Conduction Disorders in the Canine Proximal His-Purkinje System Following Acute Myocardial Ischemia

II. The Pathophysiology of Bilateral Bundle Branch Block

By Nabil El-Sherif, M.D., Benjamin J. Scherlag, Ph.D., and Ralph Lazzara, M.D.

SUMMARY

The evolution of bilateral bundle branch block (BBB) was studied in ten anesthetized dogs by recording electrical activity from the bundle of His (Hb), right (Rb) and left bundles (Lb) for precise localization and characterization of the conduction disorder. Records were obtained before and at intervals up to 8 hours after ligation of the anterior septal artery. Forty to 140 min after ligation conduction was impaired in either the Rb or Lb showing complete BBB both at rapid heart rates (tachycardia-dependent) and slow rates (bradyarrhythmia-dependent) with normal QRS or incomplete BBB at control rates. Later complete BBB became constant at all heart rates. When both branches were significantly involved, various combinations of intraventricular and atrioventricular conduction disorders occurred. Unequal delay in both bundles (unequal 1° bilateral BBB) gave rise to prolonged H-V interval and a QRS pattern of incomplete or complete bundle branch block corresponding to the branch in which the conduction delay was greater. Equal delay in both bundles gave rise to a narrow QRS with prolonged H-V interval. Second degree block in one bundle and complete (3°) block in the other manifested as 2:1, Mobitz type II, or Wenckebach A-V conduction disturbance. Unequal and asynchronous 2° bilateral BBB gave rise to complex patterns of alternating BBB associated with alternation in the H-V interval of 25-35 msec. In two experiments showing alternating BBB, longitudinal dissociation and asynchronous conduction in the distal Hb was suggested.

Additional indexing words:

<table>
<thead>
<tr>
<th>Anterior septal artery</th>
<th>Mobitz type II block</th>
<th>Tachycardia-dependent block</th>
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<td>Wenckebach phenomenon</td>
<td>Bradyarrhythmia-dependent block</td>
<td>Longitudinal dissociation</td>
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<td>Heart block</td>
<td>Intracardiac recording</td>
<td>His bundle electrogram</td>
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Matthewsen,1 Oppenheimer and Williams,2 and Cohn3 were the first to point to the role of bilateral bundle branch block in the genesis of complete A-V block. Later, Wilson and Herrman4,5 succeeded in producing complete A-V block by destroying both branches of the bundle of His while Scher and Shookhoff6 showed that all degrees of atrioventricular block could be produced by sectioning one of the bundle branches and temporarily compressing the other. Early experimental observations were later confirmed by several pathological studies.7-11 On the other hand, most of the clinical reports on bilateral and/or trifascicular bundle branch block were usually based on deductive analysis of standard electrocardiographic recording.12-19 The evolution of bilateral bundle branch block following acute myocardial ischemia has not to our knowledge been systematically studied. In this report the evolution of bilateral bundle branch block following acute ischemic injury of the canine proximal His-Purkinje system is critically analyzed utilizing recently introduced methods for recording from the specialized conduction system.20-28 These observations may prove useful in understanding similar disorders in man.

Material and Method

Forty-five adult mongrel dogs weighing 10-20 kg were anesthetized with intravenous sodium pentobarbital (30 mg/kg). The animals were intubated and placed on a mechanical respirator. Blood pressure in the femoral artery was monitored through a polyethylene catheter connected to a Statham transducer. A thoracotomy incision was made through the left fourth intercostal space. The bifurcation of
the left coronary artery was exposed by retracting the tip of the left atrial appendage and incising the epicardium overlying the proximal portions of the anterior descending and left circumflex arteries. The anterior septal artery was exposed by blunt dissection of the bifurcation and branches of the left coronary artery and a silk ligature was placed around the vessel to be occluded after control records were taken.

To record from the specialized ventricular conducting tissue, electrode catheters (5 French, bipolar rings 1 cm apart) were inserted into peripheral arteries and veins as previously described. Validation of the His bundle and proximal bundle branch potentials were carried out according to techniques reported earlier. In addition to the electrograms, two or more standard electrocardiographic leads were recorded, specifically leads II and aVR. The A-H interval was measured from the first rapid deflection of the A wave in the Hb electrogram to the first rapid deflection of Hb potential. The H-V interval was measured from the Hb deflection to the earliest ventricular activation recorded on either of the intracardiac electrograms or conventional leads. The H-Rb and H-Lb represented the interval between the Hb deflection and both right and left bundle branch potentials respectively. The Rb-V and Lb-V intervals were measured from the first rapid deflection of the right and left bundle branch potentials to the earliest ventricular activation. The measurement error was ±2 msec at a paper speed of 200 mm/sec.

Attral pacing was obtained via a bipolar plunge wire electrode inserted at the left atrial appendage. Pacing was performed with a Grass S88 stimulator and stimulus isolation unit. Vagal-induced slowing or cardiac arrest was accomplished by an already reported method. All data were processed as previously described.

Control records during sinus rhythm, vagal-induced cardiac slowing and atrial pacing up to rates that produced atrioventricular Wenckebach conduction were obtained in each experiment before the anterior septal artery was ligated. All electrograms were in place during control recordings before the artery was ligated. The recorded electrical activity was then monitored for intervals up to 8 hours after ligation. The effect of both cardiac acceleration and slowing was frequently tested throughout the experiment. In several dogs after the initial study was completed, the thoracotomy incision was closed and the dogs were followed for intervals up to seven days. Other dogs were sacrificed at variable intervals and the heart was utilized for in vitro studies utilizing conventional microelectrode techniques. In all experiments postmortem dissection was performed to see that the anterior septal artery had been completely occluded.

Definition of Terms

The diagnosis of right and left bundle branch block was made from the electrocardiographic pattern in leads II and aVR together with analysis of the changes in the bundle branch potentials recorded by catheter electrodes. Normally in the dog lead II inscribes a qR or qRS configuration with a QRS duration of 41 ± 8 msec while lead aVR has a Rs pattern. Right bundle branch block results in marked decrease of the amplitude of the R wave in lead II and the S wave in lead aVR with the development of a terminal broad frequently slurred S wave in lead II and an R wave in lead aVR. Lesser degrees of right bundle branch block give rise to intermediate changes. Right bundle branch block does not usually alter the initial QRS vectors. On the other hand, left bundle branch block gives rise to a predominantly positive frequently slurred deflection in lead II which is sometimes preceded by a small q wave and a predominantly negative deflection in lead aVR frequently preceded by a small r wave. The QRS duration during complete bundle branch block usually increases to 80 ± 10 msec.

The development of conduction delay in the bundle branch system proximal to the recording site of the bundle branch potential usually results in delayed inscription of the bundle potential which moves toward and becomes buried in the ventricular deflection. Under these circumstances when the bundle branch potential is buried in the ventricular deflection, it cannot be discerned whether complete block of conduction proximal to the recording site of the bundle branch has occurred.

“Equal” and “unequal” conduction in both bundles refers to the relative degree of conduction delay in both bundle branches. On the other hand “synchronous” and “asynchronous” conduction refers to the presence or absence of a simultaneous similar conduction pattern in both bundles, e.g., a simultaneous 2:1 conduction ratio.

Results

In all animals the control records showed a Wenckebach type second degree A-V block localized between the recording sites of the atrial and His bundle electrical activity at pacing rates of between 240-300 beats/min. Critical rates for producing Wenckebach periodicity proximal to the recording site of the His bundle deflection usually remained constant throughout the experiment. This observation suggested that the A-V node is not critically involved in the ischemia following ligation of the anterior septal artery. Furthermore, in no single experiment was aberrant intraventricular conduction observed prior to ligation up to pacing rates of 240-300 beats min. At higher pacing rates the impulse always blocked in the A-V node.

In spite of careful attention to the initial vulnerable period six dogs succumbed to ventricular fibrillation in the first 30 minutes after ligation. All but one of the 39 dogs that survived the early period of arrhythmias subsequently showed variable degrees of conduction disorders at the level of the His bundle, right and left bundle branches or any combination thereof. These dogs were grouped according to conduction disorder as previously described (table 1, reference 24). Bilateral bundle branch block was observed in ten experiments (groups IV and V) and forms the material of the present report.

Evolution of bilateral bundle branch block

Table 1 summarizes the findings in ten experiments showing bilateral bundle branch block. Disorders of conduction in the bundle branch system usually
Table 1

Critical Analysis of Ten Experiments Showing Bilateral Bundle Branch Block Following Ligation of the Anterior Septal Artery in the Dog

<table>
<thead>
<tr>
<th>Expt. no.</th>
<th>Initially observed conduction disorder</th>
<th>Bilateral bundle branch block</th>
<th>Site of A-V block</th>
<th>Time course of the conduction disturbance (in hours)</th>
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<tr>
<td></td>
<td>RBBB</td>
<td>LBBB</td>
<td>1° Unequal</td>
<td>Equal*$</td>
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*a*normal or near normal QRS pattern.
†alternating bundle branch block.

Abbreviations: RBBB, LBBB = right and left bundle branch block respectively; Hb, Rb and Lb = His bundle, right and left bundle recordings respectively; TD = tachy-cardia-dependent; BD = bradycardia-dependent; Mob. II = Mobitz type II block; WC = Wenckebach conduction; VF = ventricular fibrillation.
appeared 40 to 140 min after ligation of the anterior septal artery (average 100 min). In six out of ten experiments showing bilateral bundle branch block, normal conduction was regained within eight hours. In two experiments disorders of conduction in the bundle branch system were still observed after two and four days. Two dogs died 5 and 6 hours after ligation due to late development of ventricular fibrillation. In those dogs complete A-V dissociation with an idioventricular escape rhythm was present and ventricular fibrillation was induced by a closely coupled ventricular ectopic beat.

In all cases of bilateral bundle branch, the conduction disorder first manifested itself in either the right or the left bundle as a rate-dependent bundle branch block. Normal intraventricular conduction or incomplete bundle branch block was observed at intermediate heart rates (80-160 beats/min) with complete bundle branch block elicited both on increasing the heart rate (tachycardia-dependent bundle branch block) or slowing the rate (bradycardia-dependent block). The range of heart rates for normal intraventricular conduction or incomplete bundle branch block gradually shortened and then disappeared with the bundle branch block becoming constant at all heart rates. This occurred within a few minutes or up to 45 minutes during which the intermittent conduction disturbance could be demonstrated. Although the range of heart rates for both tachycardia and bradycardia-dependent bundle branch block varied widely from one experiment to the other, it usually ranged from 100-250 beats/min for tachycardia-dependent block and 20-80 beats/min for bradycardia-dependent block.

Figure 1 was obtained from an experiment 80 min after ligation of the anterior septal artery. Catheter electrode recordings of His bundle activity from the right and left sides of the heart (Hb (R) and Hb (L)), together with recordings of the right and left bundle branch potentials (Rb and Lb, respectively), are shown. The record illustrates the development of a tachycardia-dependent left bundle branch block. Note gradual shortening of the R-R interval with change from normal intraventricular conduction (the first two beats) to a pattern of incomplete left bundle branch block (the third beat) and complete left bundle branch block (the last two beats). During incomplete left bundle branch block the H-Lb interval prolongs while the Lb-V interval is markedly shortened. On the other hand, during complete left bundle branch block, the Lb potential is not discernable. This may be due to either marked delay in the inscription of the potential which moves toward and becomes buried in the large V deflection or to a complete block of conduction proximal to the recording site. On the other hand, the H-V interval (which was prolonged from a control value of 35 msec to 50 msec), and the H-Rb and the Rb-V intervals, remained unchanged during both normal intraventricular conduction and the left bundle branch block pattern. This denotes that during the left bundle branch block there is no further delay of activation of the right ventricle through the right bundle branch system.

Figure 2 was obtained shortly after figure 1 and shows the concomitant presence of a bradycardia-dependent left bundle branch block. The first conducted sinus beat following the long pause shows left bundle branch block with the absence of a left bundle branch potential while the next two beats with shorter cycle lengths reveal normal intraventricular conduction. Note the reappearance of the left bundle branch potential before the V deflection and the absence of change in the H-V, H-Rb, and Rb-V intervals during

Figure 1

Experiment 1, record obtained 75 min after ligation of the anterior septal artery showing a tachycardia-dependent left bundle branch block. In this and subsequent records, Hb (L), Hb (R), Rb, and Lb are catheter electrode recordings from the His bundle on the left side, His bundle on the right side, right and left bundle branches respectively. L-2 and aVr are standard electrocardiographic leads. A and V represent the atrial and ventricular electrocardiographic leads. Time lines in this and subsequent records are set at 1 sec intervals.

Figure 2

Experiment 1, record obtained shortly after figure 1 showing bradycardia-dependent left bundle branch block. The polarity of the Lb recording was reversed.
both types of intraventricular conduction. Ninety minutes after ligation, the left bundle branch block became constant at all heart rates.

Later in the experiments, when both bundle branches were significantly involved, various combinations of atrioventricular and/or intraventricular conduction disorders were observed. This is illustrated in figures 3–5 which were obtained from the same experiment shown in figures 1 and 2. Ten minutes after the left bundle branch block became constant, block of conduction in the right bundle developed, resulting in complete A-V block. An idioventricular rhythm escaped at an average rate of 57 beats/min. Complete heart block persisted for 60 min before A-V conduction resumed. This is shown in figure 3. The first half of the figure illustrates two idioventricular escape beats with a left bundle branch block pattern denoting an escape rhythm from the right ventricle. Note that the complete A-V block developed between the site of recording of the His bundle potential and both the right and left bundle potentials. The second half of figure 3 shows resumption of 2:1 A-V conduction with the conducted beats displaying a right bundle branch block pattern. Note absence of the right bundle branch potential before the V deflection which is only preceded by a left bundle potential. The H-V interval of the conducted beats with right bundle branch block pattern is prolonged compared to the conducted beats with left bundle branch block pattern in figures 1 and 2 (from 50 to 85 msec). The conduction pattern could be explained by a 2:1 conduction in the left bundle in the presence of a complete (third degree) block in the right. An alternative explanation, however, is a synchronous 2:1 conduction ratio in both bundles with an unequal conduction time (conduction is more delayed in the right bundle).

Figure 4 was obtained 5 min following figure 3. There is a 1:1 A-V conduction with a right bundle branch block pattern in the first five beats followed by sudden failure of ventricular response to the sixth sinus impulse. A detailed examination of the H-V intervals shows a perceptible increment before the blocked impulse. The difference between the H-V interval of the beat before the block and the one immediately following is 12 msec. This is an example of Wenckebach periodicity with a few msec increment. Concomitant with the change in the H-V intervals there are changes in the QRS configuration with a gradually increasing degree of the right bundle branch block pattern (compare the first and fifth QRS complexes). This may reflect an increasing degree of asynchronous activation of the right and left ventricles (a relatively greater delay in the left bundle branch system).

Figure 5 was obtained 10 min later and shows alternating left and right bundle branch block during a constant sinus rate. The first beat shows a left bundle branch block pattern with an H-V interval of 50 msec (similar to the H-V interval in figures 1 and 2).
BILATERAL BBB

followed by two beats showing right bundle branch block pattern and further prolongation of the H-V interval to 85 and 88 msec before A-V block of the fourth sinus beat. A diagrammatic analysis of the conduction pattern in figure 5 is shown at the bottom of the record and illustrates the presence of a 4:1 conduction ratio in the right bundle and a 4:2 conduction in the left. The presence of asynchronous conduction pattern with unequal conduction delay in both bundles explains the pattern of alternating bundle branch block associated with change in the P-R and H-V intervals. Conduction in the left bundle shows, in fact, a Wenckebach pattern of transmission with minimal increment of the conduction time before block of the sinus beat. The Wenckebach periodicity with a few msec increment in the left bundle is more clearly illustrated in figure 4. The difference between the conduction pattern in figures 4 and 5 is that following block of the last impulse of a Wenckebach period conduction was resumed in the right bundle in figure 5 but not in figure 4.

In all experiments, the development of unequal delay in both bundles (unequal 1° bilateral BBB) gave rise to prolonged H-V interval and a QRS pattern of incomplete or complete bundle branch block corresponding to the branch in which the conduction delay was greater. In five experiments it could be demonstrated that an equal delay in both bundles could give rise to a narrow QRS with prolonged H-V interval. This is shown in figures 6 and 7. Figure 6, panel A, shows control recording before ligation of the anterior septal artery. Figure 6, panels B and C, show consecutive portions of a continuous tracing taken within 30 seconds. The record was obtained 90 min after ligation and illustrates the development of an incomplete degree of right bundle branch block at intermediate heart rates with complete right bundle branch block being elicited both on slowing the heart rate (first half of panel B) and on gradual increase of the rate (second half of panel C). Note the presence of a slight increase of the H-V interval compared to the control record (from 34 to 40 msec). The range of incomplete right bundle branch block disappeared 5 min later with complete right bundle branch block being observed at all heart rates. Figure 7 was obtained 20 min later and illustrates the development of a tachycardia-dependent conduction delay in the left bundle. During sinus rhythm at a rate of 150 beats/min (the first two beats in figure 7, panel A), there was probably a moderate degree of conduction delay in the left bundle. This may explain the observed decrease in the degree of right bundle branch block pattern associated with increase of the H-V interval (compare the right bundle branch block pattern with an H-V of 40 msec in figure 6, panels B and C, to the pattern in figure 7, panel A, with an H-V interval of 65 msec). During atrial pacing at an average rate of 250 beats/min (figure 7, second half of panel A), the left bundle showed a greater degree of conduction delay compared to the right giving rise to a pattern of complete left bundle branch block with further prolongation of the H-V interval (from 65 to 87 msec). Figure 7, panel B, was recorded 5 min later and shows the effect of atrial premature beats of different coupling intervals (the third and fifth beats marked by asterisks). The fifth beat with a very short coupling in-

![Figure 6](http://circ.ahajournals.org/) Experiment 4, panel A, shows control recording. Panels B and C represent consecutive portions of a continuous tracing obtained 90 min after ligation of the anterior septal artery. Note the development of an incomplete degree of right bundle branch block at intermediate heart rates associated with slight increase of the H-V interval. Complete bundle branch block could be elicited both on equal-induced cardiac slowing (first half of panel B) and rapid atrial pacing (second half of panel C). X = ventricular ectopic beat.

![Figure 7](http://circ.ahajournals.org/) Figure 7

Experiment 4, record obtained 110 min after ligation of the anterior septal artery. Panel A shows the effect of rapid atrial pacing (P). Note the change from an incomplete right bundle branch block pattern to complete left bundle branch block associated with a 22-25 msec increase in the H-V interval. Panel B illustrates the effect of atrial premature beats (marked by asterisks). Note partial normalization of the QRS pattern of the first atrial premature beat associated with moderate increase of the H-V interval. The second premature beat with a shorter coupling interval shows a pattern of complete left bundle branch block and further increase of the H-V interval.
interval of 180 msec is conducted with delay in the A-V node (the A-H lengthens from 80 to 135 msec) and shows a pattern of complete left bundle branch block with an H-V interval of 95 msec. On the other hand, the third beat with a coupling interval of 220 msec shows a lesser degree of delay in the A-V node and partial normalization of the QRS configuration. Note that despite a lesser degree of delay of the third beat at the A-V node its H-H coupling interval is 15 msec longer than the H-H coupling interval of the fifth beat. The H-V interval of this beat is midway between the H-V intervals of the sinus beats and the fifth impulse. The partial normalization of the QRS configuration of the third beat is explained by a relatively equal degree of conduction delay in both bundles giving rise to a more synchronous activation of the two ventricles.

In records showing alternating bundle branch block, the change from one bundle branch block pattern to the other was usually associated with alteration of the H-V interval of 25-35 msec (see figure 5 and figure 7, panel A). In two experiments, however, minimal change in the H-V interval was observed. This is shown in figure 8, which was obtained from an experiment 100 min after ligation of the anterior septal artery. Panels A and B are continuous. The first half of panel A shows a complete right bundle branch block pattern that changes to normal intraventricular conduction on critical shortening of the cardiac cycle with intermediate degrees of right bundle branch block at the transitional zone. Panel B shows further increase of the heart rate giving rise to an incomplete left bundle branch block that changes to complete block on critical shortening of the cardiac cycle to 330 msec. The H-V intervals show a gradual minimal increment of no more than 6 msec between the pattern of complete right bundle branch block in the first half of panel A and complete left bundle branch block in the second half of panel B. The behavior of the H-V interval can be explained by suggesting the presence of a bradycardia-dependent conduction delay in the right bundle that markedly or totally improved with a critical increase of the heart rate. The associated presence of a tachycardia-dependent conduction delay in the left bundle can thus occur without leading to any appreciable change in the H-V interval because the impulse can reach the ventricles through the normally conducting right bundle.

Atroventricular block was demonstrated in all experiments. In experiments 1 to 8, the block was always localized between the sites of recording of the His bundle and the bundle branch potentials (table 1). In no single experiment was the block localized distal to the recorded bundle branch potential. In seven out of eight experiments in which recordings of the bundle branch potentials were obtained, the recording was from the proximal bundle branch system as judged by the Rb-V and Lb-V intervals as well as by the PI-V interval measured in the control record.23 28

In experiments 9 and 10, an associated intra-His bundle lesion with a split His potential (H₁ and H₂) was demonstrated. When A-V block developed it was localized between the H₁ and H₂ deflections. This is illustrated in figures 9 and 10 which were obtained from experiment 10. Figure 9 was obtained 2½ hours after ligation of the anterior septal artery and shows catheter electrode recording of the His bundle potential from the left and right sides of the heart (Hb (L) and Hb (R), respectively). Atrial pacing at a constant rate of 293 beats/min gave rise to alternating periods of right and left bundle branch block. Figure 10 was obtained 10 min later. Atrial pacing at a slightly faster rate (307 beats/min) failed to give rise to alternating bundle branch block but resulted in periods of 2:1 A-V block (figure 10, panel A). The block was initially observed to be localized distal to the recording site of both Hb (L) and Hb (R) potentials but proximal to the RB and LB potentials (not shown in the figure). However, slight repositioning (advancement) of the right catheter electrode revealed a split His bundle potential with an initial small amplitude deflection (H₁) and a second sharp deflection (H₂), (see the

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**Figure 8**

Experiment 7, record obtained 100 min after ligation of the anterior septal artery. Panels A and B are continuous and illustrate the change from complete right bundle branch block (first half of panel A) to complete left bundle branch block (second half of panel B) on increase of the heart rate associated with a slight increase of the H-V interval of 6 msec. Note gradual shortening of the Lb-V interval during incomplete left bundle branch block (marked by arrows) with disappearance of the left bundle branch potential during complete block.
magnified sections encircled). The H₁ deflection was recorded simultaneously with the Hb potential in the Hb (L) electrogram and the block was localized between both Hb deflections. Figure 10, panel B, shows that pacing from the distal His bundle gave rise to a QRS configuration similar to conducted atrial beats and a PI-V interval equal to the H₂-V interval of 30 msec. This excludes the possibility that the distal deflection was a proximal right bundle branch potential.

In the absence of the observation in figure 10, figure 9 may be explained by the presence of unequal conduction times in the right and left bundle branches with a conduction delay in the left bundle greater than that in the right by approximately 25 msec. The illustration in figure 10 of an intra-His bundle lesion with the A-V block localized between the two split His deflections rather than in the two bundle branches raises the interesting possibility of longitudinal dissociation in the distal His bundle as the underlying electrophysiological basis of the alternating bundle branch block in this experiment.

**Discussion**

Study of the evolution of bilateral bundle branch block following acute ischemic injury of the canine proximal His-Purkinje system confirms several previous interpretations that were primarily based on deductive analysis of standard electrocardiographic records and supports the concept of first, second, and third degree block in the bundle branches. However, the recently introduced observation of concomitant tachycardia- and bradycardia-dependent conduction disorder in the bundle branch system can explain the behavior of the H-V interval in certain records of bilateral bundle branch block that are otherwise difficult to interpret by traditional concepts. Thus, the change from one bundle branch block pattern to the other associated with only minimal alteration in the H-V interval cannot be explained according to conventional concepts. A complete right bundle branch block pattern, for example, generally denotes the presence of a conduction delay in the right bundle of at least 30-40 msec relative to conduction in the left bundle, the interval necessary for the activation front to proceed from the left ventricle to activate the right ventricle before the excitation wave arrives via the affected right bundle branch. Traditionally, the occurrence of conduction delay in the left bundle branch will now lead to prolongation of the H-V interval (or the P-R interval) without accompanying change of the right bundle branch block pattern. When the delay in the left bundle equals that in the right to within 30-40 msec, further delay in the left bundle will cause gradual narrowing of the QRS complex until a normal QRS pattern is inscribed. When delay in the left bundle exceeds that in the right, the QRS complex will again widen gradually until a complete left bundle branch block pattern is

Figure 9

Experiment 10, record obtained 2½ hours after ligation of the anterior septal artery, showing alternating periods of right and left bundle branch block during rapid atrial pacing. A diagrammatic analysis of the mechanism of conduction is shown at the bottom of the record. R and L represent conduction in the right and left bundle branches, respectively. The diagram shows that the first five beats are blocked in the right bundle while the sixth beat is blocked in both bundles. Successful conduction of the seventh beat in both bundles results in a normal QRS and a shorter H-V interval. This is followed by successive block of the impulse in the left bundle with no change in the H-V interval. Curved arrows represent retrograde conduction which would perpetuate the conduction failure in either the right or left bundle.

Figure 10

Experiment 10, record obtained 10 min after figure 10. Panel A shows atrial pacing at a slightly faster rate compared to figure 9. Note absence of bundle branch block and the development of 2:1 A-V block with the block localized between the two split His deflections (H₁ and H₂) in the Hb (R) electrogram. The H₁ deflection is a slow wave with small amplitude (see magnified section encircled). The Hb (L) electrogram failed to reveal the intra-His bundle lesion. Panel B shows distal His bundle pacing (PI) from the right side catheter electrode.
recorded. The P-R interval will not show any further delay more than the initial 30–40 msec increment. We have shown however (fig. 8) that in the presence of a bradycardia-dependent block in one bundle and a tachycardia-dependent block in the other bundle, a change from one bundle branch block pattern to the other may be associated with minimal change in the H-V interval.

The presence of an unequal first degree block in both bundle branches can give rise to prolongation of the H-V interval and a QRS pattern of incomplete or complete bundle branch block, depending on which branch has greater conduction delay (figure 7, panel A). We have demonstrated, however, in a previous study that the presence of a bundle branch block pattern with a prolonged H-V interval may be due to an associated intra-His bundle first degree block rather than a first degree block in the other bundle branch. If recordings of the proximal His bundle deflection are obtained alone, the intra-His bundle lesion may not be recognized in some of these cases. We have also demonstrated that an approximately equal degree of conduction delay in both bundle branches can give rise to a narrow QRS configuration with slight prolongation of the H-V interval that may not exceed 25 msec (figure 7, panel B). The changes in the P-R interval in the conventional record may be difficult to detect unless associated with significant change in the A-H interval. This suggests that interpretations of bilateral bundle branch block based on analysis of standard electrocardiographic records should be guarded especially when marked changes of the P-R interval are observed (fig. 5).

The presence of unequal and asynchronous second degree bilateral bundle branch block can give rise to very complicated electrocardiographic patterns in which alteration of right and left bundle branch block patterns associated with changes of the P-R and H-V intervals together with intermittent failure of a ventricular response may be seen (fig. 5 and 9). On the other hand, a second degree block in one bundle branch and a third degree block in the other affects A-V conduction. This can take the form of either 2:1 block (second half of figure 3), Mobitz type II block or the Wenckebach periodicity. As in the case of intra-His bundle blocks, critical analysis of several examples of Mobitz type II block at a fast paper speed will reveal that they actually represent a Wenckebach periodicity with a few msec increment (fig. 4).

Localization of the site of maximum conduction disorder during bilateral bundle branch block following ligation of the anterior septal artery is instructive. Our observation that the impulse was always blocked above the site of recording of proximal right and left bundle branch potentials suggests a site of lesion either in the very proximal portion of the bundle branches or in the distal portion of the His bundle. This observation, in addition to the frequent demonstration of an intra-His bundle block (68%), is consistent with the anatomical distribution of the pathological lesion following ligation of the anterior septal artery in dogs. Both are also consistent with recent electrophysiological studies utilizing intracellular recordings which localize the conduction disturbance following ligation of the anterior septal artery in the bundle of His and proximal bundle branches.

The possibility that longitudinal dissociation and asynchronous conduction in the distal His bundle can give rise to changes in the QRS configuration including typical bundle branch block patterns is strongly suggested by the observations in experiments 9 and 10 of the present series. Critical analysis of figures 9 and 10 suggests that alteration in the His bundle conduction time (H1-H2 interval) was associated with alteration of the pattern of right and left bundle branch block. The conduction disorder can thus be ascribed to the presence of two longitudinal pathways with asynchronous refractoriness and conduction velocities in the distal His bundle rather than asynchronous lesions in the proximal right and left bundles. Histological studies suggest an anatomic basis for the concept of longitudinal dissociation of conduction within the His bundle. Recent in vitro and in vivo observations utilizing the experimental model in the present study provide further evidence for localization of dissociation in the ischemic His bundle.

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References

3. Cohn AE: A case of transient complete auriculo-ventricular dissociation showing constantly varying ventricular complexes. Heart 5: 5, 1913-1914
5. Wilson FN, Herrmann GR: An experimental study of incomplete bundle branch block and the refractory period of the heart of the dog. Heart 8: 229, 1921

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8. YATER WM, CORNELL VH, CLAYTON T: Auriculo-ventricular heart block due to bilateral bundle branch lesions. Arch Intern Med 57: 132, 1936
17. CASTELLANOS A Jr., MAYTIN O, ARCEBAL AG, LEMBERG L: Alternating and co-existing block in the division of the left bundle branch. Chest 56: 103, 1969
30. EL-SHERIF N: Tachycardia and bradycardia-dependent bundle branch block after acute myocardial ischemia. Br Heart J. In press
31. EL-SHERIF N, SHERLAG BJ, LAZZARI R, SAMET P: The pathophysiology of tachycardia and bradycardia-dependent block in the canine proximal His-Purkinje system following acute myocardial ischemia. Amer J Cardiol, In press
35. LAZZARI R, EL-SHERIF N, SHERLAG BJ: Cellular basis of ischemic heart block and bundle branch block (abstr) Circulation 43 (suppl IV): IV-189, 1973
36. LAZZARI R, EL-SHERIF N, SHERLAG BJ: Cellular basis of ischemic heart block and bundle branch block (abstr) Circulation 43 (suppl IV): IV-189, 1973
37. LAZZARI R, EL-SHERIF N, SHERLAG BJ: Cellular basis of ischemic heart block and bundle branch block (abstr) Circulation 43 (suppl IV): IV-189, 1973
38. RODRIGUEZ J, EL-SHERIF N, SHERLAG BJ, LAZZARI R: Delta waves and bilateral bundle branch block due to dissociation in the pathologic His bundle. (abstr) Circulation 43 (suppl IV): IV-208, 1973
39. SHERLAG BJ, EL-SHERIF N, LAZZARI R: Bundle branch block due to His bundle lesions. (abstr) Am J Cardiol 33: 169, 1974
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NABIL EL-SHERIF, BENJAMIN J. SCHERLAG and RALPH LAZZARA

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