CASE REPORT

Return of Normal Conduction after Paroxysmal Heart Block

Report of a Case with Major Discordance of Electrophysiological and Pathological Findings

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SUMMARY
This report describes a 52-year-old male with paroxysmal heart block as well as left and right bundle branch block, resulting in Stokes-Adams attacks. The patient experienced a return to 1:1 atrioventricular (A-V) conduction with narrow QRS within 48 hours of the attacks and heart block never recurred. Electrophysiological studies three weeks later revealed a narrow QRS, a normal H-V interval (36 msec), 1:1 A-V conduction up to an atrial paced rate of 210 beats/min, and normal refractory periods with atrial extra-stimulus techniques (His-Purkinje system refractory periods less than 370 msec). The patient died from a cerebral embolus incurred during diagnostic left heart catheterization two days after electrophysiological studies. Postmortem examination revealed calcific aortic stenosis with calcific impingement upon the pars membranacea resulting in compression of the distal His bundle and marked disruption of the proximal portions of both bundle branches.

This report documents a major limitation of electrophysiological studies, this limitation being that these studies may be totally normal on one occasion in a patient with pathologically significant chronic conduction disease, which may become clinically manifest on another occasion.

Additional Indexing Words:
His bundle electrogram
Bilateral bundle branch block
Cardiac refractory periods
Stokes-Adams attacks

PREVIOUS STUDIES have demonstrated good correlation between the sites of conduction disease as predicted from electrophysiological studies, and pathological lesions as demonstrated by serial sections of the conduction system.1-3 In the present study, we describe a patient who had total recovery of conduction after symptomatic paroxysmal heart block. There was a marked discordance between electrophysiological findings which were totally normal (after recovery of conduction) and histological studies, which demonstrated major chronic destructive lesions involving the distal His bundle and proximal bundle branches.

The purpose of this report is not to discredit the value of electrophysiological studies in evaluating the conduction system, but to highlight a major limitation of these techniques. This case report raises more questions than it answers.

Report of Case

Clinical Summary
The patient was a 52-year-old male admitted to the Coronary Care Unit at the Highland Park Hospital on 1/29/74, following three syncopal episodes in the preceding 24 hours. The patient had been well up to the morning of

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admission, with no history of chest pain. Physical examination on admission was within normal limits.

On the day of admission, the patient developed recurrent syncopal episodes necessitating multiple cardiac resuscitations. The patient was treated with a temporary demand transvenous pacemaker implanted on the evening of 1/29. From 1/30 to 2/8 the patient remained asymptomatic, without further need for transvenous pacing.

The patient was transferred to the University of Illinois Hospital for further evaluation on 2/8/74. Physical examination on admission revealed a blood pressure of 125/80 mm Hg, a pulse of 80 beats/min, and normal carotid pulses. Cardiovascular examination revealed a grade 1/VI ejection murmur heard best at the left sternal border without radiation and a physiologically split second sound. Third and fourth heart sounds were not auscultated. Informed consent was obtained for electrophysiological studies and diagnostic catheterization. Electrophysiological studies were performed on 2/19 without difficulty. Diagnostic right and left heart catheterization was performed on 2/21. Previously undiagnosed aortic stenosis was encountered. While attempting to cross the aortic valve with a Gensini catheter, the patient had a stroke suggestive of cerebral embolization. He never regained consciousness and subsequently died with progressive hypotension on 2/27.

Analysis of Electrocardiograms

An electrocardiogram taken the morning of admission to Highland Park Hospital (1/29/74) revealed sinus rhythm at a rate of 94/min, a P-R interval of 0.18 sec, a QRS duration of 0.08 sec, a mean frontal axis of −20°, and minor T wave abnormalities (fig. 1). During the afternoon, rhythm strips recorded in the Coronary Care Unit demonstrated QRS widening, although a specific bundle branch block pattern could not be diagnosed (fig. 2A). At 5:10 p.m. on 1/29, the patient had a cardiac arrest secondary to complete heart block with prolonged asystole (fig. 2B). The onset of this attack was not recorded. Within minutes, 1:1 atrioventricular (A-V) conduction returned and 12 lead electrocardiogram (5:30 p.m.) revealed right bundle branch block with normal axis (fig. 3A). Between 5:30 and 7:30 p.m. on 1/29, the patient experienced multiple episodes of paroxysmal A-V block, each associated with prolonged asystole (fig. 2C and D). No consistent relationship to either bradycardic or tachycardic induction could be determined. At approximately 8:00 a.m. on 1/29, temporary transvenous pacing was established. Episodes of paroxysmal A-V block (type II block) continued to recur until approximately 2:00 a.m. on 1/30; however, asystole was prevented with demand transvenous pacing (fig. 2E). From 2:00 a.m. on, no further episodes of A-V block were recorded. Electrocardiogram taken on the morning of 1/30 revealed complete left bundle branch block pattern with intact A-V conduction (fig. 3B). No further episodes of A-V block were recorded on that day.

On 1/31, electrocardiogram revealed total reversal of all conduction defects, with return to the electrocardiographic pattern seen on admission (fig. 1). Specifically, no Q waves were noted, nor were other signs of acute myocardial injury with the exception of minor T wave inversion in leads I and aVL. All electrocardiograms taken from 1/31 through the day of the patient’s death (2/7), revealed narrow QRS without abnormality. Despite prolonged inpatient monitoring in the Coronary Care Units of both Highland Park

![Figure 1](image1)

Admission electrocardiogram demonstrating absence of conduction defect. There is no evidence of myocardial infarction.

![Figure 2](image2)

Figure 2

Rhythms strips (monitor lead) on 1/29 and 1/30. Panel A: Sinus rhythm with intraventricular conduction defect (QRS of 0.12 sec). Panel B: First recorded episode of asystole secondary to complete heart block. Note P waves without QRS complexes. Panel C: Onset of paroxysmal heart block which occurs after the third QRS. Panel D: Rhythm strip recorded during episode of complete heart block with A-V dissociation. Panel E: Episode of paroxysmal heart block (type II) with prolonged asystole prevented with demand pacemaker. The second P is blocked and the demand pacemaker takes over.
Electrophysiological Studies

His bundle electrograms were recorded on 2/19 using previously described catheter techniques. A quadripolar catheter was passed to the high right atrium for atrial stimulation and recording of high right atrial electrograms. Refractory periods were measured during sinus rhythm (cycle length of 777 msec) and with atrial driving (cycle length 667/min), utilizing atrial extra-stimulus technique (figs. 5 and 6). A-H interval was 88 msec (normal 92 ± 38 msec) and H-V was 36 msec (normal 43 ± 12 msec) (fig. 4A). Split H potentials were carefully searched for, but not found. Appropriate increase in A-H was noted with atrial pacing with 1:1 A-V conduction maintained to a paced rate of 210 beats/min, the maximum rate tested (figs. 4B & C). A-H at the latter rate was 275 msec. There were no blocked beats, either proximal or distal to the His bundle recording site with rapid atrial pacing. A1-A2, H1-H2 curves generated by extra-stimulus technique, were normal during both sinus rhythm and atrial driving (fig. 5). Conduction at both cycle lengths was limited by atrial refractoriness. All refractory periods were within normal limits (table 1 and fig. 6). Although bundle branch and/or His bundle refractory periods could not be measured, these had to be less than the A-V nodal functional refractory period which was 370 msec during sinus rhythm and 365 msec with atrial driving.

Pathological Findings

Postmortem examination was limited to the heart. Gross examination revealed slight right atrial, right ventricular and left atrial enlargement. The left ventricle was moderately hypertrophied and enlarged. All three coronary vessels revealed considerable atherosclerosis with moderate narrowing. There was no evidence of myocardial infarction.

The aortic valve was narrowed and abnormally formed. The posterior cusp was normally formed, while the right and left anterior cusps were combined into one cusp with a low raphe. The valve was thickened with marked calcium deposition in the aortic valve and in the pars membranacea.

The conduction system and the entire heart were examined with previously described serial section techniques. The sino-atrial (S-A) and A-V nodes and their approaches were serially sectioned with every 10th section being retained. The A-V bundle and the proximal portions of the bundle branches were serially sectioned with every 5th section being retained. The remainder of the bundle branches were serially sectioned and every 10th section was retained. The remainder of the heart was cut into blocks and two sections were taken from each block. Slides of the S-A and A-V node, A-V bundle and bundle branches were con-

Figure 3

Electrocardiograms showing complete right bundle branch block (panel A) and complete left bundle branch block (panel B). Both panels show sinus rhythm.
secutively stained with hematoxylin-eosin, Weigert-van Gieson and Gomori trichrome stains. In the remaining tissue, alternate sections were stained with hematoxylin-eosin and Weigert-van Gieson stains; 1,028 sections were thus examined. Conclusions as to pathologic changes were drawn in light of previous studies of the effect of aging on the conduction system.11

S-A node: There was moderate arteriolosclerosis, and a minimal infiltration with mononuclear cells. The nodal artery showed no changes.

Approaches to the S-A node: Occasional small scars were present, with fibrosis adjacent to the S-A node. There was occasional arteriolosclerosis.

Approaches to the A-V node: Slight fibrosis was present.

A-V node: There was marked arteriolosclerosis, with slight proliferation of sheath cells and fibrosis.

A-V bundle, penetrating portion: Arteriolosclerosis was moderate to marked. Fibroelastosis was moderate.

A-V bundle, branching portion: This was short and showed moderate fibrosis. It was cut off from the posterior portion of the main left bundle branch by fibrous tissue.

A-V bundle, bifurcation: Calcified material in the pars membranacea pressed on this region (fig. 7). There was marked fibrosis on both sides (on the left side 50-75% replacement, and on the right side 25-50% replacement).

Left bundle branch, main bundle: The posterior part was partially replaced (about 50%) by fibroelastic linear formations and was separated from the main bundle (fig. 8). The anterior part was almost completely replaced (about 90%) by fibrous tissue at the beginning (fig. 9).

Left bundle branch, distal portion: Both posteriorly and anteriorly the Purkinje cells were smaller than normal and showed acute degeneration.

Right bundle branch: There was marked fibrosis (50-75% replacement) of the beginning of the first part (fig. 10), and the middle of the second part.

Summit of ventricular septum: The marked calcification of the aortic valve also involved the pars membranacea where masses of calcium compressed the bifurcation of the A-V bundle. The summit of the ventricular septum showed marked arteriolosclerosis and sclerosis of the small coronary arteries. The mitral annulus was also considerably thickened.

Right atrium and ventricle: Moderate arteriolosclerosis was present.

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**Table 1**

Refractory Periods (in msec)

<table>
<thead>
<tr>
<th></th>
<th>CL of 777 (min)</th>
<th>CL of 667 (AP)</th>
</tr>
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<tbody>
<tr>
<td>Atrial ERP</td>
<td>160</td>
<td>180</td>
</tr>
<tr>
<td>Atrial FRP</td>
<td>275</td>
<td>190</td>
</tr>
<tr>
<td>A-V nodal ERP</td>
<td>275</td>
<td>190</td>
</tr>
<tr>
<td>A-V nodal FRP</td>
<td>370</td>
<td>365</td>
</tr>
<tr>
<td>HPS ERP</td>
<td>&lt;370</td>
<td>&lt;365</td>
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</tbody>
</table>

Abbreviations: CL = cycle length; AP = atrial pacing; HPS = His-Purkinje system; ERP = effective refractory period; FRP = functional refractory period.

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**Figure 4**

Conduction intervals during sinus rhythm and atrial pacing. Shown are leads 1, 2, 3, V., high right atrium (HRA), and His bundle (HBE). Atrial electrogram is labeled A, His bundle electrogram H, and ventricular electrogram V. Time lines are at one second and paper speed is 100 mm/sec on this and all subsequent illustrations. A-H and H-V intervals are listed. Panel A: Sinus rhythm (NSR). Note normal conduction intervals. Panel B: Atrial pacing at a heart rate (HR) of 200 beats/min. Stimulus artifacts are labeled S. 1:1 A-V conduction is present with an A-H interval of 250 msec. The QRS occurring almost simultaneously with the stimulus artifact is conducted from the previous stimulus artifact. H marked by S. Panel C: Pacing at 210 beats/min. Again, note 1:1 A-V conduction. Pacing is discontinued after the sixth stimulus artifact. The seventh QRS is conducted from the sixth pacing artifact with an A-H of 275 msec.

**Figure 5**

A1-A2, H1-H2 curves during sinus rhythm (panel A) and with atrial pacing (panel B). A1-A2 intervals are listed on the horizontal axis and H1-H2 responses on the vertical axis. Both conduction curves are normal. The shortest H1-H2 on each curve (the functional refractory period of the A-V node) was conducted to the ventricles.
PAROXYSMAL HEART BLOCK

Measurement of refractory periods with atrial extra-stimulus technique. The first two beats in each panel are conducted sinus beats and are followed by an atrial extra-stimulus (S_I). A_I and H_I are atrial and His bundle electrograms of sinus beats, and A_2 and H_2 electrograms in response to the extra-stimulus. A_I-A_2 and H_I-H_2 intervals are listed for each panel. Panel A: Responses at an A_I-A_2 interval of 570 msec. Panel B: Responses at an A_I-A_2 interval of 290 msec. The H_I-H_2 of 370 msec was the A-V nodal functional refractory period. Note that H_2 is conducted to the ventricle without either H-V prolongation or aberrant conduction. The refractory period of the His-Purkinje system is less than 370 msec (H_I-H_2). Panel C: Responses at an A_I-A_2 interval of 275 msec (the atrial functional refractory period).

**Left atrium and ventricle:** Slight arteriolosclerosis and fibrosis were present.

**Discussion**

Our patient had an initial clinical diagnosis of idiopathic paroxysmal heart block with Stokes-Adams attacks. Electrocardiograms taken close in time to paroxysmal block also revealed intact A-V conduction with patterns of both left and right bundle branch block, suggesting that the site of A-V block was in the bundle branches (bilateral bundle branch block). Subsequent pathological examination revealed calcific aortic stenosis with impingement of calcium upon the pars membranacea, resulting in compression of the His bundle at the bifurcation, with marked disruption of the proximal portions of both the right and the left bundle branches. This was associated with moderate arteriolosclerosis and marked arteriosclerosis of the summit of the ventricular septum. Similar findings have been described in previous reports of patients with calcific aortic stenosis and heart block.

The major interest of the present report resides in the series of events occurring between the episodes of heart block and the patient’s subsequent demise three weeks later. The bundle branch blocks resolved within 48 hours resulting in a narrow QRS, and A-V block never recurred. Electrophysiological studies performed approximately three weeks after the episode of A-V block were entirely normal. Specifically, A-H and H-V intervals were within normal limits; intact A-V
Conduction was noted up to an atrial paced rate of 210 beats/min, and refractory periods measured with atrial extra-stimulus technique were normal. The demonstration of normal conduction intervals in a patient with previous A-V block, or subsequent development of A-V block are not totally new. Previous studies have demonstrated that patients with bundle branch block and normal H-V intervals may develop intermittent heart block distal to the His bundle within days of the initial electrophysiological study. Narula and Samet reported that intact A-V conduction at rapid paced rates could also be present within hours of development of spontaneous A-V block. These findings were explained by postulating an appropriate decrease in refractory periods of the diseased His-Purkinje system with shortening of cycle length. However, the demonstration of normal refractory periods at several cycle lengths within three weeks of an episode of heart block presumably due to chronic conduction disease as demonstrated in our case has not been previously reported.

The present case thus highlights a major limitation of current electrophysiological techniques. Our case demonstrates that major destructive lesions may be present in the His bundle and both bundle branches without any abnormality detectable during His bundle recording with atrial stimulation. This does not imply that electrophysiological studies are without value in other cases. Previous studies have demonstrated that when electrophysiological abnormalities are demonstrated, appropriate pathological lesions are usually found. This is true for lesions at the A-V node, His bundle, and bundle branches. Electrophysiological studies may also delineate conduction disease not apparent on the surface electrocardiogram.

The present case also highlights limitation of conclusions drawn from pathological techniques for examination of the conduction system. The conduction findings were such that the pathologist might have predicted at least the presence of one chronic bundle branch block. The findings of totally normal conduc-
PAROXYSMAL HEART BLOCK

Figure 10
Fibroelastosis of first part of right bundle branch. Weigert-van Gieson stain × 31. V = ventricular myocardium; arrows point to right bundle branch.

Pathological prior to death can be explained by noting that acquired conduction disease rarely results in total destruction of either the His bundle or bundle branches. There is usually correlation between the presence of a bundle branch block and/or A-V block with appropriate pathological lesions. Our case points out that it is possible to have severe disease with normal conduction. In the pathological sections of a seriously diseased conduction structure, there are usually some apparently viable conducting cells. The pathologist cannot predict from histological findings whether the damaged structure is able to function normally, sub-optimally (incomplete block), or not at all (complete block). These electrophysiological and pathological limitations are further compounded by the tendency for all or none conduction in the His-Purkinje system. Specifically, the determinants of why the chronically diseased His-Purkinje tissue may conduct some of the time have yet to be delineated.

One of the determinants for the presence of the transient block in our case might be that some acute event may have occurred at the time the patient presented with Stokes-Adams attacks. One could postulate mechanical movement of the calcium, producing sudden compression with failure of conduction in either the distal His bundle or proximal bundle branches. The patient also had coronary athero- and arteriosclerosis. It is possible that acute reversible ischemia precipitated A-V block. However, in the absence of chest pain and absence of both electrocardiographic and pathological evidence of myocardial infarction, these hypotheses cannot be validated. The possibility of changing autonomic influences can be considered; however, the reasons for such sudden autonomic imbalance would not be ascertainable.

Clinical Implications
The clinical implications of this report are as follows: 1) Electrophysiological studies may be totally normal on one occasion in patients with clinically and pathologically significant chronic conduction disease, which may become manifest on another occasion. Thus, in patients with suspected Stokes-Adams attacks, the demonstration of normal conduction intervals, pacing responses and refractory periods, does not guarantee a normal conduction system, or eliminate intermittent A-V block as a cause of symptoms. 2) Paroxysmal A-V block may be a cause of syncope. One potential cause of sudden death in patients with calcific aortic stenosis. It is our opinion based upon the present case, other reported cases, and our unpublished experience, that conduction disease has been underestimated as a cause of syncope and sudden death in patients with aortic stenosis. It is our impression that conduction disease in aortic stenosis, as illustrated by the present case, is unpredictable in its clinical behavior. 3) Aortic stenosis should be considered in patients with idiopathic conduction disease, despite absence of suggestive physical findings. We believe that the diagnostic work-up of patients with idiopathic A-V block (and perhaps idiopathic bundle branch block) should include measurement of systolic time intervals, careful cine-fluoroscopy of the aortic valve for calcium, and echocardiography of the aortic valve. We believe that if the above examinations had been performed on our patient prior to cardiac catheterization, the existence of aortic stenosis probably would have been recognized.

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References

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