Left Posterior Fascicular Block in Canine and Primate Hearts

An Electrocardiographic Study

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SUMMARY
To determine the electrocardiographic consequences of experimentally produced left posterior fascicular block, electrocardiograms of three baboons and three dogs were recorded from extremity and chest leads, from an esophageal lead, and from atraumatic exploring epicardial leads both before and after left posterior fascicular block and again after right bundle-branch block. The following electrocardiographic changes were noted: In direct epicardial leads from sites overlying zones of delayed myocardial excitation (1) increased ratio of R to S; (2) later intrinsic deflection time; and (3) appearance or augmentation of the Q wave. These altered potentials also appeared as rotations of the mean spatial QRS electrical axis. The QRS duration increased slightly. The addition of RBBB produced a further delay in deflection time in epicardial leads from the RV and a further increase in QRS interval. The epicardial envelopment by the excitation process is portrayed in diagrams. These observations emphasize the need for caution in the diagnosis of left posterior fascicular block alone or in combination with RBBB.

Additional Indexing Words:
Right bundle-branch block
Excitation
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In previous accounts we have described the electrocardiographic consequences of experimentally produced left anterior fascicular (arborization) block (LAFB) alone or when combined with right bundle-branch block (RBBB). The present report provides corresponding definition of left posterior fascicular block (LPFB).

Methods
Studies were done on three baboons and three mongrel dogs. Baboons were first tranquilized with phencyclidine, 2 to 3 mg/kg given intramuscularly, followed by pentobarbital, 10 to 15 mg/kg given intravenously. Dogs were anesthetized with only pentobarbital; initially about 30 mg/kg was given intravenously.

The heart of each animal was exposed through a midline sternal incision. Left posterior fascicular block was produced by a ligature technic. When appropriate, the right bundle branch was severed with a knife introduced into the right ventricular cavity through the anterior free wall.

Before and after left posterior fascicular block and again after right bundle-branch block, electrocardiograms were recorded from standard unipolar and bipolar extremity leads, from unipolar chest leads, from an esophageal (posterior) lead, and from atraumatic exploring epicardial leads.

On completion of all recordings, the interior of both ventricles was examined, and the location and extent of each septal lesion were defined.

Results
Figure 1 illustrates a typical left posterior ventricular septal laceration in a baboon heart. This is a view through a posterolateral incision in which the anterior leaflet of the mitral valve was bisected and the aortic valve was divided between left and posterior cusps. The superior portion of the ventricular septum is thus
exposed from which fibromuscular bands extend toward the apex and free wall. The anterior papillary muscle can be seen at the left of the picture, the posterior papillary muscle at the right. The laceration is the vertical shadow just to the left of the posterior papillary muscle, right of the center of the photograph.

Figure 2 presents leads I, II, and III at each stage of a primate experiment. Observe the change in QRS configuration from Rs to qR in leads II and III after left posterior fascicular block alone. QRS duration increased from about 0.06 to 0.07 sec. After addition of right bundle-branch block, the initial components of QRS remained unchanged, but an S wave appeared in all three standard leads; QRS duration increased to 0.08 sec.

Neither of these electrocardiograms (that is, neither LPFB nor LPFB + RBBB) viewed in isolation from earlier or later tracings was clearly abnormal. However, records from direct epicardial leads included in figure 3 established the presence of markedly aberrant excitation. After left posterior fascicular block alone, a lead from a midposterior epicardial point (LP) displayed a qRs complex replacing the rS complex of the control. The intrinsic deflection occurred at 60 msec in contrast to

29 msec in the control. Addition of right bundle-branch block evoked little further change in form or timing of QRS deflections in this lead. By contrast, an epicardial lead from the free wall of the right ventricle (RV) showed minimal change after left posterior fascicular block alone but displayed an increased R to S ratio (RR’S form) after right bundle-branch block. Intrinsic deflection time increased from 35 to 64 msec.

Figure 4 portrays data from a canine experiment in all respects comparable to the primate experiment from which data were derived for figure 3. In the control tracings from a posterior epicardial point (LP) in the dog, the R wave was taller and the S wave shallower than from a comparable point in the baboon. Following left posterior fascicular block in the dog as in the baboon, the ratio of R to S at LP increased and a Q wave appeared. Intrinsic deflection time increased from 27 msec in the control to 40 msec after left
posterior fascicular block, a lesser magnitude of increase than that which developed under comparable circumstances in the baboon.

Following addition of right bundle-branch block, complexes from point RV showed a slightly greater R to S ratio in the dog than in the baboon, but timing of occurrence of intrinsic deflection was comparable. If, instead of choosing the same representative right mid-

ventricular lead for all animals, a lead is selected from the point where excitation is most delayed in each individual animal, arrival of excitation at this point is consistently more delayed in every baboon than in any dog.

QRS duration increased 10 msec or less following left posterior fascicular block in all animals studied, there being no clear distinction between dog and baboon. After addition of right bundle-branch block, QRS duration further increased approximately 30 msec in dogs and 20 msec in baboons to final values of about 85 msec in both species; the control

**Figure 3**

Baboon electrocardiogram at each stage of experiment of figures 1 and 2. Paper speed was 200 mm/sec to show better the form of complexes recorded from direct epicardial leads LP (left ventricle, posteriorly) and RV (right ventricle). Numbers beneath complexes represent intrinsic deflection time in milliseconds. Reference leads I, aVF, and V6 (an esophageal lead near the posterior mass of left ventricle) were chosen for approximate orthogonality. Scale divisions are as in figure 2.

**Figure 4**

Dog electrocardiograms at each stage of a typical experiment. Stages, leads, and scale divisions are same as in figure 3 for ease of comparison. See text for contrasts.

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Figure 5
Patterns of epicardial excitation in baboon heart. Anterior (A) views are at left and posterior (P) views at right. In each diagram, timing of intrinsic deflection in milliseconds is represented by lower case letters and is shaded in accord with legend at the extreme bottom of figure. (Top) CONTROL—before productions of block. (Center) LPFB—left posterior fascicular block. (Bottom) LPFB + RBBB—left posterior fascicular block plus right bundle-branch block.
Figure 6

Patterns of epicardial excitation in dog heart. Views, timing, and stages are as described for figure 5.

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Figure 7

Effects of left anterior fascicular block alone or combined with right bundle-branch block on epicardial excitation of a primate heart. Anterior (A) views are at left, posterior (P) views at right. In each diagram, timing of intrinsic deflection in milliseconds is represented by lower case letters and is shaded in accord with legend at the extreme bottom of figure. (Top) CON-
values of QRS duration, however, were about 10 msec shorter in dogs than in baboons.

With regard to shifts in late components of electrical axis, the leads aVr and Vr, respectively, in figures 3 and 4 showed less inferior and posterior axis shift in the dog than in the baboon after left posterior fascicular block alone, but more superior and anterior axis shift in the dog than in the baboon after addition of right bundle-branch block.

To portray the timing of epicardial excitation at each stage of block, the diagrams of figure 5 were prepared from study of records made at each of 33 epicardial points and averaged for the three different baboons. The control diagrams show two regions of relatively early excitation, one over the anterior midbasal wall of the right ventricle and the other over the apical portion of the left ventricle. Latest zone of excitation of the epicardium was at the posterolateral base. After left posterior fascicular block, excitation was delayed posteriorly. Following the addition of right bundle-branch block, the early focus on the anterior of the right ventricle was eliminated. Spread of excitation from the left anterior (apical) focus then enveloped the free wall of the right ventricle even more slowly than the free wall of the left ventricle.

Figure 6 displays equivalent diagrams of the spread of excitation averaged for the three different dogs. In the control state canine epicardial points were excited earlier than corresponding points in the baboon except anterolaterally where intrinsic deflection time was essentially the same in both species. Left posterior fascicular block resulted in a region of delay posteriorly which was less prolonged and less basally oriented in the dog than in the baboon. Addition of right bundle-branch block caused still further delay in both species, but less in the dog than in the baboon, except for a small zone (3 points) at the base of the heart anteriorly overlying the pulmonary outflow tract of the right ventricle. The most basal point on the anterosuperior surface of the right ventricle was last to be excited at this final stage in all three dogs, whereas the most basal point on the right lateral margin of the right ventricle was last to be excited in all three baboons.

Figure 7 from a baboon and figure 8 from a dog are derived from a previous study of left anterior fascicular block. These are counterparts of figures 5 and 6, respectively, and are included for convenience of comparison.

Discussion

In left posterior fascicular block as in left anterior fascicular block, ventricular complexes of direct epicardial leads from sites overlying zones of delayed myocardial excitation reveal the following changes compared with complexes recorded before production of block: (1) increased ratio of R to S; (2) later intrinsic deflection time; and (3) appearance or augmentation of Q. In left posterior fascicular block, the increased R to S ratio and the appearance or augmentation of Q in the appropriate direct epicardial leads may be reflected in comparable changes affecting leads II, III, and aVr. In left anterior fascicular block, similar changes may occur in leads I and aVL. These altered excitation potentials are also expressed as rotations of the mean spatial QRS electrical axis about the apex-to-base longitudinal anatomic axis of the heart. Late (major) components of the ventricular complex thus shift in a clockwise direction with left posterior fascicular block and a counterclockwise direction with left anterior fascicular block. The degree of axis shift attending either left posterior fascicular block or left anterior fascicular block is greater in the baboon than in the dog and commonly is greater in left anterior fascicular block than in left posterior fascicular block. QRS duration is slightly increased (10 msec or less) in left posterior fascicular block but not in left anterior fascicular block. This difference results from imposing in left posterior fascicular block localized delay in arrival of excitation at
Figure 8

Effects of left anterior fascicular block alone or combined with right bundle-branch block on epicardial excitation of a canine heart. Views, timing, and the three stages are as described for figure 7. Compare with figure 6 to note differences between left anterior and left posterior fascicular blocks.

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that portion of the ventricular myocardium which, under physiologic conditions, already produced potentials responsible for the final components of QRS.

Production of right bundle-branch block in a heart previously subjected to left posterior fascicular block is readily detected by delay in intrinsic deflection time in epicardial leads from the right ventricle and by further increase in QRS interval (about 20 msec in the baboon and 30 msec in the dog). Terminal S deflections in leads II, III, and aVF which were present in control tracings and disappeared when left posterior fascicular block was produced, reappear when right bundle-branch block is superimposed. A shallow S appears also in lead I. However, changes in form of QRS in standard limb and extremity leads are so slight with left posterior fascicular block either alone or in combination with right bundle-branch block that only when electrocardiograms from each stage of experimentally induced combined block can be compared, are the alterations characteristic of those stages readily identifiable.

The manner of epicardial envelopment by the excitation process in the control state, after left posterior fascicular block alone, and after left posterior fascicular block combined with right bundle-branch block in baboon and canine hearts is portrayed diagrammatically.

Recently several authors have presented evidence of the clinical importance of interruption of conduction through one or more components of the triparte bundle-branch system, a system composed of the right bundle branch, the anterior division of the left bundle branch, and the posterior division of the left bundle branch. Results of experimental studies herein reported identify the problems which beset diagnosis of block of the posterior fascicles of the left bundle branch. When in such block the QRS changes in conventional limb leads are slight, they can be distinguished from "normal" only by careful comparison with "control" tracings; when they are more marked, they resemble those of old inferior myocardial infarction. Superimposition of right bundle-branch block on preexist-

**References**

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