Transient and Persistent Atrial Standstill with His Bundle Lesions

Electrophysiologic and Pathologic Correlations

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SUMMARY

Electrophysiologic studies, including His bundle recording and atrial pacing (AP), were performed in one patient with transient (TAS) and another with persistent atrial standstill (PAS). Both subsequently expired, and postmortem examinations with serial sections of the conduction system were obtained. The patient with TAS had left bundle-branch (LBB) block, a P-H interval of 140 msec (normal, 80-140 msec), an H-Q of 40 msec (normal, 35-55 msec), and an AP threshold of 1.2 ma. Postmortem examination revealed total left circumflex occlusion proximal to the sinoatrial (SA) nodal artery takeoff, SA nodal arteriolosclerosis without infarction, and left-sided cardiac skeletal sclerosis (LSCSS) disrupting the penetrating portion of the His bundle and the LBB. Intracardiac recordings in the patient with PAS showed absent atrial activity and an H-Q of 60 msec. The atria were inexcitable with AP up to 15 ma at multiple sites. Postmortem examination revealed a previously undescribed atrial disease characterized by arteriolosclerosis, fibroelastosis, fatty infiltration, and vacuolar degeneration of muscle cells, with only moderate SA and A-V nodal involvement. LSCSS was present, disrupting the branching portion of the His bundle. Thus, TAS reflected SA nodal ischemia without infarction; PAS reflected a new atrial degenerative disease; H-Q was normal with a lesion in the penetrating portion of the His bundle and was prolonged with a similar lesion in the branching portion.

Additional Indexing Words:
Bilateral bundle-branch block  His-Purkinje system  Left bundle-branch block
Sclerosis of the cardiac skeleton  Sinoatrial node

A TRIAL standstill is an arrhythmia characterized by absence of P waves and by a regular escape rhythm of supraventricular origin.1-4 Atrial standstill is usually transient, occurring with digitalis or quinidine intoxication, hypoxia, hyperkalemia, or myocardial infarction.1-7 Persistent atrial standstill is rare, with only a few reported cases.8-13 Postmortem observations have been reported in several cases of acute standstill and in only one case of persistent standstill.9

In the present report we describe two patients, one with transient and the other with persistent atrial standstill. Electrophysiologic studies, including atrial pacing and His bundle recordings, were performed in both cases. Complete cardiac postmortem examinations were performed with serial histologic sections of the specialized conduction system. Electrophysiologic and pathologic correlations were made regarding atrial standstill, including the reporting of a previously undescribed
degenerative atrial disease in the patient with persistent standstill. In addition, observations were made regarding the significance of His bundle recordings in the diagnosis of His bundle lesions.

**Materials and Methods**

His bundle electrograms were recorded with 7F tripolar catheters passed percutaneously from the right femoral vein.\(^{14,16}\) Recordings were made on multichannel oscilloscopic photographic recorders\(^*\) at paper speeds of 50, 100, and 200 mm/sec. P-H was the interval from the onset of the P wave to the His bundle electrogram (normal, 80–140 msec), and H-Q (H-V) was the interval from the His bundle electrogram to the onset of the QRS complex (normal, 35–55 msec).\(^{15}\) Atrial pacing was attempted in both patients, with bipolar catheters.

The method of histologic study of the heart was as follows: In both cases, the sinoatrial (SA) and atrioventricular (A-V) nodes and their approaches, the A-V bundle and bundle branches up to the level of the moderator band, were serially sectioned, and every tenth section was retained. In case 1 the roofs of both atria were serially sectioned, and every twentieth section was retained. In case 2 the roofs of both atria were serially sectioned, and every twentieth section was retained. In case 2 the atrial septum in the region of the coronary sinus and the roof of the right atrium carrying the blood supply to the SA node and the atrial septum at the junction of both atrial roofs were serially sectioned, and every twentieth section was retained. In case 2, the roof of the left atrium was also serially sectioned, and every fortieth section was retained. In both cases the remainder of the heart was cut into blocks, and two sections were taken from each block. Alternate sections were stained with hematoxylin-eosin and Weigert-van Gieson stains. In case 1, 1,304 sections were studied and in case 2, 1,582 sections. Additional sections in case 2 were stained with crystal violet stain for metachromasia, and the green birefringence test was performed after congo red stain for amyloid.

The method of studying the conduction system has previously been described.\(^{18}\)

**Report of Cases**

**Case 1**

**Clinical Summary**

The patient’s first admission was from February 28, 1970 to March 11, 1970. The patient was a 69-year-old man, admitted to Cook County Hospital with a 2-week history of increasing dyspnea, orthopnea, and recurrent dizziness. There was a history of syncope 2 months and also 1 day prior to admission.

Physical examination revealed a blood pressure of 120/70 and a pulse of 112 beats/min. Neck veins were distented at 45°. Cardiac examination revealed a left parasternal lift, a soft first sound, a paradoxically split second sound, and a prominent third and fourth heart sound. A grade II/VI holosystolic murmur was heard at the cardiac apex. There were bilateral basal rales.

Admission electrocardiograms revealed sinus tachycardia at 115 beats/min, a P-R interval of 0.17 sec, and complete left bundle-branch block (fig. 1). Twelve hours after admission, the patient had a sudden drop in pulse rate to 30 beats/min. Electrocardiograms revealed the absence of sinus P waves and a slow escape rhythm with wide QRS identical to those of his previously conducted beats (fig. 2A). Intraatrial electrograms were not recorded at this time, so that complete absence of atrial activity was not documented.

The arrhythmia could thus be interpreted as

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Central progressive transvenous pacemaker in and recurrent to control necessary pacing.'7

Electrocardiographic artifact pr junctional A-V pacing sinus within 3 min was atropine administered intravenously (monitor lead). (C) February 29, 1970. Atrial pacing from the coronary sinus with 1:1 A-V conduction at a paced rate of 88 beats/min (lead Vg). (D) March 9, 1970. Sudden development of complete A-V block despite continued atrial capture from the coronary sinus pacemaker (paced rate of 136 beats/min) (monitor lead). (E) March 9, 1970. 4:1 A-V block during coronary sinus pacing (paced rate of 115 beats/min) (monitor lead).

either atrial standstill or sinus arrest with A-V junctional escape rhythm. One milligram of atropine was administered intravenously, and within 3 min sinus rhythm returned (fig. 2B), at a rate of 55 beats/min. An emergency transvenous pacemaker was passed to the midportion of the coronary sinus for atrial pacing.17 The pacemaker was set in the demand mode at 80 beats/min, and successful atrial pacing was obtained (fig. 2C).

During the next 7 days, the patient remained in paced coronary sinus rhythm. Frequent increases in pacing rate were necessary to control ventricular irritability. The patient's clinical status deteriorated with progressive central nervous system depression and recurrent episodes of dyspnea. On March 9, 1970, the patient developed sudden episodes of both complete and 4:1 A-V block despite continued atrial capture from the coronary sinus pacemaker (fig. 2, D and E). A transvenous pacemaker was passed to the apex of the right ventricle, and the coronary sinus pacemaker was withdrawn. Ventricular pacing was established.

During the next 48 hr the patient developed progressive hypotension and oliguria. On March 11, 1970, the patient had a cardiac arrest and expired.

Electrophysiologic Studies

Studies were performed at the time of initial pacemaker insertion. During the study the patient was in normal sinus rhythm at a rate of 90 beats/min with left bundle-branch block. The P-H interval was 140 msec, and HQ was 40 msec (fig. 3A).

Atrial pacing was accomplished with a catheter passed from the right external jugular vein and positioned at the lateral wall of the right atrium. Atrial pacing threshold was 1.2 ma. The atria were paced at increasing heart rates from 100–150 beats/min. P-H prolongation was noted as heart rate was increased. 1:1 A-V conduction was maintained up to a rate of 150 beats/min, at which time Wenckebach periods were noted with block occurring proximal to the recording site of the His bundle (fig. 3B). H-Q was 40 msec at all paced rates. Block distal to the His bundle was not noted.

Thus, at the time of study, atrial excitability appeared normal as manifested by normal pacing threshold. A-V and intraventricular conduction were characterized by normal intervals and responses to atrial pacing.

Autopsy Findings

Gross Examination of the Heart. The heart weighed 495 g. All chambers were thickened, and the left side was slightly increased in size. There was a large zone of calcification in the aortic leaflet of the mitral valve extending to the aortic mitral annulus and the aortic valve, where there was a Mönckeberg type of calcification as well as calcification of the noduli of Arantius. The area of calcification...
Electrophysiologic studies in patient 1. (A) Simultaneous His bundle electrogram (HBE) and electrocardiogram (ECG) during normal sinus rhythm (NSR) $P = P$ wave; $R = QRS; H =$ His bundle electrogram. Note P-H interval of 140 msec at the upper limit of normal. The H-Q (H-V) is normal at 40 msec. (B) Atrial pacing (AP) at 150 beats/min with development of Wenckebach periods. $P_i =$ atrial pacing spikes; $A =$ atrial electrograms. Note the progressive $P_i$-H prolongation (intervals listed in msec). There is block proximal to the His bundle following the fifth $P_i$ (the cycle with the arrow).

Microscopic Examination of the Heart. Arteriosclerosis was prominent in the SA node (fig. 4). There was slight infiltration of mononuclear cells. Arteriolosclerosis was also prominent in the approaches to the SA node. There was a slight-to-moderate infiltration of mononuclear cells with occasional small organizing scars. Chronic pericarditis was slight. Arteriolosclerosis as well as focal areas of fibrosis and fibroelastosis were evident in the approaches to the A-V node, involving most of these approaches (fig. 5). The beginning of the A-V node showed considerable fibroelastosis, with some arteriolosclerosis. This became less marked more distally. In the beginning, the penetrating portion of the A-V bundle showed considerable fibroelastosis and some arteriolosclerosis. As it impinged upon the calcific mass the fibroelastosis increased, with marked but not complete replacement of the bundle (fig. 6). Still more distally, it was compressed only in its lower part. There was moderate fibrosis of the branching portion of the A-V bundle. Most of the main left bundle branch (LBB) had lost its connection with the A-V bundle (fig. 7), related to fibroelastosis produced by compression of the calcific mass. There was a recent infarct of the radiations. The right bundle branch (RBB) came off the beginning of the branching portion of the A-V bundle. Its first part showed considerable fibroelastosis, and the second part was surrounded by fat tissue and empty spaces (fig. 8). There were no changes.
in the ramus ostii cavae superioris. However, it emerged from the left circumflex artery distal to its obstruction. The ramus septi fibrosi was slightly narrowed. In the atria, there were occasional minute foci of organizing necrosis. In the right ventricle, there were occasional small foci of organizing infarct. In the left ventricle, a recent infarction (less than 12 hr old) was present subendocardially in the septal, anterior, posterior, and lateral walls. This was superimposed on a small older infarct.

**Case 2**

**Clinical Summary**

The patient's first admission was from April 5, 1966 to May 6, 1966. The patient was a 73-year-old woman admitted to Cook County Hospital because of upper gastrointestinal bleeding. The patient had a history of hypertension. Physical examination revealed a blood pressure of 122/80 and a pulse of 72 beats/min, which was irregular. On cardiac examination there was a grade II/VI holosystolic murmur and an audible third sound. An electrocardiogram revealed atrial fibrillation with fine fibrillatory waves and a ventricular response averaging 72 beats/min (fig. 9). Mean QRS axis in the frontal plane was +75°. There was a QS complex in V1 and absent septal Q waves in V5 and V6. The gastrointestinal bleeding subsided spontaneously, and no bleeding site was demonstrated.

The patient's second admission was from June 10, 1967 to July 27, 1967. The patient was admitted with a 1-month history of congestive heart failure. The results of the physical examination were similar to those of the first admission with the additional findings of marked bradycardia, neck vein distension, and peripheral edema. Electrocardiograms revealed very fine atrial fibrillatory waves with high grade A-V block (fig. 10). The frontal axis had shifted to the right to +120°.
There was no clinical evidence of pulmonary embolization.

The patient was initially treated with temporary transvenous ventricular pacing with amelioration of congestive failure. Because of persistent bradycardia, a permanent transvenous ventricular pacemaker was implanted. The patient was discharged with no cardiac medications.

The patient's third admission was from March 24, 1969 to April 21, 1969. The patient was admitted with a 1-week history of dizziness. Electrocardiograms revealed a runaway pacemaker with subthreshold pulses emitted at approximately 1,200 beats/min. Because multiple pacing spikes deformed the ECG it was impossible to define the supraventricular mechanism, which may have been either atrial fibrillation or standstill (fig. 11).

The QRS was narrow and regular at a rate of 38 beats/min. The axis had reverted to +60°. Deep T-wave inversions were present in leads I, II, aV_L, and V_2-V_6. A new fixed-rate pulse generator was implanted, and ventricular pacing was restored.

The patient's fourth admission was from December 31, 1969 to January 10, 1970. The patient was seen in clinic on December 31 complaining of diaphragmatic pacing of 1 week's duration. Electrocardiograms revealed complete ventricular capture. Chest X-ray revealed the tip of the pacing catheter to be at the cardiac apex. Myocardial perforation or penetration was suspected, and because of the danger of pacing failure a temporary catheter pacemaker was passed to the right ventricular apex to be available for emergency pacing. On January 5, 1970, a new permanent demand

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Figure 5

Case 1. Approaches to the A-V node, showing focus of fibroelastosis of the myocardium. (Weigert-van Gieson stain, × 45.)
pacemaker was implanted with a new set of electrodes. The old fixed-rate generator was removed, but the old catheter was left in place. Difficulty was encountered in achieving adequate pacing with the new unit because of high pacing threshold (4.5 mA).

On January 6, 1970, loss of pacing was noted with the new unit. Electrocardiograms at this time revealed pacing spikes, but no ventricular capture. The supraventricular mechanism was atrial standstill with complete absence of atrial activity (fig. 12). The escape rhythm was A-V junctional with narrow QRS at a rate of 50 beats/min. The frontal axis was +75°. This rhythm persisted for the rest of the hospital course. An R wave was now present in V1, but septal Q waves were still absent in V5 and V6.

On January 7, 1970, a new temporary pacemaker catheter was passed to the right ventricular apex. Again, difficulty was encountered in achieving adequate ventricular pacing. The recently implanted demand unit was not inactivated at this time. It was planned to implant an epicardial pacemaker within the next few days because of the difficulty in achieving adequate transvenous pacing. On January 10, 1970, the patient was discovered to be diaphoretic and hypotensive. Electrocardiograms showed ventricular tachycardia, which progressed to ventricular fibrillation. Despite resuscitative efforts, the patient expired.
Electrophysiologic Studies

Electrophysiologic studies were performed at the time of pacemaker insertion on January 7, 1970. Right atrial and His bundle electrograms revealed complete absence of atrial activity. An escape rhythm at a heart rate of 50 beats/min was present, characterized by narrow QRS. Each ventricular electrogram was preceded by a His bundle spike with a duration of 35 msec and an H-Q interval of 60 msec (fig. 13).

Right atrial pacing was attempted at several atrial sites, including the high and low lateral right atrium, the high and low right atrial septum, and the coronary sinus. Pacing was attempted at rates between 60 and 100 beats/min. with stimulus strengths of 2–15 ma. There was complete inability to capture the atrium.

It was concluded that the patient had atrial standstill characterized by lack of atrial electrical activity and atrial inexcitability. The escape mechanism was that of a His bundle rhythm. The His bundle potential was widened and H-Q intervals were slightly prolonged, suggestive of delay in the distal His bundle.

Autopsy Findings

Gross Examination of the Heart. The heart was enlarged, weighing 524 g. There was hypertrophy and enlargement of all chambers. The right coronary artery gave off the ramus ostii cavae superioris and the ramus septi fibrosi. There was minimal evidence of sclerosis and no narrowing of the coronary arteries.
Electrocardiogram from patient 2 (first admission) showing atrial fibrillation with fine fibrillatory activity. There is a QS in V₁ and absent Q waves in V₅ and V₆.

The pacing catheter was imbedded in the myocardium of the right ventricular apex.

Microscopic Examination of the Heart. In the SA node, there was a moderate infiltration of mononuclear cells and eosinophils throughout with degenerative changes in the collagen. Some of the arterioles showed sclerotic changes (fig. 14). There was slight fatty infiltration. The node was somewhat stretched and flattened out.

Throughout both atria and the atrial septum, the musculature showed marked fibrosis, fibroelastosis, and fatty infiltration (fig. 15, left). In addition, the atrial muscle cells showed marked vacuolization (fig. 15). In some areas they were engulfed in hyalinized connective tissue (fig. 15, right). The distal portion of the left atrium, the myocardium, and endocardium were infiltrated with mononuclear cells. No amyloid was found in the atrial musculature. In the approaches to the SA and A-V nodes, the changes were similar to those found in the remainder of the atria. Arteriolosclerosis was frequently seen. The ramus ostii cavae superioris and ramus septi fibrosi were not narrowed. The A-V node lay adjacent to the atrioventricular part of the pars membranacea. Its distal part lay side-by-side with the beginning of the penetrating portion of the A-V bundle. The node showed slight fibrosis with a slight infiltration of mononuclear cells.

The penetrating portion of the A-V bundle penetrated the atrioventricular part of the pars membranacea. In its beginning it showed slight-to-moderate fibrosis.

In the branching portion of the A-V bundle, there was moderate dissolution of tissue with space formation and fibrosis (fig. 16). The branching portion was originally draped over the summit of the ventricular septum. More distally it veered toward the right side of the septum. The bifurcation showed moderate fibroelastosis. One arteriole was markedly thickened and narrowed. There was moderate to severe fibroelastosis with replacement and space formation of the junction of the posterior part of the main LBB with the branching bundle (fig. 17). The anterior part of the main LBB and the radiations showed moderate fibroelastosis. The peripheral fascicles showed a slight infiltration of mononu-

Electrocardiogram from patient 2 (second admission) showing atrial fibrillation with high grade A-V block. Note that right axis deviation is present. In some of the leads the baseline is flat, suggesting atrial standstill.
ATRIAL STANDSTILL

Figure 11

Electrocardiographic rhythm strips (leads I, II, and III) taken from patient 2 during the third admission, showing runaway pacemaker with about 1,500 subthreshold pacing spikes per min. Note that the axis has reverted to normal. The supraventricular mechanism cannot be determined because of the multiple spikes deforming the electrocardiogram, but may be either atrial fibrillation with advanced A-V block or atrial standstill.

Figure 12

Electrocardiogram taken during fourth admission (patient 2) showing persistent atrial standstill. Note that atrial activity is completely absent and that an R wave is now recorded in lead V₁. The previously implanted demand pacemaker is sensing and firing, but there are no ventricular captures.

clear cells with fibrosis. There was slight fibrosis of the first part of the right bundle branch, with space formation on the periphery. The second portion showed moderate acute degenerative changes with slight fibrosis. The third part showed slight to moderate
Atrial Standstill

Electrophysiologic studies in patient 2. (Top) Simultaneous electrocardiogram and His bundle electrogram showing absence of atrial activity. An A-V junctional rhythm is present, characterized by a narrow QRS preceded by a His bundle potential, with an H-Q interval of 60 msec. A pacemaker impulse (P_i) is shown without ventricular capture. Paper speed is 200 mm/sec. (Bottom) Recording similar to that in the top panel at a paper speed of 50 mm/sec. His bundle spikes (labeled with arrows in the bottom tracing) precede every QRS. Also note the complete absence of atrial activity.

degenerative changes with slight to moderate fibrosis.

Acute and chronic pericarditis was present throughout the ventricular myocardium but it was not as severe as in the atria. Only slight perivascular fibrosis with occasional small scars was seen in the myocardium of the right and left ventricles. Only slight arteriolosclerosis was seen. The summit of the ventricular septum presented marked fibrosis with hyalinization with thick prongs of connective tissue extending down to the annulus of the tricuspid valve. No amyloid was found in the ventricular myocardium.

Discussion

Atrial Standstill

Atrial standstill is characterized by the absence of atrial activity on the electrocardiogram with a slow escape rhythm of supraventricular origin.\textsuperscript{1-4} Corroborative evidence for the diagnosis may include absence of electrical atrial activity with esophageal and intraatrial recordings and absent mechanical atrial activity with fluoroscopy and pressure tracings.\textsuperscript{5-12} Electrophysiologic mechanisms explaining the occurrence of this arrhythmia would include sinus arrest with loss of pacemaker function, complete exit block of the SA node, and loss of...
atrial excitability. Presumably, atrial standstill may reflect both functional disturbance and morphologic abnormality of the conduction system.

Atrial standstill is usually transient, most commonly resulting from digitalis or quinidine intoxication. Other causes have included acute myocardial infarction, hypoxia, potassium overdosage, systemic lupus erythematosus, and diphtheria. Pathologic observations in patients with acute atrial standstill have been limited in number and biased strongly toward the more serious causes of this arrhythmia. In the autopsied cases, atrial standstill has frequently been a preterminal event. The most common pathologic entity associated with atrial standstill is that of arteriosclerotic heart disease. One patient reported by Magnusson had severe coronary sclerosis. James reported two cases of transient atrial standstill in an autopsy series of 11 patients with acute myocardial infarction and accompanying atrial arrhythmias. In these two cases there was proximal occlusion of the coronary artery that gave rise to the SA nodal artery with accompanying SA nodal infarction. Lippestad and Marton reported two similar cases.

In our first case, the diagnosis of atrial standstill was based on electrocardiographic findings. Intraatrial electrograms were not recorded during the episode of bradycardia so that retrograde P-wave activity in the QRS and T wave could not be ruled out. Atrial standstill was not preterminal, but preceded death by 10 days. Autopsy revealed coronary arteriosclerosis involving the three major coronary vessels. The left circumflex artery was completely occluded proximal to the SA nodal artery. In addition, there was considerable arteriosclerosis of the SA node and its approaches. We would postulate that atrial standstill represented functional depression of SA nodal function due to both ischemia and vagotonia. The latter is supported by the return of sinus rhythm after administration of atropine. This case supports previous observations that coronary occlusion proximal to the SA nodal artery may produce transient atrial standstill. It is of interest that this occurred without SA nodal infarction.

Persistent atrial standstill is a rare arrhythmia. Two cases have been associated with muscular dystrophy. Allensworth and co-workers reported three siblings with this arrhythmia, in two of which the atria were not excitable with either mechanical or electrical stimulation. A biopsy of the atrial appendage in one of the patients showed amyloid deposition. Complete autopsy examination has been reported in only one case of persistent standstill. Chavez et al. reported a case in which there was a perivascular and interstitial inflammation of the atria with marked degeneration and sclerosis of the SA node. Almost total destruction of the sinus node was felt to be the cause of persistent atrial standstill.

In case 2 we described a patient who first had fine atrial fibrillation and then developed persistent atrial standstill. The onset of the latter arrhythmia could not be precisely determined, but may have been present several years prior to the final admission. The diagnosis of atrial standstill was based upon the absence of atrial activity on both the surface electrocardiogram and intraatrial electrogram. One could question whether very fine atrial fibrillation might produce similar findings. However, in our experience, even fine fibrillation is associated with easily recorded intraatrial activity in the frequency ranges and sensitivities used in the present study. The right atrium was inexcitable and could not be paced from multiple sites. The atria were involved with degenerative disease of unknown etiology characterized by arteriosclerosis, fibrosis with hyalinization, fibrosis, fatty infiltration, and vascular degeneration of muscle cells. Atrial epicarditis was present, but appeared to be an inflammatory reaction to the degenerative atrial disease. The SA and A-V nodal approaches were markedly involved, while the SA and A-V nodes themselves were relatively spared. Sinus node dysfunction may have contributed to the arrhythmia, but the most prominent electrophysiologic and pathologic findings involved the whole atria. The slow ventricular response

Circulation, Volume XLIV, August 1971
to atrial fibrillation noted early in the patient's course would appear to have reflected involvement of the A-V nodal approaches.

A remarkable feature of this case was the selective atrial involvement. Ventricular hypertrophy and dilatation were present, but were probably related to previous hypertension, chronic loss of atrial transport function, and slow heart rate. Degenerative changes in the ventricular myocardium were minimal.

Thus, persistent atrial standstill in case 2 is due to a previously undescribed atrial disease. It is interesting to speculate that other cases of persistent atrial standstill, such as those occurring with muscular dystrophy or amyloidosis, may also represent total atrial involvement.

His Bundle Electrograms and Intraventricular Conduction

Catheter recording of the His bundle electrogram has added a useful dimension to the evaluation of conduction disease, allowing subdivision of the P-R interval into P-H and H-Q subintervals. It is not known from what segment of the His bundle the His bundle potential is recorded. The His bundle electrogram could represent depolarization of the penetrating portion, the branching portion, an intermediate area between these portions, or a composite of depolarizations from several segments of the His bundle. Despite this lack of knowledge, speculations have been made concerning the significance of P-H and H-Q prolongation. P-H prolongation is said to

Figure 15

Case 2. (Top) Atrial musculature showing marked vacuolization and irregular staining of cells, with zones of hyalinization. (Hematoxylin-eosin stain, × 130.) (Bottom) Atrial musculature showing fibrosis. (Hematoxylin-eosin stain, × 54.)
generally represent A-V nodal delay, while H-Q prolongation is said to represent delays in either the His bundle or in both bundle branches. There has been no pathologic confirmation of these speculations.

In the present report, His bundle electrograms were recorded in both patients. Each had sclerosis of the left side of the cardiac skeleton involving the intraventricular subjunctional conduction system as well as the penetrating portion of the A-V bundle in case 1. These cases have allowed us to make observations concerning the pathologic significance and sensitivity of the His bundle recording technique in the evaluation of intraventricular conduction.

In patient 1, there was marked calcification involving the mitral annulus and the summit of the ventricular septum which produced major disruption of the penetrating portion of the His bundle. In addition, bilateral bundle-branch disease was present, manifested by loss of connection of the left bundle branch with the common bundle and by moderate fibroelastosis of the first portion of the right bundle branch. The initial electrocardiograms revealed left bundle-branch block with a normal P-R interval. The P-H interval was at the upper limits of normal, and the H-Q interval was well within normal limits. The normal H-Q interval occurring in the presence of a major lesion involving the penetrating portion of the His bundle may be explained by postulating that the recording site of the His bundle was distal to the lesion. Thus, if the lesion produced slowing of conduction, this would be reflected in the P-H interval. Since the normal limits of P-H are rather wide, a small prolongation due to slowing of conduction in the penetrating portion of the His bundle might not be detected. An alternate explanation could be that conduction velocity remained normal in the His bundle tissue still unaffected by the calcific lesion. If this latter
pathologic findings, however, also incriminate the penetrating portion of the His bundle as a possible site for complete block.

In patient 2, fibrosis of the summit of the ventricular septum produced major involvement of both the branching portion of the His bundle and the proximal posterior portion of the left bundle branch. Although the typical pattern of left posterior hemiblock with rS in I and qR in III was not present, the transient right axis deviation noted during the second admission may have represented slowing or failure of conduction in the posterior division. The absent R wave in V1 and absent Q in V6 noted in the earlier admissions may also

possibility was correct, then the inference regarding the recording site of the His bundle would not be justified. In this patient with left bundle-branch block, moderate disease of the right bundle branch did not prolong H-Q. Thus, bilateral bundle-branch disease can be present without H-Q prolongation. At the time of study, A-V conduction was noted to be intact up to a paced rate of 150 beats/min. However, several days later, sudden complete A-V block developed. The total failure of conduction may have occurred in the A-V node, the penetrating or branching portion of His bundle, or the right bundle branch. The suddenness of onset without preceding Wenckebach periods suggested that block occurred in the right bundle branch, which was found to be moderately involved patho-

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have reflected delay of left septal depolarization, secondary to left posterior hemiblock.26

Slight H-Q prolongation was present and probably reflected delay of conduction in the branching portion of the His bundle. This electrophysiologic and pathologic correlation supports previous speculations that H-Q prolongation in patients with narrow QRS represents slowing of conduction in the distal portion of the His bundle.24

The present study suggests the following conclusions regarding the His bundle recording technique in the diagnosis of disease in the His-Purkinje system: (1) a major lesion may occur in the penetrating portion of the His bundle without H-Q prolongation, while a similar lesion occurring in the branching portion of the His bundle may prolong H-Q; (2) bilateral bundle-branch disease may be present in a patient without H-Q prolongation. These conclusions reflect results in two patients. It is certainly conceivable that similar lesions in other patients might produce different results. Further studies correlating conduction system pathology with the intracardiac recording of His bundle electrograms are necessary for further evaluation of this technique.

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Addendum


References

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