H-V Intervals in Left Bundle-Branch Block

Clinical and Electrocardiographic Correlations

By Kenneth M. Rosen, M.D., Ali Ehsani, M.D.,
and Shahbudin H. Rahimtoola, M.B., M.R.C.P.E.

SUMMARY

H-V interval in left bundle-branch block (LBBB) reflects conduction time in the His bundle and right bundle branch. H-V intervals were measured in 57 patients with LBBB, allowing definition of three groups of patients. Group A consisted of 14 patients with normal H-V (less than 50 msec), group B consisted of 21 patients with intermediate H-V (50–60 msec), and group C consisted of 22 patients with prolonged H-V (greater than 60 msec).

Arteriosclerotic heart disease (ASHD) was most frequent in group A (P < 0.02), while hypertension was most frequent in group C (P < 0.15). Mean P-R interval ± SEM was 0.172 ± 0.013 sec in group A, 0.185 ± 0.007 sec in group B, and 0.225 ± 0.014 sec in group C (P < 0.05). Mean QRS duration was 0.138 ± 0.004 sec in group A, 0.144 ± 0.004 sec in group B, and 0.157 ± 0.003 sec in group C (P < 0.01). Mean frontal axis was −8° ± 12° in group A, −16° ± 12° in group B, and −28° ± 8° in group C (ns).

The frequent association of LBBB, normal H-V, and ASHD suggested the presence of isolated ischemic disease of the left bundle branch. In contrast, the frequent association of LBBB, prolonged H-V, and absence of ASHD was suggestive of sclerodegenerative bilateral bundle-branch disease. In a patient with LBBB, the occurrence of both first-degree A-V block and a QRS duration of 0.16 sec or greater strongly suggested the likelihood of H-V prolongation.

Additional Indexing Words:
A-V block
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RECORDING of His bundle electrograms allows definition of the electrocardiographically silent H-V interval, from His bundle depolarization to the onset of ventricular activation.¹ ² In left bundle-branch block, this interval reflects conduction time in the His bundle and right bundle-branch system. Prolongation of H-V interval in left bundle-branch block reflects delays in the His bundle and/or the right bundle branch and may be indicative of bilateral bundle-branch disease.² ⁶

Previous workers have shown that H-V intervals are frequently prolonged in left bundle-branch block.⁷–⁹ In the present study, we have confirmed these earlier observations and examined the relationship of H-V interval to a number of clinical and electrocardiographic parameters in patients with left bundle-branch block.

From the Department of Adult Cardiology, Hektoen Institute for Medical Research of the Cook County Hospital, and the Department of Medicine, Abraham Lincoln School of Medicine, University of Illinois College of Medicine, Chicago, Illinois.

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Address for reprints: Dr. Kenneth M. Rosen, Cook County Hospital, 1825 W. Harrison Street, Chicago, Illinois 60612.

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Methods

Patient Selection

Fifty-seven patients with left bundle-branch block (LBBB) were studied. All had been seen in consultation by the cardiology service on either the medical or surgical wards of Cook County Hospital.

The criteria for diagnosis of LBBB were as follows: (1) QRS duration of 0.12 sec or greater; (2) the presence of a broad monophasic R wave in lead V_6; (3) S-T depression and T-wave inversion in V_6; (4) absence of Q waves in V_6. Patients with S waves in V_6 were included, but the presence of this atypical finding was noted.

QRS duration was defined as the longest QRS duration in the standard and augmented limb leads. P-R interval was defined as the longest interval from the onset of the P wave to the onset of the QRS in the standard and augmented limb leads.

Arteriosclerotic heart disease was diagnosed if one or more of the following criteria were present: (1) history of typical angina pectoris (12 patients); (2) previous diagnosis of definite myocardial infarction (11 patients); (3) coronary arteriograms showing 75% or greater obstruction in one or more coronary arteries (three patients). Hypertension was diagnosed if two or more blood pressures were recorded with systolic pressure greater than 140 mm Hg and diastolic pressure greater than 90 mm Hg. Most of the hypertensive patients had long histories of elevated blood pressure and eyeground changes on physical examination. A number of patients met criteria for both coronary disease and hypertension. Fifteen patients had neither hypertension nor coronary disease.

Cardiomegaly was diagnosed if the cardiothoracic ratio was 0.55 or greater. Congestive failure was diagnosed if appropriate clinical signs and symptoms were present.

Electrophysiologic Studies

Informed consent was obtained from all patients. All cardiac drugs were discontinued at least 48 hours prior to study. His bundle electrograms (H) were recorded with tripolar catheters passed percutaneously from the right femoral vein, using previously described techniques. Recordings were obtained on a multichannel oscilloscopic photographic recorder (Electronics for Medicine, DR 16, White Plains, New York), at paper speeds of 200 mm/sec. Multiple simultaneous ECG leads were recorded.

Validation of H potentials was attempted in most of the patients using the responses to single and coupled atrial pacing. In an attempt to avoid recordings of right bundle-branch potential, the catheter was withdrawn proximally until both large atrial and ventricular electrograms were recorded, suggesting an atroventricular location of recording electrodes. Validation with His bundle pacing was not attempted since pacing of either atrium, His bundle, right bundle branch, or right ventricular septum results in a QRS of LBBB pattern in patients with LBBB.

No special precautions were taken in regard to the potential development of catheter-induced complete heart block. In the event of symptomatic bradycardia, either the His bundle or the atrial pacing catheter could be passed to the right ventricular apex for emergency ventricular pacing.

The following intervals were measured. (1) P-H: from the onset of the P wave on the surface cardiomgram to the first high-frequency potential of the His bundle electrogram. This interval approximated intraatrial and A-V nodal conduction time. (2) H-V interval: from the first high-frequency potential of H to the earliest deflection of the QRS detected on multiple surface leads. There is disagreement as to the upper limits of normal for H-V interval. Intervals as low as 45 msec to as high as 60 msec have been reported as the upper limit of normal. Based on results in our own laboratory in patients without conduction disease, we have divided patients into three groups. These are: group A with normal H-V (less than 50 msec); group B with intermediate H-V (50-60 msec), and group C with prolonged H-V (greater than 60 msec).

Results

Of the 57 patients with LBBB studied, 14 (24%) were in group A with H-V ranging from 35 to 47 msec, 21 (37%) were in group B with H-V ranging from 50 to 60 msec, and 22 (39%) were in group C, with H-V ranging from 63 to 125 msec.

Age. Ages in group A ranged from 36 to 88 years (62.3 ± 3.9, mean ± se), in group B from 43 to 90 years (66.1 ± 2.9), and group C from 35 to 84 years (61.8 ± 2.2). The age differences in the three groups were not significant.

Sex. There were six males and eight females in group A, 13 males and eight females in group B, and 15 males and seven females in group C. Although females predominated slightly in group A, and males in groups B and C, the differences were not significant.

Hypertension. Hypertension was common in all groups, occurring in seven patients in

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increased incidence of hypertension in group C was of borderline significance ($P < 0.15$).

**Arteriosclerotic Heart Disease.** Arteriosclerotic heart disease was diagnosed in eight patients in group A (57%), three patients in group B (14%), and six patients in group C (27%) (fig. 1). Coronary disease was significantly more frequent in group A when contrasted with groups B and C ($P < 0.02$).

**Congestive Heart Failure and/or Cardiomegaly.** Heart failure and/or cardiomegaly was common in all groups, occurring in 12 patients in group A (86%), in 17 patients in group B (81%), and in 21 patients in group C (95%) (fig. 1). These differences were not significant.

**Electrocardiographic Parameters**

**P-R Intervals.** Individual values for P-R intervals in the three groups are shown in figure 2. P-R intervals could not be measured in four patients in group B and one patient in group C because of atrial fibrillation.

P-R ranged from 0.12 to 0.28 sec in group A, from 0.14 to 0.24 sec in group B, and from 0.16 to 0.48 sec in group C. The mean P-R was $0.172 \pm 0.013$ sec in group A, $0.185 \pm 0.007$ sec in group B, and $0.197 \pm 0.012$ sec in group C.
QRS axis, ranging from \(-75^\circ\) to \(+75^\circ\) in group A, from \(-90^\circ\) to \(+90^\circ\) in group B, and from \(-75^\circ\) to \(+75^\circ\) in group C. The mean axis was \(-8^\circ \pm 12^\circ\) in group A, \(-16^\circ \pm 12^\circ\) in group B, and \(-28^\circ \pm 8^\circ\) in group C. Although the axis was slightly more leftward in group C, the differences were not significant.

S Waves in V6. S waves in V6 were present in five patients in group A (36%), eight patients in group B (38%), and six patients in group C (27%). These differences were not significant.

**Electrophysiologic Findings**

**P-H Intervals.** P-H intervals were noted in the three groups to determine whether H-V prolongation correlated with the presence of additional proximal conduction disease. Individual values for P-H are shown in figure 4. P-H intervals could not be measured in one patient in group A because of atrial flutter at the time of study, and in four patients in group B and one in group C because of atrial fibrillation. P-H ranged from 80 to 175 msec in group A, from 93 to 240 msec in group B, and from 80 to 410 msec in group C. The mean P-H was 131 ± 9 msec in group A, 131 ± 9 msec in group B, and 151 ± 14 msec in group C. Although mean P-H was slightly increased in
group C, this was not statistically significant.

P-H prolongation (greater than 140 msec) was present in six of 13 patients in group A (46%), in five of 17 patients in group B (29%), and in six of 21 patients in group C (29%). These differences were not significant.

Heart Block. Heart block developed in two of the patients during study, presumably reflecting catheter-induced right bundle-branch block superimposed on preexisting LBBB. Heart block in each case was transient, not necessitating any therapy. It is of note that both patients with catheter-induced heart block were in group C with H-V intervals of 69 and 80 msec, respectively. The number of patients is too small to determine whether patients with LBBB and H-V prolongation are more prone to this complication.

Spontaneous heart block developed in one of the patients approximately 1 to 1½ years after initial study. This patient was originally in group C with an H-V interval of 80 msec. His bundle electrograms recorded during pacemaker insertion revealed a site of block distal to H, presumably reflecting progression of bilateral bundle-branch disease.

Discussion

H-V interval represents conduction time from the His bundle recording site to the onset of ventricular activation.1, 2 In left bundle-branch block, this interval reflects conduction time in the His bundle and right bundle-branch system.2-6 Previous observations in patients with rate-dependent left bundle-branch block have demonstrated that H-V intervals can be similar during both normal conduction and during LBBB, implying that uncomplicated left bundle-branch block does not prolong H-V interval.8 Thus, the presence of H-V prolongation in LBBB is suggestive of additional delay in either the His bundle or right bundle branch. The future occurrence of heart block, Stokes-Adams attacks, and/or sudden death may thus relate to the presence of prolonged H-V interval. If clinical and/or electrocardiographic features could be used to infer the presence of prolonged H-V, then a patient group might be defined in whom close observation and possibly more extensive electrophysiologic evaluation were indicated.

Previous reports in smaller series of patients with LBBB have suggested that H-V prolongation is frequent. H-V intervals ranged from 54 to 119 msec in 11 patients reported by Berkowitz et al.,7 from 47 to 72 msec in eight patients reported by Haft et al.,8 and from 53 to 220 msec in nine patients reported by Ranganathan et al.8 The present series is in general agreement with these previous studies. Of our 57 patients with LBBB, H-V was within the normal range (less than 50 msec) in 24%, intermediate (50-60 msec) in 37%, and prolonged (greater than 60 msec) in 39%.

We correlated several clinical features with the presence of normal, borderline, and prolonged H-V intervals. The mean age of the patients in the three groups was similar. Although females predominated slightly in the group with normal H-V intervals, and males in the group with long H-V intervals, this difference in sex incidence was not significant. All three groups had a high incidence of cardiomegaly and/or congestive heart failure, this finding being in keeping with the association of left bundle-branch block with organic heart disease.

Of interest was the incidence of both coronary disease and hypertension in the three groups of patients. Arteriosclerotic heart disease was most frequent in the group with normal H-V intervals, while hypertension, although common in all groups, was most frequent in the patients with prolonged H-V intervals. We would postulate that the group with normal H-V intervals and coronary disease had isolated ischemic lesions involving just the left bundle branch. Isolated septal infarcts involving the left bundle branch have been described by Unger et al., in patients with incomplete LBBB.12 The patients with normal H-V intervals without coronary disease may have had undiagnosed coronary disease, idiopathic degenerative disease involving just the left bundle branch, or other pathologic processes.
The patients with intermediate or prolonged H-V may have had idiopathic sclerodegenerative bilateral bundle-branch disease. The high incidence of hypertension in group C could reflect the association of bilateral bundle-branch disease with hypertension.\textsuperscript{13, 14} It is also likely that some of the patients had mixed ischemic and degenerative lesions.\textsuperscript{15} Lepeschkin suggested that P-R prolongation complicating bundle-branch block could reflect the presence of conduction delays in the functioning bundle branch.\textsuperscript{16} The present study corroborated this in patients with LBBB, since P-R intervals were significantly longer in the patients with prolonged H-V. There was overlap, some patients with prolonged H-V having normal P-R and some patients with normal H-V having prolonged P-R intervals. P-H interval, the component of the P-R interval reflecting intraatrial and A-V nodal conduction, was not significantly different in the patients with normal, intermediate, or prolonged H-V.

QRS duration appeared to correlate with H-V interval in LBBB. Patients with QRS duration of 0.16 sec or greater were likely to have prolonged H-V. This is probably not directly related to the long H-V intervals but may reflect delays in either the left bundle-branch system (central or peripheral) or in the ventricular myocardium.

Combining criteria appeared to increase the specificity of the surface electrocardiogram in detection of H-V prolongation. Nine of 11 patients with both P-R prolongation and QRS durations of 0.16 sec or greater had prolonged H-V intervals. This increase in specificity was accompanied by a loss of sensitivity, allowing a diagnosis of H-V prolongation in only nine of a total of 22 patients with prolonged H-V.

Left-axis deviation in patients with narrow QRS or with right bundle-branch block suggests disease in the anterior radiations of the left bundle branch.\textsuperscript{17} In LBBB, axis cannot be assigned a specific electrophysiologic significance, since it is not clear what role the anterior radiations play in left ventricular activation in this situation. In the present series, axis was not significantly different in the groups with normal, intermediate, or prolonged H-V, suggesting that axis was of no value in diagnosing bilateral bundle-branch disease in patients with LBBB. It has been suggested that normal axis in LBBB correlates with a more benign clinical course.\textsuperscript{18} In the present series of cases, there was no apparent clinical difference between the patients with normal or with left-axis deviation. This supports recent observations by Haft and associates, who also could not detect clinical differences between patients with LBBB and normal or abnormal left-axis deviation.\textsuperscript{19}

In summary, two electrocardiographic findings suggested H-V prolongation in LBBB, these being P-R prolongation and QRS duration of 0.16 sec or greater. Whether these will be of value in predicting the future occurrence of heart block is not known, nor is it known whether H-V interval itself is of value in this regard. Only one of the patients is known to have developed progression of conduction disease in the form of spontaneous heart block. This patient originally had a P-R interval of 0.22 sec, a QRS duration of 0.16 sec, and an H-V interval of 80 msec at original study.

At the present time, the presence of H-V prolongation in LBBB does not by itself suggest that prophylactic demand pacing is indicated. Even the development of transient A-V block during the catheterization procedure does not appear to indicate future progression of conduction disease. The clinical status of the patients reported is being evaluated regularly in an attempt to assess the prognostic usefulness of electrocardiographic and electrophysiologic measurements.

References
\textsuperscript{2} Rosen KM: The contribution of His bundle recording to the understanding of cardiac conduction in man. Circulation 43: 961, 1971
\textsuperscript{3} Nabora OS, Cohen LS, Same P, Lister JW, Scherlag B, Hildner FJ: Localization of A-V conduction defects in man by recording of the
H-V INTERVALS

His bundle electrogram. Amer J Cardiol 25: 228, 1970


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