.1 ± 4.2</td>
<td>629.1 ± 128.6</td>
<td>93.4 ± 15.3</td>
<td>21.2 ± 7.1</td>
<td>72.2 ± 15.9</td>
<td>39.8 ± 5.2</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Transposition</td>
<td>6</td>
<td>2.6 ± 1.6</td>
<td>523.6 ± 133.9</td>
<td>98.0 ± 18.8</td>
<td>27.5 ± 14.3</td>
<td>70.4 ± 5.8</td>
<td>38.5 ± 8.4</td>
</tr>
<tr>
<td>Cardiomyopathy</td>
<td>3</td>
<td>9.6 ± 4.0</td>
<td>621.6 ± 207.0</td>
<td>97.8 ± 9.8</td>
<td>21.0 ± 4.5</td>
<td>76.6 ± 6.6</td>
<td>42.3 ± 7.1</td>
</tr>
<tr>
<td>Mitral Stenosis</td>
<td>2</td>
<td>13.5 ± 0.7</td>
<td>550.0 ± 14.1</td>
<td>113.5 ± 23.3</td>
<td>22.5 ± 6.3</td>
<td>91.0 ± 16.9</td>
<td>44.5 ± 7.7</td>
</tr>
<tr>
<td>Postoperative Group</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>ASD Primum</td>
<td>7</td>
<td>8.2 ± 5.4</td>
<td>585.0 ± 116.5</td>
<td>164.0 ± 72.9***</td>
<td>26.5 ± 10.5</td>
<td>148.8 ± 78.5***</td>
<td>35.0 ± 8.6*</td>
</tr>
<tr>
<td>Pre-Op Qp/Qs &lt;2.0</td>
<td>2</td>
<td>3.5 ± 0.7</td>
<td>520.0 ± 42.4</td>
<td>153.5 ± 9.1*</td>
<td>26.3 ± 12.4</td>
<td>135.0 ± 8.4***</td>
<td>36.0 ± 4.2</td>
</tr>
<tr>
<td>2.0-3.0</td>
<td>2</td>
<td>6.0 ± 0.0</td>
<td>530.0 ± 9.0</td>
<td>115.0 ± 14.1</td>
<td>27.0 ± 14.4</td>
<td>96.4 ± 7.7</td>
<td>72.0 ± 5.3*</td>
</tr>
<tr>
<td>&gt;3.0</td>
<td>1</td>
<td>15.0 ± 0.0</td>
<td>580.0 ± 32.0</td>
<td>83.0 ± 0.0</td>
<td>30.0 ± 0.0</td>
<td>190.0 ± 0.0</td>
<td>30.0 ± 0.0</td>
</tr>
<tr>
<td>ASD Secundum</td>
<td>12</td>
<td>9.1 ± 3.8</td>
<td>697.9 ± 146.0</td>
<td>96.3 ± 23.2</td>
<td>22.5 ± 12.5</td>
<td>74.4 ± 18.4</td>
<td>36.8 ± 7.6</td>
</tr>
<tr>
<td>Pre-Op Qp/Qs &lt;2.0</td>
<td>6</td>
<td>7.6 ± 2.3</td>
<td>645.8 ± 105.3</td>
<td>90.3 ± 19.8</td>
<td>22.5 ± 12.1</td>
<td>67.8 ± 10.8</td>
<td>31.6 ± 5.4***</td>
</tr>
<tr>
<td>2.0-3.0</td>
<td>4</td>
<td>8.5 ± 3.0</td>
<td>655.6 ± 130.2</td>
<td>109.5 ± 31.1</td>
<td>23.5 ± 15.8</td>
<td>86.0 ± 27.7</td>
<td>39.2 ± 4.5</td>
</tr>
<tr>
<td>&gt;3.0</td>
<td>2</td>
<td>14.5 ± 6.3</td>
<td>880.0 ± 212.1*</td>
<td>91.5 ± 9.1</td>
<td>29.5 ± 14.8</td>
<td>71.0 ± 5.6</td>
<td>47.5 ± 3.5</td>
</tr>
<tr>
<td>VSD</td>
<td>19</td>
<td>8.3 ± 3.8</td>
<td>676.8 ± 136.5</td>
<td>110.4 ± 24.7**</td>
<td>29.0 ± 14.5**</td>
<td>81.4 ± 20.9</td>
<td>43.1 ± 8.4</td>
</tr>
<tr>
<td>Pre-Op Qp/Qs &lt;2.0</td>
<td>7</td>
<td>8.0 ± 3.2</td>
<td>664.2 ± 98.7</td>
<td>110.8 ± 13.9**</td>
<td>27.1 ± 17.1</td>
<td>83.7 ± 19.1</td>
<td>41.8 ± 7.1</td>
</tr>
<tr>
<td>2.0-3.0</td>
<td>8</td>
<td>8.3 ± 4.1</td>
<td>626.2 ± 99.2</td>
<td>110.3 ± 33.2*</td>
<td>29.3 ± 14.0*</td>
<td>81.4 ± 23.3</td>
<td>43.1 ± 10.3</td>
</tr>
<tr>
<td>&gt;3.0</td>
<td>1</td>
<td>5.0 ± 0.0</td>
<td>540.0 ± 73.0</td>
<td></td>
<td>19.0 ± 0.0</td>
<td>54.0 ± 0.0</td>
<td>39.0 ± 0.0</td>
</tr>
<tr>
<td>TF</td>
<td>28</td>
<td>8.5 ± 4.3</td>
<td>645.1 ± 118.8</td>
<td>102.3 ± 21.2*</td>
<td>22.3 ± 10.9</td>
<td>79.9 ± 19.7</td>
<td>42.6 ± 7.8</td>
</tr>
<tr>
<td>PS</td>
<td>2</td>
<td>3.0 ± 1.4</td>
<td>545.0 ± 7.0</td>
<td>94.5 ± 36.0</td>
<td>22.0 ± 9.8</td>
<td>72.5 ± 26.1</td>
<td>39.5 ± 13.4</td>
</tr>
</tbody>
</table>

Asterisks indicate significant difference from normal: *P < 0.05; **P < 0.01; ***P < 0.001.
Abbreviations: Same as in table 2 and 3; Pre-Op = Pre-operative.
prolongation of the H-V interval seen in this group of patients. Postoperatively, ostium primum lesions were no longer found to have prolonged H-V intervals although the rSR' pattern and marked left axis deviation continued to be present. This suggests that whatever the fundamental etiology of the rSR' and left axis deviation, they are permanent and probably anatomical defects rather than outgrowths of the hemodynamics of the situation, and thus not reversible when the hemodynamics is corrected. The prolonged H-V interval, however, which may reflect conduction delay in the posterior radiations, was brought into the normal range in those who had their shunt repaired, suggesting that flow across the defect and across the closely associated His bundle functionally affected the fibers destined to form the posterior radiations of the left bundle branch, but did not cause them permanent or irreversible damage.

The H-V interval was also prolonged in two other small groups of patients, both of which had marked hemodynamic insult to the LV (fig. 3). These were the group of patients exhibiting LV pressure overload (table 3) with pressures >175 mm Hg and outflow gradients >70 mm Hg, and those patients with LV volume overload having AI or MI of severe degree (table 2). The reason for this is not completely clear, but it is conceivable that chronic endocardial trauma due to LV volume or pressure overload may compromise conduction in the left bundle branch (which lies immediately beneath in the subendocardium). Depolarization of the left septum, which is normally the earliest part of the LV to be activated, may be delayed and cause a prolongation of the H-V interval.

The observation that the H-V interval was significantly shorter than normal in patients with ostium primum or ostium secundum ASD that were repaired (table 4) is curious and we have no explanation for these findings. Ostium primum ASD patients may have a more facilitated type of conduction made necessary by the trauma to the His bundle caused by the shunt. When the trauma is removed this facilitated conductive ability is unmasked and persists, as reflected in a significantly shorter H-V interval. In patients with ostium secundum ASD a similar reasoning may apply, but the less severe trauma to the His bundle, because of the position of the defect, allows this facilitation of conduction through the His bundle to maintain normalcy of the H-V interval when the shunt was present. With repair of the hemodynamic defect the facilitated conduction, now producing a short H-V, is unmasked. This theory is purely speculative but may explain our observed findings.

References

2. Brodsky SJ, Mirowski M, Krovetz LJ, Rowe RD: Recordings of His
Differentiation of Transiently Ischemic from Infarcted Myocardium by Serial Imaging after a Single Dose of Thallium-201

Gerald M. Pohost, M.D., Leonard M. Zir, M.D., Richard H. Moore, Kenneth A. McKusick, M.D., Timothy E. Guiney, M.D., and George A. Beller, M.D.

SUMMARY Myocardial thallium-201 uptake and regional blood flow by the microsphere technique were determined in anesthetized dogs undergoing either 20 min of coronary occlusion and 100 min of reperfusion (N = 10) or 120 min of occlusion (N = 4). In both groups, thallium-201 was injected intravenously after 10 min of occlusion. In transiently occluded dogs, regional flow at the time of thallium-201 administration was reduced to 8 ± 3% of normal flow in endocardial layers of the central ischemic zone. After 100 min of reperfusion, flow values were not significantly different from normal. Thallium-201 activity after reperfusion rose to 56 ± 5% of normal, demonstrating that redistribution of the radionuclide occurred during the reflow period. In animals with persistent occlusion, there was a significant relationship between thallium-201 uptake and flow (r = 0.95) and no evidence of redistribution of thallium-201 during the two-hour occlusion period. In another five dogs receiving thallium-201, serial gamma camera images obtained during reperfusion showed increasing uptake of the tracer in apical defects which returned to normal by 4 hours of reflow.

Thirteen patients with stable angina received 2 mCi of thallium-201 intravenously at peak exercise, and multiple gamma camera images obtained serially. All demonstrated zones of diminished thallium-201 uptake 10 min after exercise. Defects which partially or completely disappeared within 1–6 hours postexercise corresponded to areas supplied by coronary arteries with significant stenoses. Persistent defects were present in regions of old myocardial infarction. Six additional patients with acute myocardial infarction demonstrated thallium-201 myocardial defects which showed no significant change over 6 hours.

Thus, redistribution of thallium-201 into ischemic myocardium was demonstrated during transient coronary occlusion in dogs and after exercise stress in man. Sequential imaging after a single dose of thallium-201 at the time of exercise may provide a means for distinguishing between transient perfusion abnormalities or ischemia and myocardial infarction or scar.

IN RECENT YEARS the technique of imaging the heart using radiopharmaceutical agents for the assessment of regional myocardial perfusion, and for the detection and localization of myocardial infarcts has improved considerably.1-4 The existence of active transport mechanisms for concentrating monovalent cations in normal myocardium has led to the use of radioisotopes of potassium, rubidium, cesium and thallium for myocardial imaging. Uptake of these agents in myocardium is related to nutrient blood flow, and structural and functional integrity of the myocardial cell membrane. Areas of ischemic or infarcted myocardium appear as regions of diminished radioactivity in gamma camera images. Thallium-201, a transitional metallic element, has recently been introduced as an agent for myocardial imaging with a gamma camera.5 It appears to concentrate in myocardium in a manner similar to potassium or rubidium but to a somewhat greater degree.6 Imaging after thallium-201 administration has also been employed for delineation of myocardial defects induced by exercise stress and these defects have correlated with regions of myocardium supplied by coronary arteries with significant obstructive lesions.7 Because of the rather long physical half-life of thallium-201 (73.5 hours) a rest imaging study at least 3 days later using another tracer injection is necessary to differentiate transient perfusion abnormalities or ischemia from old infarction.

In the present study, a new method for differentiating transient perfusion abnormalities or ischemia from infarct-

A R Levin, J I Haft, M A Engle, K H Ehlers and A A Klein

_Circulation_. 1977;55:286-294
doi: 10.1161/01.CIR.55.2.286

_Circulation_ is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1977 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/55/2/286

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/