Anatomical Configuration of the His Bundle and Bundle Branches in the Human Heart

GEORGE K. MASSING, M.D., AND THOMAS N. JAMES, M.D.

SUMMARY The relationships among the His bundle, the origin of both bundle branches, and the interventricular (IV) septum were examined histologically in 32 human hearts, and the entire bundle branch systems were delineated in 13 of these. The His bundle in five hearts traversed the right IV septal crest, and the LBB origin was a very narrow stem (maximum 1.5 mm in cross-section) crossing from right to left through the inferior margin of the membranous septum. Proximal LBB anatomy was extremely variable, demonstrating multiple fiber groups which fanned out over the entire left septal surface. The LBB did not divide into two discrete divisions without multiple interconnections. The RBB formed an obtuse angle with the His bundle in 27 of 32 hearts. In those five hearts with “right-sided His bundles,” the right bundle branch was a direct continuation. The clinical, electrophysiologic, and electrocardiographic implications of these anatomical observations are discussed.

PRECISE KNOWLEDGE of the anatomy and distribution of the human atrioventricular (A-V) conduction system is important in understanding the sequence of ventricular activation and thereby the anatomical basis of a variety of conduction disorders. Such knowledge is also valuable for the cardiac surgeon who sometimes must place sutures or make incisions very near (or into) the A-V conduction system in the course of certain intracardiac operations. The anatomy of the human A-V node has been carefully studied in the past1-2 but less attention has been given to important anatomical relationships among the His bundle, the origin and course of both bundle branches, and the interventricular (IV) septum.

Previous studies of human left bundle branch (LBB) anatomy have variously described the LBB: 1) to be divided into two discrete divisions without proximal interconnections;3 2) to have three rather than two separate divisions;4 and 3) to be a diffuse fanlike structure broadly distributed over the left septal surface.5-6 Rosenbaum7 proposed a trifascicular concept of A-V conduction based upon his anatomical studies supporting bidivisional LBB anatomy, and then described electrocardiographic criteria to identify impaired conduction within the anterior and posterior divisions of the LBB. Uhley7 has subsequently

References
14. Druy A: Further observations upon intra-auricular block produced by pressure or cooling. Heart 12: 1143, 1925

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presented a quadrifascicular concept of A-V conduction by separating the LBB into three divisions. Because of these varying descriptions and the sweeping nature of the electrocardiographic interpretations that follow, the present study was conducted to re-examine the gross and microscopic anatomy of the human A-V conduction system distal to the A-V node.

Methods

The A-V conduction system of 32 human hearts was examined postmortem. Thirteen of these were selected to study in detail the anatomy of the LBB as far distally as it could be reliably distinguished, in addition to examination of the His bundle and RBB. In the other 19 hearts, only the proximal A-V conduction system was studied, but this regularly included the first 5 to 10 mm of both bundle branches. All patients in this study died either from noncardiac causes or from known cardiac disease but without electrocardiographic A-V conduction disturbance. Most hearts were obtained following routine examination by the official prosec-

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Abbreviations are MS-L = His bundle traversed both the membranous IV septum and the left septal crest; MS = His bundle coursed entirely within the membranous septum and divided into bundle branches at the crest of the muscular septum; L = His bundle coursed several millimeters below the membranous septum along the side of the IV septum; R = the His bundle coursed to the right of the IV septal crest.

All ages are given in years except those followed by d or m which indicates days or months of age, respectively. The width of LBB origin and length of the LBB are not tabulated for the neonatal hearts, to avoid inclusion of measurements from very small hearts along with measurements from larger adults hearts.

tor. Only those hearts with an intact IV septum were studied. Ages ranged from the newborn period (five hearts) to 87 years (table 1). With the exception of the five neonatal hearts, the large epicardial coronary arteries were cross-sectioned at several millimeter intervals to determine their patency. The right and left ventricular free walls were removed. The aorta was transected just above the coronary cusps and the interatrial septum was cut away one centimeter above the A-V ring. The anterior mitral valve leaflet was excised one centimeter from its insertion to the mitral annulus. Some or all of the mitral valve chordae tendineae were removed.

Viewing the left side of the IV septum of a typical prepared specimen, with the cardiac apex pointed downward (fig. 1A), the left margin of the specimen was cut through either the commissure between the right and left coronary cusps, or through the left coronary cusp. The entire right coronary cusp was thus included intact in each specimen. The left atrium forms the right upper corner in this view. The anterior papillary muscle was not included on the specimen, but the posterior papillary muscle was included in ten of the 13 hearts studied for detailed LBB anatomy.

Viewing the typical specimen from the right side of the IV septum (fig. 1B), the right margin was cut either just anterior to the base of the crista supraventricularis or more anteriorly through the pulmonary outflow tract and pulmonary valve. The left upper corner was slightly posterior and superior to the coronary sinus.

As noted above, histological examination of the A-V conduction system extended into the distal bundle branches in 13 hearts. In ten hearts beyond the neonatal period the bottom of the entire specimen was at least 25 millimeters below, and more often 40 to 60 mm below, the inferior margin of the right coronary cusp. These ten specimens were processed in two to ten blocks cut at 5 to 20 mm intervals parallel to the A-V ring. Three neonatal hearts were embedded in toto in a single paraffin block. Those 19 specimens chosen to study only the proximal A-V conduction system were cut 5 to 10 millimeters below the bottom level of the right coronary cusp, parallel to the A-V ring, and embedded entire in a single paraffin block.

All paraffin blocks were serially sectioned parallel to the A-V ring. This sectioning plane (fig. 2) was chosen as the most suitable to illustrate clearly any possible partitioning or division of the LBB system. At least every 20th section (every 10th section for the neonatal hearts) was saved, and at a minimum every 40th section was stained. In many cases every 10th or 20th section was stained in the region of the His bundle and proximal bundle branches. All histological sections were cut at 6 to 8 microns thickness and stained with Goldner’s trichrome. Examining intervals were thus about 100 to 300 microns.

Photographs were taken of the left side of the IV septum of each of the ten hearts beyond the neonatal age in which distal LBB anatomy was particularly studied. A drawing of the LBB anatomy, reconstructed following examination of the serial microscopic sections of the A-V conduction system, was superimposed on each photograph. The reconstructed LBB anatomy of the three neonatal hearts was drawn with reference to a diagram of the right and non-
coronary aortic cusps and the cut edge of the anterior leaflet of the mitral valve.

Results

Spatial Relationships of His Bundle to Bundle Branch and IV Septum

In each of the 32 hearts the A-V node abutted against the mitral annulus and penetrated the central fibrous body a variable distance to merge with the His bundle. As the His bundle was traced anteriorly, it occupied several different relationships to the membranous and muscular portions of the IV septum (table 1). In 20 of the 32 hearts a portion of the His bundle traveled within the inferior margin of the membranous septum and then along the subjacent left side of the crest of the muscular IV septum (figs. 3-5). In these hearts the LBB origin varied markedly in total width from 2 to 14 mm; the LBB regularly originated from the His bundle at the left septal endocardium. The right bundle branch (RBB) in these hearts crossed over toward the right side of the IV septum at the anterior-inferior margin of the membranous septum through a layer of collagen a millimeter or less in thickness.

In four of the 32 hearts the His bundle distal to the central fibrous body traversed the left side of the IV septal crest several millimeters below the membranous septum (fig. 5A). In one of these four hearts the His bundle in its anterior excursion burrowed into the myocardium to lie one millimeter below the left septal endocardium. The LBB in this heart originated far anteriorly, coursing through a full millimeter of IV septal muscle to reach the left septal endocardium. In the other three hearts the LBB originated directly at the left septal endocardium. The RBB in three of these four hearts crossed rightward through 2 to 4 mm of IV septal muscle (figs. 5A and 6A), and in the fourth heart the RBB crossed over through the inferior margin of the membranous septum.

In three of the 32 hearts the His bundle distal to the central fibrous body was contained totally within the inferior margin of the membranous septum (fig. 5B) until it exited via the anterior-inferior rim, thereby locating the origin of the LBB more anteriorly than usual. The RBB angled slightly to the right and inferiorly to maintain contact with the right IV septal endocardium.

In five of the 32 hearts the His bundle anterior to the central fibrous body coursed to the right of the crest of the muscular IV septum. The LBB in each of these five hearts began as a narrow stem (maximum 1.5 mm in cross section) which crossed leftward through the base of the membranous septum (figs. 6B and 7). Beyond this narrow-stem origin, the
LBB widened abruptly upon reaching the left side of the IV septum. In these five hearts with “right-sided His bundles” the RBB formed a direct continuation of the His bundle, while in the other 27 the His bundle - RBB junction formed a definite obtuse angle.

Anatomy of the Left Bundle Branch

Left bundle branch anatomy for each of 13 hearts studied especially for that purpose and reconstructed from a review of the histological sections is depicted in figures 8–13. The most impressive feature of the LBB anatomy was its marked variability. The origin of the LBB was broad in some and narrow in others (ranging from less than 1 mm to 14 mm) and was significantly influenced by the anatomical relationship of the His bundle to the IV septum. The LBB origin in hearts with “right-sided His bundles” was uniformly narrow, but even in some with “left-sided His bundles” the LBB takeoff was only 2 to 3 mm wide (cases number 8 and 15).

As it coursed down the IV septum from base toward apex, the LBB widened, in some hearts abruptly, and in others more gradually. The size, number, location, configuration, and distribution of LBB subdivisions were unpredictable. In some hearts there were several anterior subdivisions, each originating separately from the main LBB trunk (figs. 8A, 8B, 9B, 11B, 12A, 13A, 13B) while in others there was a single anterior division (figs. 9A, 10A, 10B, 11A, 12B, 13C), which then subdivided. In the latter hearts some of the anterior subdivisions interconnected proximally or distally with midseptal LBB fiber groups. However, in 11 of the 13 hearts there was no division of the LBB for at least 10 mm of its initial course, and in some this nondivision extended for 20 or more millimeters. The two exceptions were more proximal divisions (figs. 9B and 11A) but, as illustrated, these were odd in shape and could not be separated into comparable anterior or posterior fascicles. In one of these (fig. 11A) the initial division was followed by rejoining into a single sheet which then continued for 20 mm.

In examining the distribution of the LBB we arbitrarily divided the base to apex axis of the left ventricle into basal, middle, and apical thirds. In two of the 13 hearts the anterior LBB fibers were distributed toward the basal one-

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**Figure 3.** A) Histological section from Case 3 showing part of the His bundle (marked by solid arrows) within the membranous septum. B) 500 microns below the section shown in A, part of the His bundle (marked by solid arrows) travels to the left of the crest of the muscular IV septum. The open arrow marks the origin of the RBB as it angles rightward. The left portion of these photographs is posterior and the right is anterior. The abbreviations used in these figures also apply to subsequent figures: RA = right atrium; LA = left atrium; MA = mitral annulus; IAS = interatrial septum; IVS = muscular IV septum; RV = right ventricle; LV = left ventricle; TV = tricuspid valve; CSV = crista supraventricularis.

**Figure 4.** This photomicrograph from Case 20 demonstrates at higher magnification the usual course of the His bundle (open arrows) lying on the left side of the crest of the muscular septum. The typically parallel fibers of the His bundle have been cut in the horizontal plane (see fig. 2) as they thrust forward from the A-V node (AVN), one small part of which is seen here.
third of the anterior left ventricle (figs. 8A and 8B) and in three others (figs. 9A, 9B and 13A) toward the junction between the basal and middle thirds. By contrast the posterior LBB fibers were not distributed to the basal one-third of the posterior left ventricle in any heart, but approached the posterior left ventricular wall at its middle or apical thirds.

The proximal few millimeters of the LBB ranged from 4

Figure 5. A) Histological section from Case 5 demonstrating the His bundle (solid arrow) traversing the left side of the muscular IV septum several millimeters below the membranous septum. The open arrow marks the origin of the RBB as it begins to cross rightward through a relatively thick section of IV septal myocardium. RCC is right coronary cusp. B) Histological section from Case 27 depicting the His bundle (solid arrows) contained completely within the inferior margin of the membranous IV septum.

Figure 6. A) The right bundle branch of Case 5 crossed through a thick section of muscular septum, an unusual variation shown at lower magnification in figure 5A. The photographic image is reversed but the labels are correct. B) In Case 26 there is a near mirror-image of the section shown from Case 5, but two major differences are the usual right-sided position of the His bundle (AVB) and as a consequence, the narrow-stem origin of the left bundle branch (LBB).
to 25 cell layers thick and were shielded from both the endocardium and muscular septum by collagen (figs. 14–16). Subendocardial smooth muscle overlay portions of the proximal LBB. As the LBB proceeded several millimeters distally, widening anteriorly and posteriorly, the central LBB fiber groups maintained the same thickness and underlying collagen, whereas the anterior and posterior fiber groups had little or no underlying collagen, and their thickness varied from 1 to 10 cell layers. Separation and reunion of fiber groups were frequent. Gaps between LBB fiber groups of less than 100 μ were not depicted on the diagrams of LBB anatomy (figs. 8–13). Within 10 to 20 mm of its origin the subendocardial smooth muscle and collagen overlying the LBB virtually disappeared and the entire LBB lost its underlying collagen layer separating it from the muscular septum, which made accurate identification and delineation of the distal LBB more difficult (fig. 17). Cells of the distal LBB were generally larger and stained paler than the underlying cells of the ventricular working myocardium. Tracing the LBB sequentially from its origin aided distal LBB identification considerably, and we doubt that it could be reliably done with presently available techniques without serial sections.

On occasion it was difficult to determine the exact cell that marked the anterior or posterior boundary of the LBB. We could delineate LBB anatomy with a high level of confidence for a distance of 24 to 41 mm (average 33 mm) in ten hearts (excluding the neonatal hearts) (table 1). Beyond this the left septal surface was relatively diffusely covered by Purkinje fibers. The LBB-Purkinje network junction is depicted in figures 8–13 by the ragged termination of the LBB. Inspection of the left septal surface (either with the naked eye or with a dissecting microscope) was completely unreliable in predicting LBB anatomy.
Anatomy of the Right Bundle Branch

In 29 of the 32 hearts the RBB, within a few millimeters of its origin, approximated the endocardium of the right side of the IV septum before coursing apically and anteriorly, one-half to one millimeter below the right IV septal endocardium. As noted above, the RBB in four of the 32 hearts originated from a His bundle which coursed along the left side of the IV septum several millimeters below the membranous septum. In three of these hearts the RBB crossed rightward through several millimeters of IV septal myocardium, then curved apically and anteriorly one-half to one millimeter before reaching the right IV septal endocardium. In the fourth heart the RBB crossed rightward through the inferior margin of the membranous septum to reach the right IV septal endocardium. In all 32 hearts the RBB remained a narrow (less than one millimeter in size), unbranched (but variably shaped) structure throughout the IV septum. We did not follow it into the moderator band.

Discussion

Spatial Relationships of His Bundle to Bundle Branches and IV Septum

Some cardiac anatomists have noted that the human His bundle most often courses either in the base of the membranous septum or along the left side of the crest of the IV septum. It has only rarely been reported that the His bundle traversed the right side of the IV septal crest. Histological study by Truex and Bishof of ten human postmortem infant hearts with ventricular septal defects revealed two examples in which the His bundle coursed to the right of the posterior rim of the ventricular septal defect. We noted "right-sided His bundles" in five of the 32 normal human hearts. To our best knowledge, no mention has previously been made of the influence of the His bundle's course on the configuration of the proximal bundle branches or of the possible clinical significance of these variations of normal anatomy.
Within two millimeters of its origin the LBB forms two separate divisions which then reunite 5 mm distally and continue without branching for another 15 mm. B) (Case 10) Two separate slender anterior subdivisions emanate from the main left bundle branch.

Despite a narrow LBB origin, the LBB then spreads diffusely anteriorly and posteriorly, dividing into multiple interconnected subdivisions of different size and shape. B) (Case 9) Following a broad origin, the LBB widens only slightly in its first 8 mm, then divides into a slender anterior division and a broader midseptal division. The anterior division forms three major subdivisions, the first of which reconnects with the midseptal LBB fibers. One might use this case in partial support of the concept of bidivisional LBB anatomy, but this is not representative of LBB anatomy seen in the other 12 serially sectioned hearts.

In this neonatal heart the LBB has a narrow origin, then gradually and evenly widens anteriorly and posteriorly (without early division) as it courses toward the cardiac apex. Anterior LBB fibers are not distributed to the basal one-third of the anterior left ventricle. C) (Case 12) In this neonatal heart the LBB begins to widen anteriorly within a few millimeters of its origin but then turns downward and does not distribute anterior LBB fibers to the basal one-third of the anterior left ventricle.
During the first few years of open heart surgery, high degree A-V block occurred in over 5% of ventricular septal defect repairs. Subsequently cardiac surgeons have routinely located their sutures to repair ventricular septal defects several millimeters below the rim of the right side of the defect, thereby lowering the postoperative occurrence of A-V block to less than 1%. From our own anatomical observations and those of Truex and Bischof it may be anticipated that some cases of A-V block may be particularly vulnerable to marked conduction disturbances and those of Truex and others that some regions of the LBB are usually covered by collagen and endocardial smooth muscle, and that some regions of the LBB are only one or two cell layers thick, it is not surprising that LBB anatomy could not be reliably delineated by visual inspection of the left IV septal surface, even with the aid of a dissecting microscope. Iodine staining of the left IV septal surface within an hour of death is reported as useful to identify LBB anatomy. There is no question that in occasional specimens iodine staining gives good results. However, three factors make us question the reliability of iodine staining for this purpose: 1) the thickness of LBB varying from 1 to 25 cell layers; 2) the varying but considerable thickness of left subendocardial collagen and smooth muscle, and 3) the unpredictably rapid postmortem depletion of intracellular glycogen, upon which iodine staining depends.

Tawara published detailed histological studies of LBB anatomy of two human hearts (fig. 18). The similarity of one Tawara LBB reconstruction to our figure 10A and of his second LBB drawing to our figure 8B may be noted. The average length of the LBB in our ten cases (excluding the three neonatal hearts) was 33 mm, and in both of Tawara’s cases of A-V block may be due to surgical trauma to a His bundle coursing to the right of the rim of the defect.

Belief that the human LBB invariably originates from a broad segment of the His bundle has led most anatomists and electrocardiographers to assume that LBB block must result from relatively extensive lesions. However, the narrow LBB stem that crosses from right to left atop the IV septum in patients with “right-sided His bundles” (five of our 32 cases) may be particularly vulnerable to marked conduction delay produced by very small lesions. Moreover, the origin of the LBB may be much narrower than usually assumed, even when the LBB emanates from a “left-sided His bundle.”

Anatomy of the Left Bundle Branch

In view of the facts that portions of the LBB are variably covered by collagen and endocardial smooth muscle, and that some regions of the LBB are only one or two cell layers thick, it is not surprising that LBB anatomy could not be reliably delineated by visual inspection of the left IV septal surface, even with the aid of a dissecting microscope. Iodine staining of the left IV septal surface within an hour of death is reported as useful to identify LBB anatomy. There is no question that in occasional specimens iodine staining gives good results. However, three factors make us question the reliability of iodine staining for this purpose: 1) the thickness of LBB varying from 1 to 25 cell layers; 2) the varying but considerable thickness of left subendocardial collagen and smooth muscle, and 3) the unpredictably rapid postmortem depletion of intracellular glycogen, upon which iodine staining depends.

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cases was approximately 30 mm. In none of the 13 hearts that we studied nor the two published by Tawara did the LBB separate into only two major divisions with no major interconnections. The LBB in figure 12B does separate into only two divisions but a prominent interconnection between the two is obvious. In figure 11A the LBB separated into two distinct divisions within 2 mm of its origin, and 5 mm further down the IV septum, the LBB reunited to form a 7 mm broad sheet that then continued undivided down the IV septum for an additional 15 mm. If the study of the LBB had included only its first 5 mm, one would have erroneously concluded that the LBB in this heart was clearly separated into anterior and posterior divisions without proximal interconnections. Thus the need to examine the LBB more than a few millimeters distally is obvious.

Use of the terms left anterior and posterior hemiblock9 to describe electrocardiographic left and right axis deviation respectively seems undesirable to us for two reasons: 1) the human LBB is not often, if ever, anatomically organized into two distinct hemidivisions; and 2) the concept of hemiblocks is too exclusive, in that it implies that left and right axis deviation have a single electrophysiological basis—in impaired conduction within one hypothetical portion of the LBB.

Several lines of evidence now suggest that the common denominator underlying electrocardiographic left axis deviation is relative delay of activation of the anterior left ventricular myocardium with respect to posterior left ventricular activation. As a first example, left axis deviation can be produced experimentally in the dog by electrically stimulating (during a paced supraventricular rhythm) the posterobasal left ventricular endocardium a few milliseconds prior to the arrival of each supraventricular impulse at that endocardial site.20 Second, left axis deviation in ostium primum defects may be due both to early posterior left ventricular activation and delayed anterior excitation.20, 21 The A-V node, His bundle, and LBB are all displaced posteriorly in this disorder.22 Verduyn Lunel23 reported a proximal group of LBB fibers distributed directly to the posterobasal left ventricle in ostium primum defects. Presumably for one or both of these reasons posterobasal left ventricular activation begins earlier in ostium primum defect hearts than in normal hearts. Delayed anterior left ventricular depolarization is probably due to the longer distance the anterior LBB fibers must travel from their abnormal posterior origin and to their circuitous route beneath the ostium primum defect to reach the anterior left ventricular endocardium. Third, Watt, Murao, and Pruitt24 have clearly demonstrated experimentally in baboons that large vertically oriented anterior septal lacerations produce left axis deviation by in-

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**FIGURE 15.** For this and figure 16 the level of each section will be related to the initial division of His bundle shown in figure 14A. A here is about 600 microns beyond the section in fig. 14A and B is about 1100 microns (1.1 mm). The LBB (open arrows) is still a single undivided sheet, and the RBB becomes a more smoothly outlined cylinder in cross-section. There is a mixture of collagen and smooth muscle comprising the endocardial covering of the LBB, and its thickness continues to vary as does the thickness of the LBB sheet itself.
interrupting the majority, if not all, of the LBB system distributed to the anterior wall of the left ventricle. Fourth, left axis deviation frequently occurs following anterolateral myocardial infarction and may be due to peri-infarction block (delayed conduction within and around the infarcted myocardium). Fifth, if longitudinal dissociation within the His bundle does occur,\textsuperscript{28} then a focal lesion in His bundle fibers destined to distribute the electrical impulse to the anterior left ventricle might also cause left axis deviation. Similarly, anatomical partitioning by fine collagen septa within the proximal 10 mm of the undivided (in a gross anatomical sense) portion of the LBB\textsuperscript{28} may permit small lesions there to interrupt later distribution of electrical impulses to either the anterior or posterior septum or free wall.
Actually, focal His bundle lesions might cause RBB block, LBB block, or RBB block with left axis deviation. One must conclude that left axis deviation does not necessarily require slowed conduction within some postulated anatomical division of the LBB.

The absence of two discrete LBB divisions is not inconsistent with the reported increased risk of high degree A-V block in patients with RBB block and left axis deviation. If left axis deviation in these patients were due to disease in anterior fibers of the LBB system, then progression of the disease to involve more of the LBB might cause LBB block, and hence complete A-V block. If RBB block and left axis deviation were both due to focal lesions in the His bundle, then even minor progression of such disease could completely interrupt A-V conduction.

In five of our 13 detailed LBB studies one or more anterior LBB subdivisions were distributed from the proximal portion of the anterior LBB toward the basal third of the anterior left ventricular wall. In all 13 LBB studies, by contrast, the posterior LBB fibers approached the posterior left ventricular wall only after coursing one-half to two-thirds of the way from base to apex.

Durrer and his colleagues studied the sequence of ventricular activation in six postmortem beating human hearts and demonstrated three simultaneous areas of earliest left ventricular endocardial activation: basal anterior paraseptal; mid-septal; and posterior paraseptal about two-thirds of the distance from base to apex. Our anatomical studies do not include the anterior and posterior paraseptal free walls, therefore we cannot determine precisely the anterior and posterior terminations of the LBB, but in five of our cases LBB distribution was a close anatomical fit with Durrer's data on onset of activation.

Grant demonstrated that frontal plane QRS mean axis was unrelated to the anatomical long axis of the heart. The marked variation in human LBB anatomy probably has a significant effect on the sequence of left ventricular depolarization, and in turn on frontal plane QRS axis. A mean QRS frontal plane axis of minus 30 degrees in an apparently normal individual might indicate that the proximal LBB fibers to the anterior wall are not distributed to the basal third, thereby slightly delaying basal anterior left ventricular
activation and shifting the terminal portion of the QRS vector superiorly.

Anatomy of the Right Bundle Branch

In some previous anatomical studies the RBB has been described to be a direct continuation of the His bundle. This was so only in five of our cases with "right-sided His bundles." In the other 27 the RBB originated from the His bundle at an obtuse angle.

A number of anatomical differences between the left and right bundle branches probably result in important electrophysiological differences in their behavior. Continuation of collagen partitioning from the His bundle into the first few millimeters of the LBB, plus proximal spreading and branching of the LBB, may effectively partition longitudinal conduction so that focal His bundle lesions can readily delay delivery of the electrical impulse to specific local areas of the left ventricle. Conversely, focal His bundle lesions probably do not result in selective delay of conduction to local regions of the right ventricle because the cylindrical RBB is not partitioned by collagen, has no internal orderly cellular organization, and has no proximal branches. The anatomical arrangement of the right bundle branch does not appear suitable for partitioned conduction from the His bundle to the right ventricle.

References

Anatomical configuration of the His bundle and bundle branches in the human heart.
G K Massing and T N James

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