Catheter-Induced Intra-Hisian and Intrafascicular Block During Recording of His Bundle Electrograms

A Report of Two Cases

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SUMMARY
Mechanical injury of the atrioventricular (A-V) node, His bundle, and right bundle branch is a potential complication of His bundle recording. In the presence of left bundle branch block, catheter-induced injury of the right bundle branch may cause prolongation of A-V conduction time or even complete A-V block. Two cases are reported in which catheter-induced transient high grade Mobitz II A-V block, acute reversible prolongation of infranodal conduction time with probable intra-Hisian conduction delay, and persistent (6 hours duration) complete A-V block occurred in patients with left bundle branch block. The observations made indicate that A-V conduction times and the appearance of "split His" potentials may be influenced by the recording catheter itself, and suggest that the operators be prepared to institute immediate right ventricular pacing when His bundle recordings are obtained in patients with left bundle branch block.

Additional Indexing Words:
Split His potentials Complete atrioventricular block Mobitz II atrioventricular block Left bundle branch block

His bundle electrocardiography has greatly advanced the understanding of cardiac conduction abnormalities and arrhythmias. Recently, several excellent critical reviews detailed the limitations of this technique, but little mention was made of complications or of conduction abnormalities induced by the electrode catheter used for these studies. However, the occurrence of certain problems is theoretically predictable since the catheter is purposely positioned in close proximity to the His bundle and right bundle branch. In the presence of left bundle branch block (LBBB), for example, trauma to the right bundle branch (RBB) can result in atrioventricular (A-V) conduction disturbances. This report describes the occurrence of advanced Mobitz II A-V block, complete A-V block, and acute reversible prolongation in infranodal conduction times with probable intra-Hisian conduction delay during catheter recording of His bundle electrograms in patients with LBBB.

Materials and Methods
His bundle electrograms were obtained in two patients with LBBB as part of an interhospital prospective study assessing the natural history of patients with chronic intraventricular conduction disturbances. The study was approved by the University of California, San Francisco, Committee on Human Experimentation and informed consent was obtained prior to the study.

His bundle electrograms were obtained using standard techniques. In brief, a multipolar electrode catheter is inserted into the femoral vein and positioned across the tricuspid valve. The electrodes are connected to a switching box connected to an Electronics for Medicine (DR-12) recorder with filter settings at 40-500 cps. The catheter is withdrawn across the tricuspid valve until a discrete spike is displayed between prominent atrial and ventricular electrograms. His bundle and intra-atrial electrograms as well as scalar X,
Y, and Z (or inverse Z) leads of the Frank orthogonal lead system are recorded simultaneously. Atrio-ventricular nodal conduction time (A-H) is measured from the initial rapid deflection of the atrial electrogram to the initial deflection of the His bundle depolarization, while His-Purkinje conduction time (H-Q) is measured from the initial His deflection to the earliest onset of ventricular activation in the surface electrograms. In our laboratories, the normal intervals are 70-120 msec for A-H and 35-55 msec for H-Q.

**Case Reports**

**Patient No. 1**

An 81-year-old man who had had two recent near-syncopal attacks was studied. Serial 12 lead electrocardiographic tracings as well as two days of continuous monitoring in a coronary care unit showed LBBB with a QRS duration of 130 msec, a P–R interval of 170 msec, and no premature beats or periods of second or third degree A-V block. During the study, control recordings revealed a normal A–H interval of 90 msec, a prolonged H–Q interval of 60 msec, and no premature beats. Suddenly, a 14 sec period of Mobitz II A-V block occurred, preceded by premature beats originating distal to the His bundle (fig. 1A). The LBBB pattern of the premature beats suggested that their origin was in the right ventricle. The conduction ratio was 4:1 in the first conducted beat of this series and 3:1 in all other conducted beats. The A-H interval of 90 msec was unchanged during Mobitz II block, but the H-Q of conducted beats was markedly prolonged to 280 msec in the first and 180 msec in the remaining conducted beats. The QRS complex changed from LBBB with normal QRS axis to RBBB with left axis deviation (left anterior fascicular block pattern), indicating that conduction was now occurring through the left posterior

![Figure 1](http://circ.ahajournals.org/)

A) Continuous His bundle electrograms obtained in patient no. 1. Two sinus beats with LBBB configuration and normal A-H but slightly prolonged H-Q intervals are followed by a ventricular premature beat (PB); then a somewhat aberrantly conducted sinus beat (AVC) (or fusion beat) followed by another PB. These PBs are not preceded by His deflections, indicating their origin distal to the His bundle. Following the second PB, a 14 sec period of Mobitz II A-V block occurs in which the QRS complex configuration and axis have changed. B) This strip shows the reappearance of the initial LBBB configuration in patient no. 1. A-H = A-V nodal conduction time, H-Q = infranodal conduction time, X, Y, 1/Z = leads of Frank orthogonal lead system, HBE = His bundle electrogram, and IAE = intra-atrial electrogram obtained from a proximal electrode pair. In all illustrations time lines are 1 sec apart and paper speed is 100 mm/sec.

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A-V block, which was capable of conducting supraventricular impulses in a 3:1 ratio. Thus, the “complete” LBBB seen in the control tracings probably represents complete block in the anterior division of the LBB and functional (rate related) block in the left posterior fascicle.

It is conceivable that when the right bundle is intact, the posterior fascicle is kept refractory because of concealed retrograde conduction; damage to the right bundle branch might, therefore, allow for conduction through the left posterior fascicle. The initial LBBB configuration reappeared in a period of 2:1 Mobitz II A-V block (fig. IB), but the markedly prolonged H-Q of 150 msec in this beat indicated that although the RBB was again able to conduct, it was doing so at a much slower conduction velocity than at the onset of the study. Although the catheter was not manipulated during the recordings, spontaneous motion of the catheter with each heart beat was observed fluoroscopically and probably accounted for both the premature ventricular beats and injury to the RBB.

Shortly after return to baseline conduction, again without catheter manipulation, a premature QRS complex originating in either the distal His bundle or the proximal RBB initiated a period of H-Q prolongation in which a new deflection (H') appeared in the H-Q interval (fig. 2). The H-Q lengthened from 60 to 90 msec in the initial beat, but over a period of several beats the H-Q interval returned to baseline values pari passu the gradual fusion of H' with H. The brief duration of this phenomenon precluded validation of these spikes as being Hisian in origin. This sequence suggested that trauma to the His bundle induced by the catheter resulted in decreased conduction velocity from the His bundle to the ventricles possibly due to delayed conduction within the His bundle. At the termination of the study the H-Q interval had returned to its control value. The patient refused permanent pacemaker insertion, and denies further neurologic symptoms.

**Patient No. 2**

A 56 year old man with ischemic heart disease who had three episodes of palpitations, lightheadedness, and syncope over a three month period was studied.

![Figure 2](http://circ.ahajournals.org/)

A) This strip obtained in patient no. 1 shows a premature beat (PB) with configuration similar to that of the initial sinus beat but with a shorter H-Q interval, indicating its origin in the distal His bundle or proximal RBB. Following this beat, the A-H interval remains unchanged but the H-Q becomes more prolonged and another deflection (H') appears in the H-Q interval. B) Two continuous strips obtained in patient no. 1 show a similar sequence but in addition display the gradual shortening of the abruptly prolonged H-Q interval to previous values pari passu the gradual fusion of H' with H.
Ventricular tachycardia was documented during each of these occasions. Electrocardiograms obtained during the previous two years revealed LBBB with a QRS duration of 170 msec and a prolonged P-R interval of 240 msec. Initial His bundle recordings revealed prolongation of the A-H time to 146 msec and the H-Q time to 61 msec (fig. 3, upper strip). When the catheter was manipulated to obtain a larger His deflection, transient Mobitz II A-V block was observed but not recorded and was followed by the onset of complete A-V block (fig. 3, lower strip). When a very slow idioventricular rhythm followed, the electrode catheter was immediately positioned into the apex of the right ventricle and temporary demand pacing initiated. Repeated attempts to discontinue pacing resulted in prolonged ventricular asystole followed by a slow ventricular escape rhythm. Therefore, the study was terminated and the patient was transferred to the coronary care unit for continuous electrocardiographic monitoring. Six hours later 1:1 A-V conduction reappeared, with the same P-R interval and QRS configuration and duration as before the study. Pacing was discontinued and the patient recovered without complications. A permanent transvenous demand pacemaker was implanted one week later and the patient remained asymptomatic for three months until he died suddenly while eating lunch.

**Figure 3**

Noncontinuous His bundle electrogram strips obtained in patient no. 2. Top strip reveals control findings of LBBB and prolongation of P-R, A-H, and H-Q intervals, while bottom strip shows complete A-V block occurring below the His bundle and a slow idioventricular rhythm.

**Discussion**

The incidence of complete A-V block occurring during cardiac catheterization in adults is quite low (0.01%). Almost all of the reported cases in adults occurred during right heart catheterization of patients with pre-existing LBBB and were transient. However, two instances of chronic complete A-V block occurring in this setting have been reported. Current electrophysiologic and anatomic evidence suggests that in the presence of LBBB (and in the absence of accessory A-V pathways) a supraventricular impulse can be conducted to ventricular tissue only by traversing the RBB, which is located quite superficially in the right side of the interventricular septum. Thus, in patients with LBBB, injury to the RBB may result in delayed or blocked A-V conduction. It is not surprising that conduction abnormalities might be induced during His bundle electrocardiography, when the recording catheter is purposely manipulated in the area of the His bundle and RBB in patients who may have pre-existing cardiac conduc-
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A-V bundle potentials report instances of transient A-V block were encountered. Although atrioventricular block occurring with right heart catheterization in patients with LBBB is a well recognized entity, the possibility of spontaneous A-V block (unrelated to catheterization) cannot be excluded since both of our patients had had presyncopeal symptoms. Both patients, however, underwent prolonged electrocardiographic monitoring in a coronary care unit and high degree of complete A-V block was never documented. It would seem highly unlikely that high degree of complete A-V block occurred spontaneously at the exact instant of His bundle recording. In one of our patients, high grade Mobitz II A-V block was immediately preceded by a ventricular premature beat presumably of right ventricular origin (fig. 1). Previous reports of A-V block occurring during right heart catheterization have seldom focused on the beat preceding the A-V block, but in two patients with LBBB mention is made of the occurrence of ventricular premature beats (of unspecified ventricular origin) just before the onset of complete A-V block.

Massumi (unpublished observations) suggested that transient A-V block after a premature ventricular beat results from decreased coronary perfusion following the premature beat, the subsequent ischemia producing block in the A-V conduction pathways. Such a phenomenon would, however, be expected to perpetuate itself unless the slow heart rate occasioned a favorable myocardial oxygen balance such that ischemia would in fact be relieved. While this mechanism might explain the transient Mobitz II block in one of our patients, it is unlikely to have caused the prolonged complete A-V block, which persisted for six hours, in the other patient. More likely, premature ventricular beats induced by the catheter caused mechanical injury of the RBB by the catheter and resulted in blocked conduction in this fascicle. Similarly, the premature distal His (or proximal RBB) beats preceding acute prolongation of the H-Q interval and the "split His" potentials were most likely effected by direct catheter contact with and damage to the His bundle.

An alternative explanation for the appearance of an additional deflection following the His bundle depolarization is that it represents the proximal RBB depolarization. Conceivably, catheter-induced damage to the RBB might have resulted in delayed conduction both proximal and distal to the RBB potential. The latter explanation, although consonant with the prior description of catheter-induced complete RBBB, appears to be unlikely for several reasons. Repeated attempts to record RBB potentials during the initial phase of the study were unsuccessful and it would seem unlikely for this potential to fortuitously appear only during abrupt prolongation of the H-Q interval. In addition, since there was no apparent change in catheter position, delayed conduction in the proximal RBB should have made recording of this potential even less likely. Finally, the gradual merging of both potentials into a single deflection of short duration is not consistent with the known normal conduction times from the His bundle to the proximal RBB.

Although a split His potential is generally interpreted as delayed conduction through an organically diseased common bundle, our observations suggest that such potentials may be produced iatrogenically by the catheter. The findings reported herein of acute reversible delays in infranodal conduction in a patient with LBBB are unique and suggest that catheter-induced injury may prolong H-Q time without causing complete RBBB. If, in fact, catheter-induced injury can prolong intra-Hisian conduction time, acute H-Q prolongations might also occur in His bundle studies of A-V conduction in patients without LBBB.

The observations presented raise important practical points relative to catheter recording of His bundle electrograms. First, equipment for ventricular pacing utilizing the recording catheter should always be readily available when His bundle electrographic studies are performed in patients with LBBB and by inference when LBBB electrograms are recorded in patients with RBBB; and isoproterenol should be available in the event that placement of the recording catheter into the right ventricle proves difficult. Secondly, the method of recording His bundle deflections may influence the measurements obtained. The occurrence of transient RBBB (and therefore of complete or high grade A-V block) during right heart catheterization of patients with LBBB should suggest the probability of catheter-induced injury to the RBB. Such injury may, indeed, have been responsible for the recently reported totally unexpected instance of acute onset of complete A-V block following
lidocaine administration in a patient with LBBB during His bundle recording. Less well appreciated is the finding that lesser degrees of injury to the RBB may result in prolongation of the H-Q interval without actual cessation of A-V conduction. During the study of patient no. 1, for example, a wide range of H-Q intervals and split His potentials were recorded even in the presence of 1:1 A-V conduction. Therefore, caution should be exercised in attributing acute changes in A-V conduction to spontaneously occurring or drug-induced events when intracardiac recording techniques are used.

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References

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