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

I. The Pathophysiology of Intra-His Bundle Block

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SUMMARY

An intra-His bundle lesion developed in 26 out of 38 dogs (68%) that survived the initial period of 
arrhythmias that followed ligation of the anterior septal artery. The lesion was isolated in the His bundle in 
ten experiments and was associated with bundle branch block in 16. The evolution of intra-His bundle block 
(IHBB) was evaluated utilizing standard ECG leads, catheter electrode, and plunge wire recordings from 
the His bundle and bundle branches. In all experiments the His bundle potential recorded by the catheter 
electrode was split into at least two distinct deflections (H₁ and H₂). This was always associated with a 
progressive increase of the H₁-V interval and represented "first degree IHBB." A "second degree IHBB," 
characterized by intermittent block of the atrial impulse between the two His deflections, consistently 
developed either spontaneously or in response to atrial pacing. The first "stage" of second degree IHBB had 
a Mobitz type II pattern with a constant P-R, H₁-V, and H₁-H₂ intervals of the beats preceding and following 
the block. In 20 of 26 observations, this stage merged into the second stage of second degree IHBB 
characterized by a progressive increment of the H₁-H₂ intervals prior to failure of a ventricular response. 
The increment of conduction time, which was at first in the range of a few msec, would not be recognized in 
standard ECG recordings. This increment could increase up to 180 msec, giving rise to an obvious 
Wenckebach periodicity that would be recognized at the usual 25 mm/sec paper speed. Complete (third 
degree) IHBB was observed either in the form of paroxysmal or persistent block. Paroxysmal block occurred 
spontaneously or could be induced by a critical rate of rapid atrial pacing during early stages of second 
IHBB. Complete IHBB that developed later was persistent and usually associated with a slow 
idioventricular rhythm. The study suggests that Mobitz type II and the Wenckebach patterns of conduction 
disturbance in the His bundle are different stages of the same electrophysiological disturbance, with Mobitz 
type II block representing the initial stages of failure of impulse propagation.

Additional Indexing Words:
Anterior septal artery  Second degree atrioventricular block  His bundle stimulation
Wenckebach phenomenon  Bradycardia-dependent block  Mobitz type II block
Paroxysmal A-V block

Recent studies have provided a wealth of information on the localization of the sites of 
atrioventricular (A-V) conduction disorders in man.1-8 Very little is known, however, about the 
clinical evolution of most of these disorders. We have recently developed an experimental model for study 
of the evolution and nature of the various ischemic conduction disorders in the proximal His-Purkinje 
system following ligation of the anterior septal artery in dogs.8* Our observations may prove useful for a 
better understanding of similar disorders in man. Although any combination of conduction disorders in 
the canine proximal His-Purkinje system can follow ligation of the anterior septal artery, this report will 
deal primarily with evolution of intra-His bundle lesions.

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, into the left or right common carotid artery to the aortic root for recording His bundle activation; into the left femoral artery to the left ventricle in order to record from the proximal or distal left bundle branch; into both right and left femoral veins to the right ventricle in order to record the His bundle deflection as well as from the proximal or distal right bundle. In one half of the experiments, after placing the ligature around the anterior septal artery, the left thoracotomy was closed and the animal turned in order to expose the heart through a right thoracotomy. Two to three pairs of plunge wire electrodes (0.003 inches in diameter) were placed in the His bundle to obtain His bundle activation from proximal and distal portions. In addition to the electrograms, two (II, aVR) or more standard electrocardiographic leads were recorded. Validation of the catheter electrode and plunge wire recordings were made by pacing from the various sites, together with analysis of the QRS configuration of the paced impulse (PI) and the relationship of the paced impulse to the QRS complex (V): PI−V = 25−35 msec for His bundle pacing and PI−V = 15−20 msec for proximal right and left bundle pacing. In case of a distal bundle branch recording, the presence of a sharp rapid deflection shortly preceding V (5−15 msec) was considered satisfactory evidence that the recording originated from Purkinje tissue. Measurements were accurate up to ±3 msec at a paper speed of 200 mm/sec.

Atrial pacing was produced via a bipolar plunge wire electrode inserted in the left atrial appendage. Pacing was performed with a Grass S88 stimulator and stimulus isolation unit. Slowing or cardiac arrest induced by vagal stimulation was accomplished by delivery of 0.05 msec square wave pulse of 1 to 20 volts intensity at a frequency of 20 Hz through silver electrodes inserted into the left or right cervical vagosympathetic trunk. All records were obtained on a multichannel oscilloscopic photographic recorder (E for M DR-8) at paper speeds of 25 to 200 mm/sec with the filter frequencies of 0.1–200 Hz for ECG leads and 40–200 Hz for electrogram recordings. Some of the recordings were stored on a magnetic tape recorder (Honeywell 5600) and replayed so that selected sections could be transferred to photographic paper for detailed analysis.

Control records during sinus rhythm, vagal-induced cardiac slowing, and atrial pacing up to rates that produced the atioventricular conduction of the Wenckebach type 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 varying 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, as well as to verify the position of the plunge wires which were used to record His bundle electrical activity. In addition, measurements of interelectrode distance of the plunge wires as well as the distance between each pair of wires were done in each experiment in which plunge wire recordings were obtained. The spatial alignment of the wires to each other as judged at postmortem was always correlated with the temporal relationship of the rapid activation spike of the His bundle potential recorded by each pair of wires.

Results

In all animals the control records showed a Wenckebach type second degree A-V block localized between the sites from which the atrial and His bundle deflections were recorded at pacing rates between 240–300/min. Critical rates for producing Wenckebach periodicity proximal to the His bundle recording site 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. Dissection of the anterior septal artery per se was not accompanied by any significant trauma to the coronary vascular tree. This was shown in five experiments in which the artery was exposed by dissection and a silk ligature was placed around the vessel but was not occluded. Follow-up observations up to two hours showed no change in the surface electrocardiographic leads and in the electrograms recording proximal His-Purkinje potentials.

Thirty seconds to two minutes following ligation of the anterior septal artery, ST-T changes of variable magnitude were observed in the surface electrocardiographic leads. A few minutes later, ectopic ventricular activity developed in the form of single or multiple beats closely coupled to the sinus beats. The frequency of these arrhythmias usually reached a peak after 15–20 minutes and subsided within 30 minutes. The initial burst of ectopic activity was followed by a quiescent period for 6–12 hours succeeded by a second surge of multifocal ventricular arrhythmia which lasted for several days. The average rate of this late ventricular ectopic rhythm was usually about equal to the sinus rate and could be suppressed by pacing the atrium at a rate slightly higher than the sinus rate. The initial burst of ventricular arrhythmia was more serious and could culminate in ventricular fibrillation, but it could be suppressed by vagal-induced cardiac slowing. In spite of careful attention to the initial period of arrhythmia, six dogs succumbed to ventricular fibrillation in the first 30 min after ligation. All but one of the 39 dogs that survived the early
arrhythmic period subsequently showed varying degrees of conduction disturbances at the level of the His bundle, right and left bundle branches or any combination thereof. These cases can be broadly classified into five groups (table 1). Group I included two experiments in which isolated right bundle branch block developed. Groups IV and V that were composed of ten experiments showing bilateral bundle branch block will be reported separately. In this communication 26 experiments showing intra-His bundle block, both isolated or accompanied with bundle branch block (groups II and III respectively), are critically analyzed (table 2).

Evolution of Intra-His Bundle Block

In the control state the His bundle deflection recorded by the catheter electrode was a sharp biphasic or triphasic deflection with a duration of 10–20 msec (average 15 msec). On the other hand, the control His bundle potential, as recorded by the plunge wire, was usually a sharp biphasic deflection of shorter duration, 8–12 msec (average 10 msec). In all our experiments, the interelectrode distance of the plunge wire electrode did not exceed 2 mm. In none of the cases in this report did the placement of plunge wire electrodes result in any recognizable trauma. In half the experiments in which plunge wire electrodes were placed, the recorded His bundle potential showed a relatively smaller amplitude immediately following insertion of the wires, with the deflection getting larger 2-10 min later. In all experiments utilizing plunge wire electrodes, control recordings were obtained only after the recorded His bundle potential had been stable for at least 15 min. To show that the observed changes in the His bundle deflection after ligation of the anterior septal artery were not related to possible traumatic injury resulting from the plunge wire electrode, five control experiments were performed in which the electrodes were inserted in the usual manner but ligation of the artery was delayed for two hours. No change in the His bundle potential recordings was observed during this two hour period. Also, experiments utilizing catheter electrode recordings alone and in those in which both catheter electrodes and plunge wire recordings were obtained were not different.

The first sign of conduction disorder in the His bundle was reflected in the catheter electrode or plunge wire recordings as a decrease in the amplitude, slurring of the configuration, and increase in the duration of the deflection. These changes appeared as early as 20 min following ligation but were sometimes delayed up to 2 hours (average 90 minutes following ligation). An example of the conduction disturbance is shown in figure 1 in which a His bundle recording from a catheter electrode in the aortic root (Hb(L)) and two plunge wire recordings, Hb1 and Hb2, were obtained. The two plunge wires at postmortem examination were shown to be 2 mm apart. Panel A shows control recordings. Note the sharp configuration of the plunge wire recordings especially the Hb2. Panel B was recorded 20 min following ligation and shows decrease in the amplitude of the Hb(L) recording, and increase in the duration (from 12 to 15 msec), together with slurring and slow rise of the ascending limb of the deflection. Changes in the plunge wire recordings were less conspicuous. Panel C was recorded 45 min after ligation and shows the development of complete right bundle branch block. The Hb(L) recording has split into two parts: the first is a low amplitude deflection followed by a relatively sharp RS deflection (see the magnified section encircled). On the other hand, the plunge wire recordings showed significant changes with marked decrease of the amplitude of the deflec-

### Table 1

**Localization of Conduction Disorders in the Canine Proximal His-Purkinje System (HPS) Following Ligation of the Anterior Septal Artery in 38 Experiments**

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<th>Number of experiments</th>
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**Figure 1**

Recordings of His bundle potential by catheter electrode in the left ventricle, Hb(L) and two plunge wires (Hb1 and Hb2) showing evolution of His bundle lesion following ligation of the anterior septal artery. Panel A shows control recordings. Panels B and C were obtained 20 and 45 minutes after ligation. See text for details.
Table 2

Critical Analysis of 26 Experiments Showing Intra-His Bundle Block Following Ligation of the Anterior Septal Artery in the Dog

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*Dog sacrificed for in vitro study.

Although the block occurred between the two His deflections in the Hb(L) recording, the initial His potential was small and sometimes difficult to detect. To simplify the illustrations, only one of the plunge wire recordings in figure 1 (Hb2) is shown in figure 2. Note that the Hb2 deflection is simultaneous in timing with the first deflection of the His recording in the Hb(L) electrogram. The Hb2 electrogram shows clearly that the block is occurring below the site of recording of this His deflection. However, the Hb(L) electrogram might be misinterpreted as showing A-V nodal block because it failed to show clearly the proximal His bundle deflection.

In all experiments showing an intra-His bundle lesion with the His bundle potential split into two deflections (H1 and H2), validation of the His deflections was carried out. This is shown in figure 3, which was taken from an experiment in which catheter electrode recordings from the left and right sides of the heart were obtained (Hb(L) and Hb(R) respectively). Figure 3, panel A, was obtained 2½ hours after ligation of the anterior septal artery. The right-side catheter...
the presence of a prolonged H₁-V interval and the gradual increment of the H₁-H₂ intervals before the occurrence of sudden intra-His bundle block of the fourth atrial beat. Figure 3, panel B, shows validation of H₂ by pacing from the left side catheter electrode, giving rise to a QRS configuration similar to that of the conducted sinus beats and a PI-V (PI-R, fig. 3) interval equal to the H₂-V interval. Note the 1:1 conduction from the distal His bundle pacing site at a pacing rate was higher than normal sinus rhythm that was shown in panel A to be associated with spontaneous intra-His bundle block. In figure 3, panel C, H₁ deflection is validated by vagal stimulation. Note that the fourth atrial beat has blocked in the A-V node and is not followed by a His bundle deflection while the fifth atrial beat conducts with delay in the A-V node, giving rise to marked lengthening of the A-H₂ interval. The H₁-H₂ interval of the fifth atrial beat is shorter by 10 msec than the H₁-H₂ interval of the first three beats.

When multiple recordings of the His bundle activity were made, a more accurate localization of the site of intra-His bundle block could be made. This is shown in figure 4 taken from an experiment in which a catheter electrode recording from the left side, Hb(L) and three plunge wire recordings from proximal, middle, and lower His positions (Hb₁, Hb₂ and Hb₃, respectively) were obtained. Ninety minutes after ligation of the anterior septal artery, a constant incomplete right bundle branch block developed that progressed to a higher degree of bundle branch block when the rate was increased. At the same time, an intra-
His bundle block was demonstrated by atrial pacing at a critical rate of 218 beats/min, with change from 1:1 A-V conduction to 2:1 A-V block and with the block localized between the proximal recording sites (Hb1 and Hb2), and the distal ones (Hb(L) and Hb(R)). During 2:1 A-V conduction there is an alternation of the amplitude and a slight increase in duration of the Hb2 deflection of the blocked beats.

In this pathological setting a typical example of intra-His bundle block showed fractionation of the His bundle potential into at least two deflections followed shortly after by the occurrence of intermittent block of an atrial impulse between the two deflections. This block occurred spontaneously but could also be induced by atrial pacing at a rate faster than the sinus rate. The P-R interval, H1-V, and H2-H3 intervals remained constant in the beats preceding the blocked impulse and in the beat following it. This represented a “Mobitz type II block.” Later in the experiments, the H1-V interval lengthened gradually and the H1-H2 intervals showed gradual increment before the blocked P wave. The increment, which appears first in the order of a few msec, may gradually increase up to 180 msec giving rise to an obvious “Wenckebach periodicity” in the ECG leads. The sequence of second degree intra-His bundle block is shown in figures 5 and 6 which were obtained from the same experiment. Figure 5, panel A, was recorded 90 min after ligation of the anterior septal artery. The Hb(R) recording shows a split Hb deflection (H1 and H2). Note the presence of sinus rhythm at a constant rate with sudden A-V block of the fourth sinus impulse. The block is localized between the H1 and H2 deflections. The H1-V interval preceding the blocked beat and the one following the block are essentially constant (barely a 2 msec increment). Figure 5, panel B, was obtained 15 min later and shows atrial pacing at an almost constant rate of 162/min. There is a gradual increment of the H1-H2 intervals of 18 msec before failure of ventricular response to the sixth atrial impulse. Figure 6 was obtained 2½ hours after ligation and shows catheter electrode recordings from the right and left side of the heart (Hb(R) and Hb(L) respectively). Note the presence of a clear Wenckebach periodicity in the ECG leads and an increment of 57 msec in the H1-H2 intervals before block of the atrial impulse. The Hb(L) recording showing only the distal Hb deflection may be misinterpreted as an A-V nodal Wenckebach conduction pattern. Complete intra-His bundle block developed later in the experiment.

Complete intra-His bundle block was usually transient and in 80% of the experiments in which it occurred it reverted to 1:1 conduction within 4-8 hours following ligation. Only in three out of 18 experiments showing complete intra-His bundle block was a distal His bundle pacemaker observed. The escape beats showed a QRS configuration similar to conducted supraventricular beats and were preceded by a His bundle deflection at an interval equal to the H2-V interval. In 15 experiments an escape rhythm from the bundle branches or distal Purkinje system occurred. In these cases the QRS configuration of escape beats showed a slight or greater degree of aberration. In seven experiments, bundle branch escapes were
diagnosed because the ventricular deflection was preceded by the corresponding bundle branch potential together with a shortened H-V interval. In the remaining eight experiments, neither the His bundle deflection nor the bundle branch potentials preceded the ventricular deflection of escape beats. This suggested that their origin was in the distal Purkinje system.

In five experiments in which complete intra-His bundle block developed, 1:1 A-V conduction could be demonstrated during vagal-induced sinus bradycardia. This is shown in figure 7 which was obtained from the same experiment illustrated in figure 4. Figure 7, panel A, was obtained 2½ hours after ligation and shows the development of complete intra-His bundle block, with a slow escape rhythm at an average rate of 31 beats/min. The QRS configuration of the escape beats is narrow (42 msec) as compared to conducted sinus beats (50 msec) and the escape beats are preceded by Hb(L) and Hb₂ deflections with a shorter H-V interval (23 msec) compared to 30 msec in conducted sinus beats. This suggests an escape focus in the proximal right bundle branch system (see discussion). Note that the His bundle deflection has a reversed polarity in the Hb₂ recording (a QR pattern instead of an RS configuration during conducted sinus beats), while the Hb(L) recording shows minimal change. Figure 7, panel B, was obtained shortly after the period shown in panel A. The escape rhythm was temporarily suppressed by pacing the ventricles for 30 sec from the Hb₂ electrode (distal to the site of block) at a rate of 200/min. After termination of pacing marked sinus bradycardia was obtained by vagal stimulation which resulted in 1:1 A-V conduction at a rate of 19 beats/min which is much slower than the rate of the escape focus in panel A. Note that the conducted sinus beats still show a pattern of incomplete right bundle branch block.

In six out of 26 dogs (23%) that showed intra-His bundle lesion, the conduction disorder did not progress beyond the stage of Mobitz type II block or a Wenckebach periodicity associated with an increment of only a few msec. In these experiments, however, as in several other experiments showing intra-His bundle lesion and Mobitz type II block, paroxysmal A-V block could be induced by atrial pacing at a critical rate faster than the sinus rate. The block was constant as long as the critical pacing rate was maintained. This is illustrated in figures 8 and 9. Figure 8 was obtained two hours after ligation of the anterior septal artery, and shows catheter electrode recordings from the left and right side of the heart. Note that the catheter electrode recording from the right side depicts two Hb deflections (H₁ and H₂) while the catheter electrode...
on the left side only records the distal H₂ deflection. The H₁-V interval is prolonged (45 msec). Figure 8, panel A, illustrates a spontaneous "Mobitz type II" intra-His bundle block while panel B shows the pattern when the distal His bundle is paced from the left side catheter electrode. Figure 9 was obtained 5 minutes later. The first two beats represent 1:1 A-V conduction at a rate of 153 beats/min. Atrial pacing (PI) with a gradual increase of the rate started at the third P wave. Note the development of 2:1 intra-His bundle block followed by complete intra-His bundle block at a critical atrial cycle length of 290 msec (rate of 207/min). Paroxysmal A-V block which consisted of intermittent complete block with delayed emergence of an escape pacemaker was usually obtained at a relatively early stage after ligation, and when Mobitz type II block is demonstrated.

At a later stage, and when Wenckebach periodicity with a greater degree of increment of conduction delay was observed, rapid atrial pacing usually gave rise to an advanced type of A-V block (2:1, 3:1, or 4:1) but not to paroxysmal A-V block.

In all our observations on intra-His bundle lesions, an increase in the degree of conduction disorder could usually be obtained by shortening of the cardiac cycle (tachycardia-dependent block). In only six out of 26 observations (23%) an increase in the degree of intra-His block was observed in the first beat following a long interval (bradycardia-dependent block). This always took the form of an increase of the H₁-H₂ interval but never resulted in block of the atrial impulse. In some observations both bradycardia- and tachycardia-dependent conduction disorder in the His bundle could be demonstrated respectively in the two beats following a long pause. This is shown in figure 10 in which catheter electrode recordings from the right and left side were obtained. The Hb(R) recording demonstrates a split His bundle deflection (H₁ and H₂) while the Hb(L) recording shows only the distal Hb deflection. Note that the first conducted beat following the long interval has a long H₁-V interval (90 msec) as compared to the last two beats in the record (75 msec) which terminate shorter cardiac cycles. This is primarily due to a longer H₁-H₂ interval and reflects the presence of a bradycardia-dependent conduction disorder in the bundle of His. However, the second beat in the record shows a longer H₁-V interval of 135 msec. This is a manifestation of a tachycardia-dependent block in the His bundle for this beat terminates a short cycle which is preceded by a very long one. Analysis of the A-H₁ interval of the four beats in the record showed that the first, third, and fourth intervals are within normal limits while the second one is prolonged, which is an expected normal physiological response of A-V nodal conduction.

**Discussion**

The anatomical distribution of the His-Purkinje system in dogs and the canine blood supply is sufficiently similar to the human to make the dog an appropriate experimental model. However, there is a distinct species difference in the distribution of the septal arteries between dog and man. Also in man, lesions of the conduction system are frequently present that morphologically antedate acute infarction. Despite these differences, the animal model allows the systematic study of basic electrophysiological disturbances of conduction following acute ischemic injury of the His-Purkinje system.

Localization of the conduction disturbance in the proximal His-Purkinje system in our experiments is consistent with the anatomical distribution of the anterior septal artery in dogs. Ligation of the latter usually produces ischemia in that area of the muscular interventricular septum which contains the bundle of His and major portions of the right and left bundle branches. It is 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 high incidence of intra-His bundle lesions in this report (68%) is in accord with these studies. Intra-His bundle lesions in man have been infrequently reported in English literature. In contrast, French investigators found an incidence of intra-His lesions in 20% of patients following acute myocardial infarction and 18% to 19% of patients with chronic A-V block.

In the presence of an intra-His bundle lesion with a split His bundle deflection, validation of the latter is important especially when one of the deflections, usually the H₁, has a relatively smaller amplitude. In these cases the H₁ deflection may have to be differen-
tiated from the terminal portion of the atrial wave, or from a possible A-V nodal potential. This could be done by either rapid atrial pacing, closely coupled premature atrial beat, or by the use of vagal stimulation (fig. 3). All these procedures result in marked lengthening of the A-H interval. In cases of vagal slowing of the heart an increase in the amplitude of the H1 deflection was frequently observed with shortening of the H2-H3 interval. It is interesting to observe that the response of the H1 deflection to the above procedures is diametrically opposite to the reported response of the so-called “A-V nodal potential.”

Our findings are in agreement with other studies which suggest that the A-V nodal potential is probably not recorded by extracellular electrodes. The illustration of a bradycardia-dependent intra-His bundle block reveals a His bundle response to bradycardia similar to the bundle branch system. The concept of bradycardia-dependent intra-His bundle block may be involved in some examples of bradycardia-dependent A-V block and can provide an explanation for some observations interpreted as true supernormal conduction in the His bundle (Narula, figures 24 and 25).

Our occasional finding of an escape rhythm with a narrow QRS complex in the presence of a bundle branch block pattern in the conducted beats (fig. 7, panel A) may suggest that the escape focus is located in the same bundle branch system showing conduction delay. In this case “normalization” of the bundle branch block pattern can be explained by suggesting that the time necessary for retrograde arrival of the ectopic impulse to the other bundle branch system may be equal to the delay in orthograde conduction in the diseased bundle branch system which may result in synchronous activation of both ventricles. Recently a similar explanation has been suggested to explain premature beats with narrow QRS in the presence of bundle branch block.

Several of our observations on intra-His bundle lesions are highly pertinent for a critical appraisal of the value of His bundle recording in humans for precise localization of the site of A-V conduction disorder. The finding of a Wenckebach periodicity above the recorded His deflection does not necessarily mean an A-V nodal Wenckebach conduction; it may represent an intra-His bundle lesion with failure of detection of the proximal His deflection (fig. 6). On the other hand, the presence of an intra-His bundle Mobitz type II block with failure to record the proximal His deflection may suggest the presence of an A-V nodal Mobitz type II block (fig. 8). Several recent reports discuss the presence of a Mobitz type II block in the A-V node. In some of these reports, the disorder can be explained by the presence of a marked depression of conduction in the A-V node (advanced first degree A-V nodal block) with slight sinus arrhythmia and/or variation in the parasympathetic tone. In other reports, however, the disorder may be explained by an intra-His bundle lesion with failure of recording of a proximal His bundle deflection. In the presence of an intra-His bundle block associated with a bundle branch block, the failure to record a proximal His bundle deflection may falsely impart a favorable prognosis because the recorded H-V interval — which is in fact an H2-V interval — will usually be of normal duration. This situation may explain some studies reporting normal H-V intervals in patients with bundle branch block who developed serious disturbances of A-V conduction. In these cases failure to recognize an already existing intra-His bundle lesion is a possibility. On the other hand, the recording of a proximal His bundle deflection with failure to detect a distal potential in the presence of an intra-His bundle lesion associated with bundle branch block may be taken to represent a bilateral bundle branch block because the H2-V interval will usually be prolonged. This trend has been recognized in several reports showing conventional electrocardiographic recordings as well as His bundle electrograms.

In these cases the presence of an intra-His bundle Wenckebach conduction disturbance may be attributed to a Wenckebach periodicity in the remaining functioning bundle branch. Furthermore, physiological studies of the refractory periods of the His-Purkinje system in similar cases is usually considered to represent the functional capacity of the other bundle branch or fascicle (in the presence of right bundle branch block with left axis deviation, for example) without considering the possibility that the limiting factor may be the conduction in the His bundle. In conclusion, this study suggests that some examples of intra-His bundle lesions in humans may be misinterpreted as occurring in either the A-V node or bundle branches. Probably a guarded interpretation of the His bundle electrogram stating that the conduction disorder may be localized above or below the “site of recording” of the His bundle potential should be always exercised.

Appraisal of Intra-His Bundle Blocks

The present study has shown a characteristic pattern for evolution of intra-His bundle lesions following acute myocardial ischemia. These always started with fractionation of the His bundle potential associated with slurring, loss of the sharp configuration, decrease in the amplitude and increase of the
duration of the potential before it is split into at least two frequently distinct deflections. This process is always associated with progressive increase of the H-V (or H₁-V) interval and represents “first degree intra-His bundle block.” Fractionation of the His bundle electrogram and the later development of an apparent isoelectric interval between the split His bundle potential probably reflects a relatively localized significant slowing of conduction in a certain portion of the His bundle with partially depolarized Purkinje fibers generating small action potentials. The filter frequency used in recording of the His bundle electrogram may play a role. The latter, however, has not been systematically analyzed in this study.

Following first degree intra-His bundle block a second degree block characterized by intermittent failure of a ventricular response develops either spontaneously or in response to rapid atrial pacing. The first “stage” of “second degree intra-His bundle block” is the occurrence of a ” Mobitz type II” pattern characterized by intermittent block of an atrial impulse with a “constant” P-R, H₁-V and H₁-H₂ intervals of the beats preceding the block and in the beat following it. Although no measurable increment in conduction time was observed at this stage, the limitations of both the measurement error and our available recording technique may preclude detection of minimal increments. This stage merges imperceptibly into the second “stage” of second degree intra-His bundle block which is characterized by a gradual increment of intra-His bundle conduction time (H₁-H₂) before failure of a ventricular response (a Wenckebach periodicity). The increment which is first in the range of a few msec may gradually increase up to 180 msec giving rise to an obvious Wenckebach periodicity at the usual 25 mm/sec paper speed. Our study suggests that Mobitz type II and the Wenckebach type of conduction in the His bundle are different stages of the same electrophysiological disturbance, with Mobitz type II representing the initial stages of decrement of conduction before failure of impulse propagation.

Complete (third degree) intra-His bundle block was observed either in the form of a paroxysmal or persistent block. Paroxysmal block characterized by repetitive block of a large number of atrial impulses with the delayed emergence of an escape rhythm can occur spontaneously (fig. 2) but was usually induced by a critical rate of rapid atrial pacing during the early stages of second degree intra-His bundle block (fig. 9). Once a higher degree of Wenckebach increment developed, rapid atrial pacing usually resulted in a higher degree of block rather than intermittent complete A-V block. Persistent complete intra-His bundle block usually developed spontaneously later in the experiment and took the form of complete A-V dissociation between the atrial rhythm and a fairly regular slow escape rhythm. That complete A-V dissociation does not necessarily assure that permanent interruption of the conduction is present could be occasionally demonstrated by the resumption of 1:1 conduction during vagal induced slowing of the sinus rate (fig. 7). This is consistent with the electrophysiological definition of complete A-V dissociation as the presence of an escape rhythm at an R-R interval shorter than the effective refractory period of the A-V pathway.44

Our understanding of the electrophysiological basis of A-V conduction disorders is far from complete. The experimental model utilized in this study offers a reasonable approach for further in vivo and in vitro studies on the subject.

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INTRA-HIS BUNDLE BLOCK


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