Atrioventricular Conduction System in Left Bundle-Branch Block with Normal QRS Axis

By David S. Cannom, M.D., Bruce N. Goldreyer, M.D., and Anthony N. Damato, M.D.

SUMMARY
In nine patients with left bundle-branch block (LBBB), normal QRS axis, and normal P-R interval, atrial, His bundle, and ventricular electrograms were recorded. The extrastimulus method was utilized to determine the relative refractoriness of the various components of the atrioventricular conducting system.

The functional refractory period (FRP) of the A-V node was normal in all but one patient. In six patients, the effective refractory period (ERP) of the ventricular specialized conducting system (VSCS) was greater than the FRP of the A-V node. The differential refractoriness within the three components of the VSCS was determined. Before the ERP of the VSCS was reached, a leftward shift in the mean QRS axis was noted in all six patients, thus documenting participation of the left anterior division of the LBB in ventricular depolarization. We found no evidence that the left posterior division participated in ventricular activation and conclude that it is functionally silent in LBBB. Conduction abnormalities in the right bundle branch were demonstrated both by long H-V intervals and the fact that its ERP was prolonged.

The increased refractoriness of all three divisions of the VSCS suggests that the conduction abnormalities producing evidence of LBBB on the surface are trifascicular in nature.

Additional Indexing Words:
Conduction defects
Effective refractory period
Intraventricular conduction
Ventricular specialized conducting system
Extrastimulus method
A-V nodal conduction
Right bundle-branch block

Little direct information is available about the functional status of the atrioventricular conducting system (AVCS) in patients with left bundle-branch block (LBBB). Both the clinical and pathologic features of LBBB have been studied, but a systematic definition of the functional A-V conduction abnormalities in LBBB has not been undertaken.

Ideally, the site and magnitude of conduction impairment in LBBB could be determined by direct measurement of conduction velocities in each component of the AVCS. At present, this is technically not feasible in man. Short of this, we employed indirect technics which measure the differential refractoriness of the various components of the AVCS. The extrastimulus method in conjunction with intracardiac electrograms recorded from the atrium, His bundle, and ventricle allowed comparison of the refractoriness of the subdivisions of the AVCS in patients with LBBB.
Method

Nine patients whose 12-lead electrocardiograms were diagnostic of LBBB, as defined by the New York Heart Association, form the basis of this study. In addition to the LBBB, the ECG of each patient demonstrated: (1) normal sinus rhythm, (2) normal P-R interval, and (3) normal mean QRS axis. These criteria were utilized to achieve maximum patient uniformity. None of the patients was receiving any medication known to alter the refractoriness of the atrioventricular system.

All patients underwent right heart catheterization in the nonsedated, postabsorptive state. The voluntary nature of the study was explained to each patient and informed consent obtained. With techniques previously described in detail, a tripolar electrode catheter* was positioned to record the His bundle electrogram. A quadripolar electrode catheter was advanced via the right basilic vein and positioned against the lateral wall of the right atrium. The proximal two terminals of the atrial catheter were located high in the right atrium adjacent to the area of the sinus node, and were used to record an atrial electrogram. Atrial stimulation was accomplished with the distal two electrodes of this catheter.

Square-wave cathodal stimuli, 1.5 msec in duration and twice diastolic threshold, were provided by a series of waveform and pulse generators. During sinus rhythm the right atrium was stimulated after every tenth sinus beat. The prematurity of the test stimulus was progressively decreased by 5- to 10-msec intervals until the duration of atrial excitability had been scanned and atrial refractoriness encountered (fig. 1). This sequence of atrial premature stimulation was repeated during atrial pacing at three cycle lengths in excess of that of the sinus typically 800, 630, and 500 msec. At every rate, between 20 and 50 test cycles were analyzed.

This stimulation sequence described permitted analysis of both A-V nodal and VSCS refractoriness. The stimulus to the atrium evokes a premature atrial response whose propagation to the His bundle defines the conduction characteristics of the A-V node. In a parallel manner, the premature His bundle depolarization thus produced is, in fact, the premature stimulus applied to the VSCS. Analysis of ventricular responses to premature His bundle depolarizations allows the refractoriness of the VSCS to be determined independent of events within the A-V node.

During all stimulation sequences, atrial, His bundle, and ventricular electrograms together with ECG leads I, II, III, V1, and V6 and a time calibration signal with a 10-msec cycle length were displayed on a multichannel switched-beam oscilloscope† and recorded on magnetic tape. When records were later transferred from tape to photographic paper for analysis, intracardiac electrograms were displayed at filter frequencies between 50 and 400 Hz. During analysis, in addition to standard interval measurements, careful attention was given to the QRS axis and morphology of test responses conducted to the ventricle. All equipment used during the procedure was equipotentially grounded.

Definitions

The following definitions are used in this study:

A-H interval. The interval from the initial sharp deflection of the right atrial electrogram to the onset of the His bundle deflection.

H-V interval. The interval from the onset of the His bundle deflection to the onset of ventricular depolarization as measured by either the R wave of the surface electrocardiogram or the initial deflection of the ventriculogram recorded by the His catheter, whichever is earlier.

Atrioventricular conducting system (AVCS). The cardiac conduction tissues, including the atrium, atrioventricular node, His bundle, right bundle, common left bundle, the two divisions of the left bundle (the anterior-superior and posterior-inferior), and the distal Purkinje fibers.

Ventricular specialized conducting system (VSCS). The entire AVCS below the A-V node.

Functional refractory period (FRP) of the A-V node. The shortest H1-H2 interval which is conducted from any A1-A2 interval.

Effective refractory period (ERP) of the VSCS. The shortest H1-H2 interval at which H2 conducts to the ventricle and results in ventricular depolarization.

Results

The extrastimulus method when utilized in this series of nine patients with LBBB allowed the functional refractory period of the A-V node to be determined in seven, and the effective refractory period of the VSCS to be determined in six. These results will be presented according to the anatomy and sequence of conduction within the A-V conducting system and are summarized in table 1.


†Electronics for Medicine, White Plains, New York.
Method used to determine the refractoriness of the AVCS. Six panels illustrate changes seen with progressive shortening of the interval between the sinus (A₁) and test beats (A₂). In each panel are ECG leads 1, 2, and 3, atrial electrogram (AE), and His bundle electrogram (HBE). Atrial (A), His bundle (H), and ventricular (V) depolarizations are identified. The stimulus artifact (S) is indicated by an arrow. The vertical black bar aligns sinus beats preceding A₂.

(Panel A) A₂ is introduced 710 msec after the preceding sinus beat (A₁-A₂ = 710). There is no delay in either the A-V node or VSCS so that both H₁-H₂ and V₁-V₂ intervals are 710 msec. (Panel B) The A₁-A₂ interval is shortened to 440 msec. A 40-msec conduction delay in the A-V node causes both the H₁-H₂ and V₁-V₂ intervals to be 480 msec. (Panel C) The A₁-A₂ interval is shortened by only 10 msec to 430 msec. A-V nodal delay is unchanged. At this H₁-H₂ interval (460 msec) the effective refractory period of the VSCS is exceeded. Conduction of the test impulse fails distal to the His bundle (H₂), and no ventricular complex is seen. (Panel D) The A₁-A₂ interval is shortened to 350 msec. Delay in the A-V node is increased to 70 msec. The H₁-H₂ interval is 420 msec, 40 msec shorter than the H₁-H₂ interval in panel C. The FRP of the A-V node is shorter than the ERP of the VSCS. (Panel E) Shortening the A₁-A₂ interval to 310 msec causes marked A-V nodal delay. The H₁-H₂ interval (450 msec) is longer than in panel D, even though the A₁-A₂ interval is shorter. (Panel F) The stimulus is 10 msec earlier than in panel E. The absence of an atrial response indicates atrial refractoriness has been encountered.
### Table 1

**Electrophysiologic Data**

<table>
<thead>
<tr>
<th>Patient</th>
<th>Cycle length (msec)</th>
<th>A-H interval (msec)</th>
<th>H-V interval (msec)</th>
<th>FRP of A-V node (msec)</th>
<th>ERP VSCS (msec)</th>
<th>Axis shift V2 (msec)</th>
<th>Initial site of AVCS block</th>
</tr>
</thead>
<tbody>
<tr>
<td>W.M. Sinus (1280–1080)</td>
<td>85</td>
<td>85</td>
<td>440</td>
<td>485</td>
<td>—</td>
<td>VSCS</td>
<td></td>
</tr>
<tr>
<td>J.T. Sinus (660–620)</td>
<td>70</td>
<td>70</td>
<td>340</td>
<td>410</td>
<td>540–410</td>
<td>VSCS</td>
<td></td>
</tr>
<tr>
<td>A.S. Sinus (900–780)</td>
<td>75</td>
<td>75</td>
<td>430</td>
<td>450</td>
<td>470–460</td>
<td>VSCS</td>
<td></td>
</tr>
<tr>
<td>S.G. Sinus (1400–1260)</td>
<td>100</td>
<td>70</td>
<td>460</td>
<td>520</td>
<td>—</td>
<td>VSCS</td>
<td></td>
</tr>
<tr>
<td>M.K. Sinus (1040–940)</td>
<td>120</td>
<td>90</td>
<td>400</td>
<td>490</td>
<td>—</td>
<td>VSCS</td>
<td></td>
</tr>
<tr>
<td>A.C. Sinus (750–730)</td>
<td>80</td>
<td>70</td>
<td>400</td>
<td>490</td>
<td>—</td>
<td>AVN</td>
<td></td>
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<tr>
<td>W.C. Sinus (790–700)</td>
<td>70</td>
<td>80</td>
<td>390</td>
<td>420</td>
<td>500–430</td>
<td>VSCS</td>
<td></td>
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<tr>
<td>A.H. Sinus (900–800)</td>
<td>120</td>
<td>60</td>
<td>&lt;450</td>
<td>&lt;450</td>
<td>480–450</td>
<td>Atrium</td>
<td></td>
</tr>
<tr>
<td>B.S. Sinus (1000–950)</td>
<td>75</td>
<td>90</td>
<td>&lt;400</td>
<td>&lt;400</td>
<td>—</td>
<td>Atrium</td>
<td></td>
</tr>
</tbody>
</table>

Abbreviations: FRP of A-V node = functional refractory period of A-V node; ERP VSCS = effective refractory period of ventricular specialized conducting system; axis shift V2 = range of H1-H2 intervals over which a shift was noted in the mean QRS of the ventricular test response; — = no shift in mean QRS noted.

### A-V Nodal Conduction

During sinus rhythm, the A-H interval of our nine patients with LBBB varied from 70 to 120 msec (mean, 90 msec). These values are within normal limits for our laboratory and in the other laboratories.11

It is known that the conducting tissues can be abnormally refractory even though conduction times may fall within the normal range. Therefore, although A-H intervals were normal in all patients, the extrastimulus method was employed to determine the refractoriness of the A-V node. With this method a precise value for the A-V nodal functional refractory period (FRP) can be determined only if the FRP of the A-V node exceeds the refractory period of the atrium. This conduction obtained in seven of our nine patients.

In our patients, A-V nodal FRPs at sinus rates varied from 340 to 520 msec (table 1). Since the FRP of the A-V node does not vary greatly with rate,12 it was possible to compare findings in our patients with those on a group of normal patients similarly studied.8 With the exclusion of one patient (A.C.) these values for A-V nodal FRP are not significantly different from normal in six of the remaining seven patients.

In the two other patients (A.H., B.S.) in whom atrial refractoriness precluded precise

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11. [Cannom et al.](http://circ.ahajournals.org/) | 12. [Cannom et al.](http://circ.ahajournals.org/) | 8. [Cannom et al.](http://circ.ahajournals.org/)

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measurement of A-V nodal refractoriness, by definition, the FRP of the A-V node would have to be less than the shortest H₁-H₂ recorded during the stimulation sequence. As can be seen (table 1), A-V nodal refractoriness in both patients fell within the normal range (FRP <450 and <400 msec).²

Thus, A-V nodal conduction times were normal in all nine patients with LBBB and normal P-R intervals, and A-V nodal refractoriness was normal in all but one.

VSCS Conduction

The H-V interval is a measure of the minimal conduction time within any conducting portion of the ventricular specialized conducting system (see Discussion). In all nine of our patients with LBBB the H-V interval was prolonged (60 to 90 msec; mean, 77 msec). These intervals do not overlap with the H-V intervals in patients with normal intraventricular conduction reported from other laboratories.¹¹

As in the case of the A-V node, precise values for VSCS refractory periods can be determined by the extrastimulus method only if the effective refractory period of the VSCS exceeds the functional refractory period of the A-V node. In six of our nine patients with LBBB the VSCS was more refractory than the A-V node—this occurs infrequently in patients with normal intraventricular conduction.⁸ In two patients atrial refractoriness precluded determining the FRP of the A-V node and in only one patient (A.C.) did A-V nodal refractoriness preclude measurement of the effective refractory period of the VSCS. The conduction characteristics of the VSCS will be presented in detail for the six patients in whom our methods allowed its determination.

Axis Shift at Short H₁-H₂ Intervals

Surface ECG leads I, II, III, V₁, and V₆ were all recorded during each stimulation sequence so that changes in QRS morphology and axis of the conducted ventricular premature test response (V₂) could be determined. In all six patients, late ventricular test responses (long H₁-H₂ intervals) showed intraventricular conduction with LBBB configuration identical to ventricular responses resulting from conducted sinus impulses. In each patient as H₁-H₂ intervals shortened, a critical interval was reached at which intraventricular conduction suddenly changed. The mean QRS axis of the ventricular test response shifted to the left (fig. 2). The H₁-H₂ intervals at which this intraventricular aberration occurred are given for each patient in table 1. The extent of this characteristic leftward shift in mean QRS axis was from 6° to 68°. The shift in axis is shown for each patient in the graph in figure 3.

There were characteristic similarities in all patients when this QRS-axis shift was observed. (1) The shift in axis occurred suddenly after a series of longer H₁-H₂ intervals during which V₂ had a typical LBBB configuration. (2) The mean QRS axis, once having shifted, continued to demonstrate this

Figure 2

The leftward shift in mean QRS axis seen in all patients in whom the VSCS was the most refractory component of the AVCS is demonstrated in patient A. S. Shown are ECG leads 1, 2, and 3, atrial electrogram (A), and His bundle electrogram (H). Atrial (A), His bundle (H), and ventricular (V) depolarizations are identified. The stimulus artifact is shown by the arrow in the atrial electrogram. Vertical time lines appear at 100-msec intervals. At an H₁-H₂ interval of 460 msec, the premature ventricular depolarization in lead 2 shows diminished R wave and a deep S wave, which together with deepening of the S wave in lead 3 indicates a leftward shift in the mean frontal QRS axis from +47° to −13°, a total of 60° (arrows). Note that the H-V interval of this beat remains 75 msec.
pattern over the entire range of shorter V1-V2 intervals. (3) No other changes in QRS morphology were observed, and no QRS complexes intermediate between those with typical LBBB pattern and those with LBBB and a leftward shift in axis were ever observed. The leftward-axis shift was consistent only with the hypothesis that in patients with LBBB and normal QRS axis the left anterior-superior division of the left bundle still participates in antegrade conduction and depolarization of the high lateral portion of the left ventricle (see Discussion).

**Failure of Conduction below the His Bundle**

In all six patients in whom VSCS refractoriness could be determined, our methods allowed this because the effective refractory period of the VSCS as a whole exceeded the functional refractory period of the A-V node. Stated in another way, in all six patients, premature test responses evoked in the His bundle (H2) failed to conduct to the ventricles and evoke V2; i.e., H2 was “blocked” below the His bundle.

Since in each patient the ERP of the left anterior division of the left bundle was exceeded at a specific H1-H2 interval, V2’s conducted at shorter H1-H2 intervals could only have been conducted to the ventricles via the right bundle branch (see Discussion). As a result, the shortest H1-H2 interval at which H2 still conducted to the ventricles represents the effective refractory period of the right bundle branch. The ERP of the right bundle determined in this manner varied from 410 to 520 msec—and was distinctly prolonged when contrasted to that of patients with normal intraventricular conduction similarly studied.8

**Functional Characteristics of the Ventricular Special Conduction System**

In five of the six patients in whom the ERPs of the VSCS were determined at sinus rates, the effect of alteration in basic cycle length on VSCS refractoriness was determined. In each patient, when the basic cycle length was shortened by atrial pacing, the ERP of the VSCS (the minimum H1-H2 where H2 still evoked a ventricular response) shortened as well (table 1). The shortening of VSCS refractoriness at increasing paced rates is expected13 and demonstrates that although VSCS refractoriness is prolonged in patients with LBBB and normal-axis rate related changes in refractoriness may still be produced (fig. 4).

**Discussion**

Historically, the specific ECG patterns of right and left bundle-branch block have been
The effect of cycle length on the ERP of the VSCS is shown for each of the five patients in whom the ERP of the VSCS could be determined at more than one cycle length. The ERP of the VSCS is plotted on the abscissa and the basic cycle length (BCL) on the ordinate. The squares represent the ERP of the VSCS at sinus cycle lengths and are identified with the patient's last initial. The circles represent the ERP of the VSCS at decreasing cycle lengths (faster paced rates). The downward shift in VSCS refractoriness for each patient is indicated by the direction of the arrows. It is clear that the ERP of the VSCS shortens as a function of decreasing basic cycle length.

Figure 4

The results of a study show that conduction abnormalities observed in the electrocardiographic QRS complex are due to altered refractoriness within the three fascicles of the VSCS. Prior work using plaque electrodes in the intact canine heart demonstrated that premature stimulation of the atrium or His bundle can produce both delayed conduction within the fascicles of the VSCS and an abnormal QRS configuration. Thus, what appeared to be bundle-branch block was in fact altered ventricular depolarization caused by delayed and not interrupted conduction within that bundle of the VSCS which had the longest refractory period.

In our patients with LBBB and normal axis, altered refractoriness was demonstrated in all three fascicles of the VSCS. We observed a characteristic leftward shift in the mean QRS axis during the stimulation sequence in all six patients in whom the VSCS was the most refractory component of the entire AVCS. Two conclusions follow: (1) The leftward shift in axis demonstrated that before the shift occurred the anterior division of the left bundle was contributing to ventricular depolarization. At long H1-H2 intervals the mean QRS axis is maintained between 0° and +50° because conduction proceeds down the left anterior division and results in depolarization of the anterolateral portion of the left ventricle. (2) At a characteristic, shorter H1-H2 interval the effective refractory period of the anterior division is exceeded and its contribution to ventricular depolarization is lost. As a result, the mean QRS axis shifts to the left. Since this occurs at H1-H2 intervals longer than those which result in a failure of ventricular activation, this means that the ERP of the anterior division of the left bundle is longer than that of another conducting portion of the VSCS.

Demonstration of altered VSCS refractoriness in LBBB was not limited to the anterior fascicle of the LBB however. All nine of our patients as well as previously reported cases of fixed LBBB have typically had prolonged H-V intervals. However, the etiology of this delay between His bundle depolarization and ventricular septal activation was previously unexplained for, when Amer and associates surgically produced LBBB in dogs, there was no increase in the interval between His bundle depolarization and activation of the right interventricular septum. Therefore, anatomic disruption of the left bundle, analogous to the...
concept of blockade of the LBB, cannot be responsible, in and of itself, for the prolonged H-V conduction time noted in patients with LBBB.

The extrastimulus method allowed precise measurement of the ERP of the right bundle in six patients. After the ERP of the left anterior division had been exceeded, the shortest H₁-H₂ interval which still resulted in ventricular depolarization determined the ERP of the right bundle branch. The ERP of the right bundle was prolonged (410 to 520 msec) when compared to that of patients with normal intraventricular conduction. At H₁-H₂ intervals shorter than the ERP of the left anterior division, A-V conduction could be demonstrated to be occurring only via the right bundle. Thus the prolonged H-V time is a reflection of the increased refractoriness of the RBBB.

We found no evidence to suggest that the posterior division of the left bundle contributed to ventricular depolarization. With progressive shortening of the H₁-H₂ interval, no rightward shift in axis or morphologic changes in ventricular depolarization were seen which would have indicated prior contribution of the left posterior division to ventricular depolarization. Although at variance with the anatomic considerations of Rosenbaum and co-workers, our data suggest that in LBBB the left posterior division has the longest effective refractory period of any portion of the AVCS and that its refractoriness was exceeded even at the initiation of the stimulation sequence.

The marked refractoriness of the left posterior division of the left bundle noted in our patients with LBBB might theoretically be expected to produce an initial rightward orientation of the mean QRS axis, analogous to intermittent blockade of the left posterior division seen in normals during premature atrial stimulation. However, our finding that the conduction characteristics of the other two fascicles of the VSCS are altered in our patients with LBBB suggests why the mean QRS axis is between 0° and 50°. In patients with LBBB and normal axis the right bundle branch is less refractory than the anterior division of the left bundle. This is in contrast to normals in which they are either equally refractory or the right bundle is more refractory than the anterior division of the left bundle.

In LBBB, the right bundle initiates septal and ventricular activation and when fused with the contribution to depolarization made by the left anterior division results in a mean QRS axis within the normal range. When the refractory period of the left anterior division is exceeded during the stimulation sequence, the result is a still more marked leftward shift in the QRS axis.

The unexpected finding that the left posterior division is the most refractory portion of the VSCS in LBBB is in contrast to subjects with normal intraventricular conduction in which the left posterior division is generally the least refractory of the three fascicles. When left posterior blockade does occur in the normal heart, assuming almost equal conduction times in the right bundle and the left anterior division and equal contribution to ventricular depolarization by these two divisions, the result is a rightward shift in the QRS axis caused by the relatively late activation of the posterior-inferior wall of the left ventricle.

This study of nine patients with the ECG pattern of LBBB and normal mean QRS axis has suggested that, rather than blockade of the left bundle at some undetermined anatomic site, the characteristic pattern of ventricular depolarization is caused by increased refractoriness in all three divisions of the VSCS. The left anterior division contributes to ventricular depolarization but demonstrates increased refractoriness even when compared to the prolonged effective refractory period of the right bundle. The left posterior division is the most refractory portion of the entire AVCS, and is functionally silent even at the longest H₁-H₂ intervals. Its failure to participate in ventricular depolarization is primarily responsible for the QRS configuration in LBBB.

References


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