Site of Antegrade and Retrograde Functional Right Bundle Branch Block in the Intact Canine Heart

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SUMMARY The purpose of this study was to determine the site of conduction delay or block in situ during antegrade and retrograde functional right bundle branch block (FRBBB) and whether reentrant excitation can occur in both directions in the blocked bundle branch, depending on whether the site of block occurred proximally or distally. Studies were performed in 20 adult mongrel dogs during cardiopulmonary bypass, using direct endocardial extracellular bipolar recordings of activation in the His-Purkinje system. In 20 dogs subjected to premature atrial stimulation, 30 sites of antegrade conduction delay or block were documented: 17 were in the proximal portion of the right bundle branch (RBB), seven were in the distal portion and six were diffusely delayed throughout. In nine dogs subjected to premature right ventricular stimulation, nine sites of retrograde conduction delay or block were noted: eight were in the distal portion of the RBB and one was in the proximal portion of the RBB. When antegrade FRBBB occurred, the impulse traveled to the ventricle over the left bundle branch (LBB) and reentered retrogradely the previously blocked RBB. When retrograde FRBBB occurred, the impulse traveled retrogradely over the LBB and reentered anterograde the previously blocked RBB. Both proximal and distal block and both types of reentry could be produced in the same dog. Ventricular tachycardia developed in two dogs, who had 2:1 (or greater) block between activation of the ventricular muscle and RBB. We conclude that antegrade FRBBB usually occurs in the proximal RBB and retrograde FRBBB usually in the distal RBB, but may occur at other sites as well. Reentry in the RBB can occur in either direction, depending on the site of the block. Sustained bundle branch reentry involving the RBB was not a likely cause for sustained ventricular tachycardia in this dog model.

FUNCTIONAL right bundle branch block (FRBBB) is common. Moe et al. concluded from canine studies that the impulse blocked in the upper portion of the right bundle branch (RBB) system during antegrade FRBBB. However, direct recordings from the bundle branches were not obtained in that study. In a canine study, we recorded directly from the distal free-running right ventricular false tendon in situ and showed that antegrade FRBBB occurred proximal to the false tendon recording site. Premature stimulation of the false tendon resulted in retrograde FRBBB. Data obtained by “peeling back” the refractory period of the His bundle and proximal portion of the RBB with atrial preexcitation, analogous to what Moe et al. had done, were consistent with a proximal site of block during retrograde FRBBB.

The site of antegrade FRBBB has been studied in man and dog. Data from direct recordings of the proximal RBB have shown proximal delay and/or block during antegrade FRBBB. The site of retrograde FRBBB in man has not been reported. However, the production of probable bundle branch reentry by premature right ventricular stimulation is most compatible with a distal site of FRBBB. This conclusion is based on the assumption that a distal site of block allows the proximal portions of the RBB to achieve sufficient recovery time to be excitable when the retrograde impulse arrives through the left bundle branch (LBB). An example from a dog study supporting that concept has been published.

Thus, both animal and human studies suggest that antegrade FRBBB after premature atrial or His bundle stimulation occurs in the proximal RBB. However, the data on the site of block during retrograde FRBBB are not consistent.

Using direct endocardial extracellular bipolar recordings of activation in the His-Purkinje system, we performed the present experiments to determine the site of conduction delay or block in situ during antegrade and retrograde FRBBB, and the relationship of bundle branch reentry to the site of block; that is, whether reentrant excitation could occur in both directions in the blocked bundle branch, depending on whether the site of block occurred proximally or distally.

Methods

Studies were performed on 20 adult mongrel dogs of either sex that weighed 16–32 kg. The dogs were sedated with morphine (4 mg/kg intramuscularly), then anesthetized with α chloralose (60 mg/kg intravenously, repeated as needed), intubated and ventilated using a Harvard respirator. The thorax was opened in the midline and the heart was suspended in a pericardial cradle. The temperature, maintained at 37–38°C with a heating blanket, was monitored by a thermistor probe placed in the left jugular vein. Arterial blood pressure was continuously recorded with a cannula in the right common carotid artery connected to a Statham 23Db pressure transducer. To facilitate atrial pacing at slow heart rates, both stellate ganglia were cut and the sinus node was crushed.

Cardiopulmonary Bypass

After i.v. heparin, 3 mg/kg, total cardiopulmonary bypass was achieved using standard techniques.
Large-bore catheters were inserted into the superior and inferior venae cavae through a right atriotomy after ligating the ayzygous vein. Venous blood was shunted into a filtered cardiotomy reservoir (Bentley Labs, model Q220F) primed with 500 ml of blood and 1000 ml of crystalloid. Blood was also collected from a suction catheter placed in the chest cavity and from a left ventricular vent and directed into the reservoir. The blood was then pumped to an oxygenator (Bentley Labs, pediatric size Temprol, model Q110) where it was warmed with a self-contained water bath and aerated with 100% oxygen, before being pumped into femoral arterial cannulas using a peristaltic pump. Mean systolic arterial blood pressure was maintained at 50–60 mm Hg by adjusting venous return, altering arterial flow rate or by infusing normal saline; vaso-pressors were not used. Arterial blood gases were obtained frequently and PaO2 was maintained greater than 100 mm Hg, PCO2 20–35 mm Hg and pH 7.3–7.4. Sodium bicarbonate was added as necessary to correct the pH.

**Recording and Stimulating**

The right atrium was opened with a 3-cm incision parallel to the atiroventricular (AV) groove. An acrylic plaque that contained five stainless-steel electrodes 2½ mm apart was sewn over the His bundle. The ventricles were then fibrillated by rapid stimulation. The right ventricular free wall was incised parallel to the AV groove. A multipolar plaque electrode was sutured over the RBB (fig. 1). The inferior border of the plaque was positioned just superior to the base of the anterior papillary muscle and the upper margin of the plaque was just below the septal insertion of the tricuspid valve leaflets. Bipolar hook electrodes were placed in the right ventricular endocardium just below the plaque, near termination of the RBB at the base of the anterior papillary muscle; through the interventricular septum to record LBB activity; in the left and right ventricular epicardium; and around the right ventricular false tendon. In some experiments, LBB activation was recorded with a bipolar catheter electrode (poles 8 mm apart) in the left ventricle. All electrodes were placed with care to avoid damaging the conduction system. If block or conduction delay occurred spontaneously in either bundle branch system during normal, nonpremature cycles, it was considered to be due to trauma from the electrodes and the data were discarded. Because of the technical demands, this entire recording array could not be achieved in all dogs.

The ventricles were then defibrillated, usually with a single 10-W-sec direct-current discharge (American Optical) across stainless-steel paddles (6 cm in diameter) applied directly to the heart. Three to six pairs of plaque electrodes that gave the largest amplitude of RBB excitation and represented activation from portions of the proximal, middle and distal RBB, a His bundle electrogram, a false tendon electrogram, and assorted ventricular epicardial, endocardial and LBB electrograms, were amplified (Medical Electronics Consulting Engineers) filtered (40–500 Hz), displayed by a memory oscilloscope (Tektronix D11) along with lead II, and recorded on a strip-chart recorder (Honeywell 1858 Visicorder) at a paper speed of 100–500 mm/sec.

The right atrium or epicardial surface of the right ventricle was paced with bipolar hook electrodes at the longest cycle length that maintained capture, using constant current, rectangular stimuli (1-msec duration, two to four times diastolic threshold) delivered from a pulse generator through an isolation transformer (WP Instruments). After eight basic cycles, a premature stimulus was delivered beginning in late diastole to the right atrium or ventricle at decrementing intervals to the point of atrial or ventricular refractoriness (extrastimulus technique).

To avoid disturbing the electrode array, all dogs were maintained on cardiopulmonary bypass throughout the experiment. At necropsy, the ventricular conduction system was stained with Lugol’s solution and the position of all electrodes was verified. Eleven dogs received only premature right atrial stimulation. Nine were studied after both premature right atrial and ventricular stimulation.

**Definitions**

Antegrade FRBBB is defined as conduction delay or block in the normal antegrade activation sequence of the His bundle, RBB and right ventricular electrogram. Retrograde FRBBB is defined as conduction delay or block in the normal retrograde activation sequence of the His bundle and RBB.

Proximal delay or block is defined as delay or block that occurred between the His bundle and the first RBB electrogram recording or between the first and second RBB recordings. Distal delay or block is defined as delay or block between the most distal RBB recording and the right ventricular apical endocardial recording or between the false tendon recording site.
and the right ventricular apical endocardial recording site. Diffuse delay is defined as conduction delay throughout the course of the RBB.

Results

Site of Antegrade FRBBB (table 1)

In the 20 dogs, a total of 30 sites of conduction delay or block in the RBB were documented during antegrade FRBBB after premature right atrial stimulation: 17 were proximal, seven were distal and six were diffuse. Thus, during antegrade FRBBB, the site of conduction delay or block occurred proximally in most dogs. However, diffuse delay or distal block could also occur and cause FRBBB.

An example of diffuse antegrade conduction delay is shown in figure 2. Premature right atrial stimulation (S2) resulted in a prolonged HV interval and conduction delay in both bundle branches, more so in the RBB than in the LBB. In the dogs that had diffuse delay, bundle branch reentry (or reflection) was not observed.

Bundle Branch Reentry During Antegrade FRBBB

When antegrade block was produced proximally in the RBB, the impulse reached the ventricle over the LBB and reexcited the RBB retrogradely. In figure 3, the RBB was activated in a base-to-apex direction (from RB1 to RB2) in the S1 complex. Premature right atrial stimulation (S2) resulted in FRBBB distal to the His bundle recording site. The LBB was activated antegrade with minimal delay and the RBB then was discharged retrogradely, in an apex-to-base direction. The interval between antegrade His discharge and retrograde RB2 activation was only 120 msec and may have been too brief for His to have reexcited retrogradely. Although a sharp deflection was recorded in the His bundle lead, its timing was not consistent with a His echo.

Reentry into part of the RBB was observed when the impulse blocked partway in the RBB. In the example in figure 4, block after S2 occurred between the RB1 and RB2 sites. The retrograde impulse penetrated the RBB from apex-to-base, to the RB2 site. Retrograde activation could not be recorded at the RB1 site. This observation can be explained by the insufficient recovery time afforded RB1 between the time it was activated antegrade and would have been depolarized retrogradely.

Site of Retrograde FRBBB (table 1)

Retrograde FRBBB was produced in nine dogs by premature right ventricular stimulation. In eight dogs, the block or delay was distal and in one dog it was proximal. In these same dogs, antegrade FRBBB occurred more often proximally (table 1). Thus, in contrast to antegrade FRBBB, during retrograde FRBBB, the site of major conduction delay or block was almost always located in the distal RBB.

Bundle Branch Reentry During Retrograde FRBBB

When retrograde block was produced in the RBB, the impulse reached the His bundle over the LBB and reexcited the RBB antegrade. In the example figure 5, the RBB was activated in an apex-to-base direction (from FT to RB1) in the S1 complex. Retrograde activation of the His bundle and sites LB1 and LB2 of the LBB were obscured by the septal components of the electrograms. Premature right ventricular stimulation (S2) resulted in retrograde FRBBB at a site between the right ventricular electrogram recording and the FT site. The LBB was activated retrogradely from apex to base, but the RBB was activated antegrade (base to apex) from RB1 to FT. This sequence of activation is best explained by bundle branch reentry into the previously blocked RBB. Retrograde activation of the His bundle occurred just after activation of RB1, simultaneously with RB2, because of the more proximal position of the His bundle recording site. The reentrant impulse then blocked distal to the FT and failed to reexcite the ventricle and produce another ventricular complex.

Very small changes in the S2S4 interval determined whether bundle branch reentry occurred. In the example in figure 6, an S2S4 interval of 336 msec resulted in delayed retrograde activation of the RBB. An S2S4 interval only 2 msec shorter produced retrograde FRBBB and antegrade activation of the RBB, consistent with bundle branch reentry.

Reentry into part of the RBB occurred when the impulse blocked retrogradely partway into the RBB, in a manner analogous to that shown in figure 4. In the ex-
FIGURE 2. Diffuse conduction delay in the right bundle branch (RBB) system. In the $S_1$ complex, conduction proceeded without delay down both bundle branches. Premature right atrial stimulation at an $S_1$ interval of 135 msec resulted in antegrade conduction delay, primarily in the RBB. The interval between each RBB deflection exceeded the interval between the His bundle deflections by 6-13 msec. The sharp deflection recorded in the false tendon (FT) electrograms became lost in the premature complex. Minimal delay (3 msec) occurred in the left bundle branch (LBB) deflection recorded in the left septum (LS). Increased conduction delay between right ventricular (RV) endocardial deflections was 17 msec, and between left ventricular (LV) endocardial deflections was 10 msec. RV deflections in $S_2$ followed onset of LV electrogram, consistent with RBB block. The electrogram from RB$_d$ exhibited splintered activity in both basic and premature interval complexes. HBE = His bundle electrogram; RB$_1$, RB$_2$, RB$_3$ = electrograms from proximal middle and distal RBB; BCL = basic cycle length. Numbers are in msec. Numbers listed vertically refer to the time interval between successive depolarizations of the His bundle, RBB, LBB, RV and LV. Electrograms retraced for clarity. Schematic interpretation of activation at lower right. More conduction delay occurred in the RBB than in the LBB.

FIGURE 4. Antegrade block in the right bundle branch (RBB) with retrograde activation. In the $S_1$ complex, conduction proceeded down the His bundle and RBB, from RB$_1$ to RB$_2$. At a premature interval of 303 msec, $S_2$ blocked distal to the RB$_1$ recording site. The RBB then was activated retrogradely from RB$_d$ to RB$_5$. Retrograde activation did not reach RB$_1$ and the His bundle recording site. Abbreviations and conventions as in figure 2.
FIGURE 3. Antegrade block in the right bundle branch (RBB) with retrograde activation. In the S1 complex, antegrade activation proceeded without delay down the His bundle and RBB, from RB1 to RB2. Premature right atrial stimulation at an S1S2 interval of 391 msec resulted in block between His bundle and RB1 recording sites. Conduction traveled down the left bundle branch (LBB) without delay between LBB deflections and a 13-msec delay between left ventricular (LV) deflections. The RBB was activated retrogradely, from RB4 to RB3 to RB2. Retrograde RBB deflection in RB5 cannot be seen. Abbreviations and conventions as in figure 2.
ample in figure 7, S₂ resulted in retrograde block between recording sites RB₂ and RB₃. Electrical activity recorded at RB₁ and RB₂ appeared to be nearly simultaneous, although RB₁ preceded RB₂ by 1 or 2 msec. Therefore, it is likely that the impulse traveling antegrade penetrated the RBB to the RB₁ recording site but failed to reexcite the rest of the RBB because the latter had not yet recovered excitability from its previous depolarization.

**Ventricular Tachycardia**

Despite marked delay in antegrade RBB activation in some dogs, ventricular reexcitation (Vₜ phenomenon) occurred only twice during the many episodes of bundle branch reentry elicited after premature right ventricular stimulation. When Vₜ did occur, it was not followed by a V₄.

Two dogs spontaneously developed sustained ventricular tachycardia. In both instances, 2:1 (or great-
er) block between activation of the ventricular muscle and the RBB resulted. Ventricular tachycardia occurred at a cycle length of 240 msec, and activated the RBB retrogradely in alternate cycles (fig. 8). These phenomena during ventricular tachycardia, that is, block between ventricular muscle and the RBB, could be produced in most dogs simply by stimulating the ventricle at rapid rates.

**Discussion**

**Antegrade FRBBB**

Moe et al. provided indirect data to support the conclusions that shortening the \( H_1H_2 \) interval resulted in delayed RBB conduction in some dogs and sudden RBB block in others. In the latter, bundle branch reentry occurred. The experiments reported here sup-

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**Figure 6.** Retrograde conduction delay and retrograde block in the right bundle branch (RBB) with antegrade activation. Retrograde activation of the RBB in \( S_1 \) cannot be seen. (A) Premature right ventricular (RV) stimulation at an \( S_1S_2 \) interval of 336 msec resulted in retrograde activation of the RBB, proceeding from RB to RB. (B) Premature RV stimulation at an \( S_2S_2 \) interval of 334 msec resulted in antegrade activation of the RBB, from RB to RB. Abbreviations and conventions as in figure 2.
port those conclusions. Diffuse RBB conduction delay (fig. 2) does occur and can explain the gradual development of changes in ventricular activation, while sudden activation changes are probably due to complete FRBBB (fig. 3). Reentry of the blocked bundle branch is common and may occur into only portions of the bundle branch (fig. 4). In addition, we found that antegrade FRBBB was more common in the proximal RBB (table 1). Lyons did not note distal block and diffuse delay in the RBB in a recent study.

Retrograde FRBBB

Prompted by the initial study of Moe et al., we investigated the nature of FRBBB while stimulating and recording from the false tendon in vivo. We did not study retrograde FRBBB produced by premature ventricular (myocardial) stimulation. Under the conditions of these experiments, antegrade and retrograde FRBBB occurred between the His bundle and false tendon recording site; the latter was located at least 5 mm proximal to the insertion of the false tendon in the right ventricular free wall. Block was not observed between the false tendon site and the ventricles. In one experiment, preexcitation of the His bundle by only 4 msec eliminated the retrograde block, while in another experiment, 26 msec was required. We concluded that retrograde FRBBB produced by premature false tendon stimulation probably occurred in the proximal portion of the RBB, possibly sharing a common site with antegrade FRBBB. In interpreting these data, it is important to emphasize that retrograde block was produced by stimulating a site in the specialized conducting system proximal to the “gates” described by Myerburg et al.

We postulated the phenomenon of bundle branch reentry to explain the presence of a ventricular reciprocal beat that followed a retrograde His without intervening atrial activity in a patient who had a retrogradely conducting (concealed) accessory pathway. For such an event to occur, the site of retrograde FRBBB should be located in the distal, not proximal, RBB system. This concept seemed at odds with our initial study, but it really was not; the retrograde

**Figure 7. Partial retrograde penetration into the right bundle branch, with block and partial reentry. In the basic complex (S), retrograde activation from false tendon (FT) to RB, and then to RB, can be seen. After premature right ventricular (RV) stimulation at an S,S interval of 350 msec, retrograde activation proceeded from the FT to RB, and then to RB,. However, retrograde activation in RB, and RB, did not occur due to block between RB, and RB,. RB, and RB, were activated antegradely. Abbreviations and conventions as in figure 2.**
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FRBBB in man was produced by right ventricular endocardial stimulation, a site distal to the "gates." This phenomenon subsequently was characterized extensively in man,7,11 and the data convincingly support the presence of bundle branch reentry in response to premature right ventricular stimulation. The sequence of activation is most consistent with retrograde conduction in the LBB and antegrade conduction in the RBB; occasionally, the reverse loop results.11 The site of retrograde block has not been established from these studies in man.

In a canine study similar to ours, Lyons and Burgess8 demonstrated that retrograde FRBBB occurred between the right ventricular myocardium and the distal RBB recording site. Activity in the false tendon was not recorded. Bundle branch reentry similar to that shown in figure 5 was noted. These investigators found, as we did, that the reentry uncommonly completed the loop to reexcite the ventricles. They did not note proximal block, as we did.

The precise site of retrograde block is not known. In our studies, it occurred between right ventricular endocardium and false tendon (fig. 5), so the probable location was at the Purkinje fiber-muscle junction, or gates.10 Given the relatively high safety margin of conduction from muscle to Purkinje fiber,12 block probably occurred at the gates. More refined mapping of these events is important.

Hypothesis to Explain Proximal Antegrade and Distal Retrograde FRBBB

It may seem paradoxical for the same heart, and indeed the same RBB, to exhibit a different site of block, depending on the direction of the premature impulse. In vitro, the duration of the bundle branch action potential and refractory period lengthens18 from its origin at the bifurcation of the His bundle to a point 2–3 mm proximal to its termination in ventricular endocardial muscle.10 An early premature impulse invoked in the atrium or ventricle will block at the first site at which it encounters fibers with an increased refractory period. This site is not necessarily the site at which the longest refractory period of the total system is located. Traveling antegrade, the impulse will encounter the first cells with longer action potential duration and refractoriness just distal to the His bundle, in the early portion of the RBB10 (fig. 2), and, if the impulse arrives sufficiently early, it will block at that site. Conversely, traveling retrogradely, the impulse will encounter cells with longer action potential duration and refractoriness at the site of the gates,10 in the distal RBB.
Bundle Branch Reentry

Although these studies and others establish fairly conclusively the existence of bundle branch reentry, additional experiments are needed. For example, the precise site of “turnaround” from retrograde LBB conduction to antegrade RBB conduction must be established. More accurate mapping of activation in the LBB system and the effects of left ventricular premature stimulation would be of interest.

Our studies support many of the observations reported in man by Akhtar et al.7, 11 Our data are also consistent with contemporary concepts that bundle branch reentry may not be a common cause of ventricular tachycardia in man.14, 16 Reentry over the RBB cannot sustain spontaneous ventricular tachycardia characterized by 2:1 conduction between ventricular myocardium and RBB (fig. 8). Naturally, we cannot exclude the possibility of bundle branch reentry over divisions of the LBB alone. It is obvious that ventricular tachycardia in our animal model may not be similar to that in man. Nevertheless, in reported clinical and animal studies on bundle branch reentry, sustained ventricular tachycardia due to bundle branch reentry appears to be very uncommon.

References

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