Focal Lesions in the Canine Bundle of His
THEIR EFFECT ON VENTRICULAR EXCITATION

By Thomas B. Watt, Jr., and Raymond D. Pruitt

ABSTRACT
The alterations in ventricular excitation resulting from production of discrete punctate electrocautery lesions in the canine bundle of His were studied. Electrocardiographic changes were correlated with lesions identified anatomically by microscopic examination of the atrioventricular bundle. Although partial or complete atrioventricular block developed from lesions at several levels of the atrioventricular bundle, changes in the form of the QRS complex appeared only when lesions involved the distal portion of the bundle. Such QRS changes were recorded in 21 of 32 instances when the lesion involved the branching portion of the common bundle but in none of 6 instances when the lesion involved only the nonbranching portion of the common bundle. This study, then, provides evidence for functionally distinct longitudinal fascicles only within the distal portion of the common bundle. The syncytial character of the terminal fascicular (Purkinje) network may minimize the electrocardiographic evidence for such functional specificity within the common bundle.

KEY WORDS atrioventricular block Purkinje fibers partial bundle branch block fascicular block intraventricular conduction disturbances focal lesions electrocardiographic changes

In both canine and primate hearts, interruption of some, but not all, of the fibers in the left bundle branch and its terminal fascicular network at midseptal level delays excitation of that portion of the left ventricular wall supplied by the interrupted fibers (1-4). This delay is reflected in specific, characteristic electrocardiographic changes in direct epicardial leads. Coincident changes in standard limb and precordial leads may or may not permit identification of the experimentally induced lesion. When experimentally induced fascicular block is compounded by intramural block, excitation of the involved segment of the ventricular wall is further delayed and the associated electrocardiographic changes in direct epicardial leads are accentuated. Total block of the left bundle branch greatly delays excitation of the entire left ventricle and obscures the electrocardiographic changes specific to block of only part of the left branch system (5).

In several earlier clinical reports, the electrocardiographic changes produced by partial block of the left bundle branch have been postulated (6-10). Left anterior fascicular block is reflected in electrocardiograms of humans by left axis deviation and is probably the commonest cause of that phenomenon (6, 11). Combined with right bundle branch block, left anterior fascicular block produces a more extreme deviation of the mean QRS axis in a superior, anterior direction (3). Such combined block frequently is associated with fibrosis in the anterolateral portion of the ventricular septum (9, 12) and often coincides with or precedes some degree of atrioventricular block (7, 9, 10). Left posterior fascicular block causes less dramatic alteration of QRS form and is, therefore, more difficult to identify, especially in the presence of right bundle branch block (4).
Sections perpendicular to the long axis of the bundle of His show features identifying index planes employed to locate longitudinally electrocautery lesions. Arrows indicate critical regions in each portion of illustration. 

A: Most distal section of proximal bundle still not separated from atrial septum by continuous strand of fibrous tissue. 

B: Most proximal section of mid-portion of bundle. Bundle is separated by continuous strand of fibrous tissue (arrow) from atrial septum. This section is defined as the proximal index plane. 

C: Last section of common bundle not showing a continuous strand of conduction fibers linking common bundle with left branch. The top arrow indicates lower end of strand of fibers emerging from common bundle.
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Results of studies of transient block of left branch fibers produced by instillation of cocaine into either the ventricle itself (13) or the region of the common bundle (14) suggested that the electrocardiographic changes characteristic of incomplete left bundle branch block can result from delay of excitation either at the terminal fascicular (Purkinje) network or within the main stem of the left bundle branch. Sherf and James (15) concluded that, even at the atrial level, fiber tracts destined for particular regions of the ventricular myocardium can be identified. These same investigators have presented histologic observations that support longitudinal separation of fiber tracts within the common bundle (16).

In the present investigation we attempted to determine whether a punctate lesion at any given site in the common bundle would produce alteration of ventricular excitation, as evidenced electrocardiographically by a change in the form of the QRS complex, and, if so, to correlate the nature and the location of the lesion with its electrocardiographic consequences.

Methods

Mongrel dogs weighing about 20 kg were anesthetized with sodium pentobarbital, 25-30 mg/kg body weight, iv. The chest of each dog was opened through a midline sternotomy, and the pericardium was fashioned into a sling which supported the heart in an essentially normal, semivertical position. A needle electrode of the type used in electromyography, insulated except at the tip, was bent into a slight arc and was used as an exploring electrode paired with the conventional Wilson central terminal. Electrode contact with the bundle was defined by the appearance of an injury potential after the bundle spike. Once such an injury potential was recorded, the needle electrode was disconnected from the electrocardiograph and connected to the active terminal of a Bovie electrocautery. "Medium" cutting or coagulating settings were used for most experiments, and current was applied to the electrode for about 2 seconds. Subsequent to each period of cautery, the electrocardiograph was reconnected to permit detection of possible changes in rhythm, form of the bundle complex, or form of the QRS complex. Intermittent cautery was continued until one of the following events occurred: (1) an arrhythmia that lasted more than a few beats appeared, (2) the His bundle deflection disappeared, or (3) identical alterations of QRS form were observed in two or more consecutive conducted beats. When one of these events occurred, the needle electrode was withdrawn. Finally, the right bundle branch was severed at midseptal level by transecting it with the cutting edge of a slender steel knife introduced into the right ventricle via a point on its anterior free wall about halfway between base and apex.

At each of the three stages of each experiment—control, after cautery, and after right bundle branch block—electrocardiograms were recorded from standard bipolar and unipolar limb leads, from unipolar chest leads at positions V₆ and V₇, as best as these could be defined in the open-chest preparation, from an esophageal lead at the level of the left ventricle, and from a unipolar exploring electrode (V₆) placed sequentially on 33 epicardial sites that had been preselected and marked with India ink. At 3 of these sites, which represented areas activated primarily by the right branch, the left anterior fascicles, and the left posterior fascicles, respectively, additional electrocardiograms were recorded using contiguous bipolar electrodes. This procedure was carried out to assess with particular precision the timing of arrival of excitation at these representative regions.

After each dog was killed, a block of tissue was removed from the heart. This block contained an inferior portion of the atrial septum, the fibrous tissue at the root of the aorta, a portion of the central fibrous body with the root of the septal leaflet of the tricuspid valve, the nonbranching and branching portions of the bundle of His, and a superior portion of the ventricular septum. Sections 6 μ thick were prepared from the block and every twentieth section was stained by the Goldner trichrome technique and examined microscopically. The location and extent of lesions produced by cautery were determined. Damage to conduction tissue was assessed with particular care.

To permit accurate localization of lesions, we

and the bottom arrow indicates upper end of strand of fibers entering the posterior left branch. D: Left branch fibers form continuous strand with common bundle (arrow). This section is defined as the distal index plane. The bar in the center of the figure represents 1 mm.
defined two reference or index planes each perpendicular to the long axis of the common bundle of His. The proximal index plane is illustrated in Figure 1B. At this level, fibrous tissue of the central fibrous body first formed a continuous strand separating the common bundle from the atrial septum. No such continuity of this strand is seen in the stained section 0.12 mm closer to the atrioventricular node, as illustrated in Figure 1A. The distal index plane is illustrated in Figure 1D. This is the level in the bundle where a continuous strand of fibers proceeds from the common bundle into the left bundle branch. No such continuous strand of fibers connecting the common bundle and the left bundle branch is seen in the stained section 0.12 mm closer to the atrioventricular node (Fig. 1C). If no portion of a lesion extended proximally beyond the proximal index plane (Fig. 1B) or distally into the distal index plane (Fig. 1D), then that lesion could not possibly involve the node or the bundle branches. Such lesions were termed midbundle lesions. If any portion of a lesion extended proximally beyond the proximal index plane, then involvement of the atrioventricular node was not excluded; such lesions were termed proximal bundle lesions. Similarly, if any portion of a lesion extended distally into the distal index plane (Fig. 1D), then bundle branches or their roots might be involved in the lesion; such lesions were termed distal bundle lesions. These index planes thus divided the common bundle into a proximal portion at the head of which lay the atrioventricular node, a middle portion composed of the nonbranching portion of the common bundle with both penetrating (Fig. 1B) and nonpenetrating (Fig. 1C) components, and a distal portion that included the origin of the bundle branches. Lesions were classified as midbundle lesions only if no part of the lesion involved either the proximal bundle or the distal bundle.

Results

Table 1 summarizes the results of 96 technically successful experiments from a series of 111 experiments. Although nearly every lesion in its extreme dimensions involved the midbundle, only 4 lesions extended above the proximal index plane and only 6 were exclusively midbundle in location (Table 1, lines 1 and 2). The remaining 58 identifiable lesions extended into or beyond the distal index plane. Of these, 34 lesions extended into sections where bundle branching was observed but did not involve the branches themselves and 24 extended into the most proximal fibers of the bundle branch, but no lesions involved the bundle branches exclusively (Table 1, lines 3 and 4). This numerical preponderance of lesions in the distal bundle was unintentional and developed because the technique used made insertion of the cauterizing needle into the proximal bundle most difficult.

A conservative interpretation of the data in Table 1 would demand elimination of all experiments in which lesions could not be satisfactorily identified (line 5), experiments in which the lesion might have involved bundle branches (line 4), and all experiments in which no electrocardiographic alteration occurred (column 1). After these eliminations, the data show that QRS changes resulted from 21 of 32 lesions in the branching portion of the common bundle and that no lesion in the nonbranching portion caused QRS changes. In other words, 11 lesions in the branching portion and 6 lesions in the nonbranching portion had no effect on the appearance of the QRS complex. The data, therefore, suggest that any lesion located totally above the branching portion of the common bundle is unlikely to produce a detectable change in QRS form. Fischer's exact method for stati-

<table>
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<th>Type of Lesion</th>
<th>NC</th>
<th>IAVB</th>
<th>CAVB</th>
<th>QRS</th>
<th>QRS&amp;IAVB</th>
<th>QRS&amp;CAVB</th>
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<td>0</td>
<td>0</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>3. Distal bundle</td>
<td>4</td>
<td>7</td>
<td>4</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>24</td>
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<td>0</td>
<td>1</td>
<td>1</td>
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<td>6</td>
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<td>5. Unidentifiable</td>
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<td>1</td>
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Definitive experiments are grouped by the boxes. All 6 of the definitive lesions which did not involve the distal bundle resulted in no change in the QRS complex. Of the 32 definitive lesions involving the distal bundle, 11 caused no change in the QRS complex and 21 produced QRS alterations. NC = no change, IAVB = transient incomplete atrioventricular block, CAVB = transient complete atrioventricular block, QRS = alteration of QRS without atrioventricular block, QRS & IAVB = QRS alteration plus incomplete atrioventricular block, and QRS & CAVB = QRS alteration plus complete atrioventricular block.
tically testing these data gives a probability of $0.0045$ that such a distribution could be due to chance alone. If the reduction of these data is liberalized to include from Table 1 either experiments involving bundle branches (line 4) or experiments in which no electrocardiographic alteration occurred (column 1), or both, a still lower probability of chance distribution is obtained. Obviously, the experiments in which lesions could not be identified (line 5) cannot be included.

In the 24 experiments in which transient atrioventricular block occurred without associated aberration of the QRS complex (Table 1, columns 2 and 3), an assortment of arrhythmias occurred. Transient third degree atrioventricular block was observed in 16 experiments (column 3). Figure 2 illustrates several stages of block in such an experiment.

Figure 3 from this same experiment is a photomicrograph taken at the cross-sectional level showing the greatest areas of damage within specialized conduction tissue. In the center of each lesion was an open hole sometimes containing amorphous gelatinous material, probably protein denatured by the cautery. Surrounding this was a region of charred tissue in which cellular features were totally unrecognizable. Still more peripheral was a zone in which many nuclei were pyknotic; the cytoplasm stained darkly with less than the usual structural detail, but the cell walls appeared intact. Outside this zone the intercellular spaces were widened, presumably by edema, and contained erythrocytes and leukocytes either in clusters or widely scattered. Borders between zones were indistinct. Only the three inner zones, that is, those zones

![Figure 2](Image)

**Figure 2**

Electrocardiograms showing stages of block in a single experiment in which the form of the QRS complex was essentially unaltered even though varying degrees of atrioventricular block (AVB) were present. A, B, C, and D are sequentially arranged with electrocauterization occurring between A and B. Times shown are intervals after cauterization. Note similarity, but not identity, in the form of QRS complexes during control and recovery periods. P-R interval remains slightly prolonged in the recovery tracing and repolarization changes persist.
FIGURE 3
A punctate cauterization lesion from a section made perpendicular to the long axis of the common bundle of His. View is oriented away from the atrioventricular node toward the bundle branches with the left ventricle on the left and the right atrium and ventricle on the right. At top center is the central fibrous body flanked on the left by the root of the posterior (noncoronary) aortic valve cusp and on the right by the most inferior portion of atrial septum. The triangular cross section of the common bundle lies a little left of center. The ventricular septum occupies the midportion of the lower third of the picture. Two arrows on the left indicate a line of cleavage between the septal myocardium on the right and the fibrous connective tissue on the left. As yet, no cellular components of the left bundle branch are identifiable within this fibrous connective tissue. The irregularly shaped ring outlines the presumed borders of the lesion as described in the text. In this section, which was located 0.72 mm proximal (toward atrioventricular node) to the distal index plane, the area of the lesion in the common bundle was greater than in any other section in the series. The lesion...
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showing some evidence of cellular destruction, were included in schematic representations of lesions. These three zones are included within the irregular boundary line drawn on Figure 3. Interrupted portions at the base of this line identify the probable division between bundle and ventricular septum.

No qualitative difference between lesions that produced atrioventricular block and those that did not was detected. Moreover, no characteristic differences were observed in lesions from an additional seven experiments in which third degree atrioventricular block developed and persisted throughout the rest of the experiment, thus preventing evaluation of conduction from a supraventricular focus. These latter lesions were not included in Table 1 since they were considered technically unsatisfactory. Lesions in five of these seven experiments were in the distal bundle, and those in the remaining two experiments were not identifiable in sections obtained.

Analysis of results beyond this point permitted only qualified interpretation. In the 26 experiments showing QRS alteration without atrioventricular block, ventricular excitation patterns were remarkably varied. This variation, together with the difficulty of delineating boundaries of lesions, seriously limited the information that could be derived from any single experiment. Two sets of representative results are presented in Figures 4–6.

In the top portion of Figure 4, unipolar QRS complexes on the left were recorded from the epicardium over the free wall of the right ventricle, as indicated by guidelines. Before production of the lesion the intrinsic deflection occurred 30 msec after onset of the QRS complex; after cauterization the intrinsic deflection occurred 48 msec after onset of the QRS complex, a delay of 18 msec. The area of the R wave increased and that of the S wave decreased concurrently. QRS complexes presented in the center of the top portion of Figure 4 were recorded from a point over the anterior aspect of the left ventricle as indicated by the guidelines. These complexes were not changed significantly after production of the lesion. QRS alterations, reciprocal in character to those recorded from right ventricular epicardial sites, were observed in complexes recorded from a point over the posterior aspect of the left ventricle, as shown on the right of the top portion of Figure 4. Shading of corresponding anterior and posterior sketches of the heart shows that delayed zones generally conformed to the configuration of the right ventricle, with the smallest delay near the apex and the greatest delay near the right posterior base. In the canine heart these changes are characteristic of right bundle branch block.

Figure 5 depicts the bundle lesion producing the electrocardiographic changes shown in the top half of Figure 4. The total length of the lesion in the common bundle slightly exceeded 2.04 mm, and its center lay about 1.56 mm distal to the reference plane of earliest left bundle branching. The lesion did not involve the branches themselves. It was crescent-shaped and concave upward from inferior right to superior center. Presumably it blocked all or nearly all fibers destined for the right branch, although enough fibers destined for the left branch were spared to permit normal left ventricular excitation.

Figure 6 presents the results of the experiment illustrated in the bottom portion of Figure 4. In this instance, the bundle lesion delayed left ventricular excitation. QRS complexes recorded from an anterolateral epicardial point are shown in the bottom center of Figure 4 between the anterior and the posterior heart sketches. The intrinsic deflection occurred at 31 msec before and at 41 msec after production of a punctate lesion in the common bundle, a delay of 10 msec. Again, the area of the R wave was increased in relation to the area of the S wave. Shading itself extended 0.24 mm proximally and 0.96 mm distally to the section shown. Common bundle fibers lying superiorly and to the right of the lesion appear to have been damaged very little. The bar (top left) represents 1 mm.
Electrocardiographic changes in two experiments in which QRS form was altered. Top: Experiment resulting in right bundle branch block. Bottom: Experiment resulting in a degree of left bundle branch block. For each experiment, anterior and posterior views of the heart are presented; the darkest shading represents those areas of ventricular epicardium showing the greatest delay in intrinsic deflection time. The scale in the center of the figure defines the magnitude of delay in each of the four zones shown (ms = msec). Note that the first zone (lightest shading) also includes regions of no delay. Measurements from unipolar QRS complexes recorded from 33 epicardial sites were programed onto an IBM 360/50 computer system to give these shaded zones. Also, for each experiment, QRS complexes before (above) and after (below) production of bundle lesions were recorded from a unipolar exploring electrode at designated epicardial sites. Those on the left were recorded over free wall of the right ventricle, those in the center over anterolateral free wall of left ventricle, and those on the right over posterior (inferior) free wall of left ventricle, as indicated by broken guidelines. Numbers below each complex indicate intrinsic deflection time in milliseconds for that particular complex.

on the heart sketches indicates predominantly left posterolateral delay. Figure 6 shows a cross section of the lesion responsible for these changes. The most proximal component of this lesion began 1.20 mm proximal to the index plane of the first left branching in the lower
Photomicrograph, prepared as in Figure 3, showing electrocautery lesion which produced right bundle branch block in the experiment illustrated in the top half of Figure 4. In this particular cross section, the area of the lesion as it affected the common bundle was greater than in any other section in the series. The lesion originated 1.08 mm proximal to this section in the right inferior aspect of the first part of the branching common bundle and terminated 0.96 mm distal to this section in the superior aspect of the distal part of the branching common bundle; it did not involve the branches themselves. The figure shows the central fibrous body (top left), connective tissue in the region of the inferior border of the atrial septum (top right), lesion obliterating all but right and left inferior corners of common bundle (left of center), and ventricular septum occupying most of lower portion of the figure. Left branch fibers tunneling out of common bundle can be seen to left of arrows. Continuity as seen here between common bundle and left branch fibers can also be identified in sections as far as 1.56 mm further proximally. The bar (center right) represents 1 mm.
FIGURE 6

Photomicrograph, prepared as in Figure 3, showing electrocauterization lesion which produced left bundle branch block in the experiment illustrated in the bottom half of Figure 4. As in Figure 5, this section was chosen to portray greatest involvement of conduction tissue. The lesion extended 0.96 mm proximally and 0.60 mm distally. Arrows indicate faint line (rather distinct in stained slides) separating ventricular septum on right from conduction tissue on left. Continuity between this conduction tissue and left bundle branch fibers was evident in section 0.24 mm further distally. This lesion was classified as a distal bundle lesion though its maximal diameter was located in unbranching portion of common bundle. The bar (top left) represents 1 mm.

left portion of the common bundle. As the damaged tissue was traced through successive-
ly more distal sections, the involved area extended superiorly and rightward to include
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all but the most leftward, inferior portion of the common bundle which, in turn, appeared to move across the crest of the ventricular septum toward the right, ultimately contributing also to right branching. Termination of the lesion was observed in the apex of the common bundle some 0.36 mm distal to the plane of the first left branching—a total length of 1.56 mm. This, then, was indeed a common bundle lesion centered in the distal portion of the zone designated as midbundle. The location of the lesion was such that sparing of extreme left posterior fibers would appear more plausible than sparing of right branch

FIGURE 7

Septal surface of interior of left ventricle from dog in which small punctate lesion high on septum blocked anterior fascicles of left bundle branch. The electrocautery needle, covered by a fibrin clot, is still in place. The needle penetrated wall of aorta just above sinus of Valsalva of right cusp of aortic valve and entered ventricular septum at a point just posterior to middle of that cusp. Tip of needle rested on the most anteriorly placed fascicles of dividing left bundle branch. Small crescent-shaped, vertically placed white line (a photographic highlight) lies just anterior and inferior to terminal 3 mm of needle and just anterior to most distal component of presumably undivided left bundle branch. The burn underlies distal end of this exposed portion of the cautery needle and extends barely beyond the tip of the needle. Iodine stain reveals conducting fibers as dark fine lines emerging from aortic valve region near top center and spreading apically, across the septum in an inverted Y-shape toward anterior and posterior papillary muscles located, respectively, in left and right lower portions of figure.
fibers. Yet, left ventricular excitation was more delayed than was right.

The results illustrated in Figures 4-6 were but two of the several forms of QRS alteration encountered in beats of supraventricular origin from experiments having identifiable lesions extending into the distal portion of the common bundle. It is from this distal portion of the common bundle as we have defined it that the fascicles of the left bundle branch emerge. That damage to these fascicles just after they have emerged from the distal bundle can produce altered left ventricular excitation is illustrated in Figures 7 and 8. In Figure 7, the lesion is shown to be approximately 3 mm in diameter. It damaged the most anteriorly placed fibers of the left bundle branch as that bundle assumed a subendocardial position just below and slightly posterior to the middle of the right cusp of the aortic valve. Electrocardiographic changes produced by this lesion are illustrated in Figure 8. They included a 10-msec delay in the occurrence of the intrinsic deflection in unipolar epicardial leads obtained from an area about 2 cm in diameter on the anterolateral aspect of the free wall of the left ventricle about midway between base and apex. Neither the degree of excitation delay nor the area involved by it was as great as that encountered in left anterior fascicular block in dogs in which the blocking lesion was a septal laceration extending from base to apex (1). The limited nature of the changes produced by a focal lesion may be explained by the presence of unblocked collateral pathways to the left anterior ventricular wall via interconnecting fibers which can be seen between anterior and posterior fascicular groups in the center of Figure 7. The observed electrocardiographic alterations were entirely consonant with a mild degree of left anterior fascicular block in the canine heart. The mean electric axis shifted only slightly to the left from +80° to +70°.

**Discussion**

Results of these studies suggest some measure of functional specificity or tract isolation in the distal portion of the common bundle. They provide no evidence of such functional specificity in the proximal part of the common bundle. Lesions in the proximal portion of the bundle did not produce notable QRS changes and, inferentially, did not alter remarkably the course of ventricular excitation. This fact must be assessed in relation to the observation that some of these same lesions did produce atrioventricular block, either temporary or sustained. Presumably, if functional specificity of fiber tracts existed at a certain level in the common bundle, a smaller
lesion would be required to block one of these tracts than to block the entire bundle. Hence, the consistent failure of lesions in the proximal bundle and the midbundle to alter the course of ventricular excitation and to evoke QRS changes probably was not because they were too small to destroy a functionally specific fiber tract.

Functional specificity of common bundle tracts might be obscured, however, by the syncytial nature of the specialized conduction pathways which form the peripheral Purkinje network. In earlier studies on the effects of septal lacerations on ventricular excitation (1-5), the lesions were designed to sever the connections of a major segment of that network not only with the main left bundle branch but also with the remainder of the network. That a properly placed small circumscribed lesion interrupting anterior fascicles of the left branch can evoke changes of left anterior fascicular block detectable in both direct and indirect electrocardiographic leads is supported by the final experiment reported in this paper (see Results). The degree of change in ventricular excitation evoked by this lesion, however, was slight compared with the change produced by the large septal lacerations of earlier experiments. In the case of the circumscribed lesions, both the delay in arrival of excitation and the area involved in that delay were less than half the corresponding changes produced by some of the large septal lacerations. Moreover, we surmise that the circumscribed lesion of Figure 7 was advantageously located with respect to its blocking effects: the lesion destroyed those fibers that composed the most anteriorly placed components of the peripheral Purkinje network. Presumably, input into such a blocked portion of the network could occur only from components of the network posterior to it. A lesion more centrally placed in the left main branch would block a correspondingly central component of the peripheral network. Input into this blocked central component could occur from adjacent portions of the network both anterior and posterior to it. By this argument, a high septal lesion affecting either extreme anterior or extreme posterior fibers of the left bundle branch would disrupt normal ventricular excitation more than would a centrally placed left branch lesion.

Almost certainly the syncytial character of the peripheral Purkinje network reduces disruption of ventricular excitation that would otherwise be evoked by a focal lesion in the common bundle or in the left main branch. Nonetheless, our findings indicate that QRS changes do occur with common bundle lesions but only when a punctate lesion involves the distal portions of the common bundle and not when the lesion is confined to the midbundle or proximal bundle. This evidence suggests that tract specificity in the common bundle is related to the streaming of fibers about to emerge from the bundle into a fasciculus of the bundle branch.

The question of tract specificity has substantial clinical significance. If such specificity existed and was not regularly obscured by the syncytial character of the peripheral Purkinje network, then a properly placed punctate lesion in the bundle could produce changes in ventricular excitation that only a much more extensive septal or endocardial free wall lesion could evoke. The data reported in this paper do suggest that a small lesion that blocked fascicles of the left bundle branch just as they emerged from the common bundle might indeed produce detectable electrocardiographic changes even in the clinical setting. Certainly such a small lesion can produce right bundle branch block. However, our data also suggest that a punctate lesion nearer the atrioventricular node than the branching portion of the common bundle would be unlikely to produce clinically detectable changes in ventricular excitation.

James and Sherf (16) recently have reported studies of the fine structure of the His bundle by light and electron microscopy in canine and human hearts. They concluded that "the general organization of the His bundle is thus into multiple strands of Purkinje cells, and these strands are largely separated from one another by collagen. This longitudinal separation of Purkinje strands by
collagen, plus the specialized nature of intercellular junctions within each strand, form an anatomic basis for suspecting longitudinal separation of conduction within the normal His bundle."

Wennemark and Kossmann (17) have reported results obtained on study of the spread of excitation through the main stem of the right bundle branch in a section of the isolated, perfused canine myocardium. Their data support existence of functionally significant bridges between fibers composing that bundle and, inferentially, the absence of tract specificity.

Final resolution of the issue of tract specificity within the common bundle must await accumulation of additional data. Particularly desirable would be a study of lesions produced experimentally in primate hearts. That the results of such primate studies would differ remarkably from those obtained in the present study of canine hearts seems unlikely. Our experience suggests that the delays in left ventricular excitation produced by septal lesions are comparable in the two species. Only the shift of mean electric axis in the frontal plane was much greater for a corresponding left anterior septal lesion in the primate heart than it was in the canine heart (1, 2). Data in the present study were accumulated so as to permit recognition of even minor delays in excitation of any portion of the ventricular epicardium. These data, therefore, probably tell most of what can be learned about functional specificity of tracts in the common bundle when altered ventricular excitation is used as the index of block within the substance of that bundle. Another index might give another answer, but the clinical significance of tract block without altered ventricular excitation is not evident.

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