Electrophysiologic and Pathologic Correlations in Two Cases of Chronic Second Degree Atrioventricular Block with Left Bundle Branch Block

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SUMMARY
This study concerns two cases of chronic 2° atrioventricular (A-V) block with left bundle branch block (LBBB). Pathological studies included serial section of the conduction systems. Case 1 had type I 2° block with LBBB. Electrophysiological studies revealed type I 2° block proximal to the His bundle recording site and a prolonged H-V interval (60 msec). Pathologically there was a moderate to marked fibrosis of the approaches to the A-V node and of the A-V node, marked fibrosis of the left bundle branch, and moderate involvement of the right bundle branch. The changes proximal to the His bundle were more marked than the changes distal to this bundle. Case 2 had type II and 2:1 2° A-V block with LBBB. Electrophysiologically the site of block was distal to the His bundle recording site, and there was a prolonged A-H interval (145 msec). Pathologically there was marked fibrosis of the A-V node and severe involvement of both bundle branches. The changes distal to the His bundle were more severe than the changes proximal to the His bundle.

This study reveals that the electrophysiologic data more closely approximated the pathologic findings than did surface electrocardiographic data alone. It also emphasizes that there may be multiple sites of disease in chronic 2° block with bundle branch block.

RECENT STUDIES from our laboratories have demonstrated excellent correlation between sites of conduction disease predicted with His bundle recording, and sites subsequently demonstrated with serial sectioning of the conduction system. These previous studies were concerned with correlations in patients with complete heart block1 and in patients with bundle branch block and intact atrioventricular (A-V) conduction.2 In the present study, results of serial section of the conduction system are reported in two patients with chronic 2° A-V block and left bundle branch block (LBBB), who had electrophysiological studies prior to death.

Case Reports
Clinical and electrophysiological data from these two cases have been previously reported (Cases 2 and 7).3 This section will briefly recapitulate previous data and summarize the subsequent clinical course.

Case 1 (Case 2)3

Clinical Review
The patient was a 52-year-old male with known congestive heart failure, diabetes, hypertension, arteriosclerotic heart disease, and peripheral vascular disease. The earliest available electrocardiogram for analysis was taken in June 1972 and revealed first degree A-V block with a P-R interval of 0.36 sec and atypical LBBB (fig. 1).

The patient was admitted on 9/14/72 with type I 2° A-V block (fig. 2). Prior to his admission, the patient had been on 0.25 mg of digoxin per day. Electrophysiological studies at this time revealed 2° block
proximal to the His bundle recording site with a prolonged H-V interval of 60 msec (fig. 3). Despite discontinuance of digoxin, 2° block persisted. A permanent pacemaker was recommended but refused by the patient.

The patient was then followed in the clinic from October 1972 to July 1973. Electrocardiograms taken in the clinic revealed LBBB, with varying degrees of A-V conduction disturbances, e.g., first, second, and third degree A-V block.

The patient was admitted to the hospital on July 18, 1973, with jaundice, and died three weeks later with progressive hepatic insufficiency. Electrocardiogram on the terminal admission revealed first degree A-V block (0.32 sec) and LBBB.

Postmortem Examination

Aside from the findings in the heart, the pathologic diagnoses were: (1) pulmonary edema, (2) carcinoma of the pancreas, (3) chronic pancreatitis, (4) chronic cholecystitis, (5) arterionephrosclerosis.

The Heart. The heart was enlarged weighing 480 g. There was hypertrophy and enlargement of all the chambers. A depression was present at the base of the
ventricular septum posteriorly close to the posterior commissure of the mitral valve. The endocardium was whitened in this area. The right coronary artery was occluded about 3 to 4 cm from its origin. The left circumflex coronary artery was almost occluded and the anterior descending coronary artery markedly narrowed.

Microscopic Examination

These studies were carried on without a knowledge of the electrocardiographic and electrophysiologic findings.

Methods

The sinoatrial (SA) and A-V nodes and their approaches were serially sectioned and every tenth section was retained. The A-V bundle and beginning of the bundle branches were serially sectioned and every fifth section was retained. The remainder of the bundle branches up to the region of the moderator band were serially sectioned and every tenth section was retained. The atrial tracts were serially sectioned and every fortieth section was retained. 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 this way 1044 sections were examined. This method of examination has previously been reported. The findings were controlled by studies of aging of the conduction system.

This included 14 hearts from patients aged 51-60 and 11 hearts from patients 61-90 for the study of the SA node, and 12 hearts from patients 51-60 and eight from patients 61-81 years of age for the study of the A-V node, bundle, and bundle branches. These patients had no clinical heart disease nor evidence of diastolic hypertension, and pathologically there was no evidence of heart disease.

Findings

SA Node. The medial part of the SA node showed a marked loss of cells. The lateral part was well preserved.

Approaches to the SA Node. Numerous small microscopic scars and large areas of fibrosis were present. Several large arterioles showed intimal proliferation and narrowing.

Approaches to the A-V Node. There was moderate to marked fibrosis (fig. 4, left panel) and moderate fatty infiltration. The ramus septi fibrosi was normal.

A-V Node. Moderate fibrosis (fig. 4, right panel) and slight arteriolosclerosis were present.

A-V bundle, penetrating portion. There was moderate fibrosis. This portion was very short.

A-V bundle, branching portion. Fibrosis was moderate to marked, and moderate arteriolosclerosis was present. There was partial disruption in continuity at its junction with the posterior part of the left bundle branch (LBB) (fig. 5, left panel).

LBB: Main bundle. The posterior part was partially disrupted (fig. 5, left), while the anterior part was more disrupted at its junction with the bundle of His and this part showed moderate to marked fibrosis (fig. 5, right panel).

Periphery. The posterior and anterior portions showed moderate fibrosis.

Right bundle branch (RBB). All its parts showed moderate fibrosis (fig. 6). There was a slight infiltration of mononuclear cells in the third part.

Atrial tracts. Moderate to marked fibrosis with scar formation was present.

Ventricles. The anterior and posterior walls of the left ventricle and the ventricular septum including the summit were the seat of an old infarct, in spots subendocardial and in spots intramyocardial. A moderate number of small scars were present in the anterior wall of the right ventricle. The summit of the ventricular septum showed marked arteriolosclerosis.

Case 2 (Case 7)

Clinical Review

The patient was a 78-year-old male, first seen in August 1970 with a history of angina pectoris, hypertension, and congestive heart failure. Electrocardiogram at that time revealed sinus rhythm, first degree A-V block with a P-R interval of 0.22 sec, and complete LBBB (fig. 7). He was managed medically and did well until 10/15/71 when he was admitted with syncope. Admission electrocardiograms revealed Mobitz type II and 2:1 2° A-V block (fig. 8). Electrophysiologic studies on admission showed type II and 2:1 A-V block distal to the His bundle recording site with a prolonged A-H interval of 145 msec and a prolonged H-V interval (conducted beats) of 75 msec (fig. 9). The patient initially refused a pacemaker. However, because of recurrent syncope, a permanent demand pacemaker was implanted in December 1971.

Electrocardiograms between December 1971 and February 1974 revealed pacemaker rhythm with A-V dissociation with occasional periods of sinus capture. In March 1973, the patient developed atrial fibrillation. All subsequent electrocardiograms revealed atrial fibrillation and pacemaker rhythm with A-V dissociation and occasional supraventricular captures.

The patient underwent a battery replacement in August 1973. In March 1974, he was admitted with progressive congestive heart failure and died.

Postmortem Examination

Aside from the findings in the heart, the pathologic diagnoses were: 1) pulmonary infarct, infected, right
Figure 4

Case 1. The approaches to the A-V node and the A-V node. Left panel) Approaches. Weigert-van Gieson stain X 15. Right panel) A-V node. Weigert-van Gieson stain X 22.5. Note the marked increase in fibrous tissue in the approaches (left panel) and the moderate increase in the node (right panel). Arrows point to the node.

Figure 5

Case 1. Bundle of His, posterior part of main left bundle and bifurcation. Left panel) Branching bundle of His and posterior part of left bundle branch (LBB). Hematoxylin-eosin stain X 31.5. Right panel) Bifurcation. Weigert-van Gieson stain X 31.5. Note the partial disruption in continuity of the branching portion of the A-V bundle with the posterior part of the LBB on the left, and the disruption in continuity of the anterior part of the LBB at the bifurcation on the right. B = branching portion of bundle; V = ventricular septum. Arrows point to disruption of LBB.
upper and right lower lobes; 2) pulmonary edema; 3) arterio- and arteriolonephrosclerosis; 4) marked chronic passive hyperemia of visceral organs.

The Heart. The heart was enlarged, weighing 645 g. There was hypertrophy of all chambers of the heart especially of the left atrium and left ventricle with enlargement of the left atrium and left ventricle. The aortic valve showed calcification of the left aortic cusp at its base and generalized thickening and atherosclerosis of the sinuses of Valsalva of the other cusps. The anterior descending coronary artery was the seat of moderate atherosclerosis and calcification with slight narrowing. The right coronary artery showed patchy calcification with no narrowing.

Microscopic Examination

Methods. The SA and A-V nodes and their approaches were serially sectioned and every tenth
His bundle electrograms from case 2 during Mobitz type II A-V block. Shown are leads I, II, III, V₁, and three His bundle electrograms. Note that the third P wave is blocked distal to the His bundle without any preceding change in conduction intervals. Both A-H and H-V are prolonged. The QRS following the blocked H potential is an escape (arising from the right ventricle). This illustration is a modification of figure 4 from Dhingra et al. with permission of American Heart Association.

- **Figure 9**

section was retained. The A-V bundle and the main LBB and the first part of the RBB were serially sectioned and every fifth section was retained. The remainder of the bundle branches up to the region of the moderator band were serially sectioned and every tenth section was retained. The roof of the right atrium and the superior and middle preferential pathways were serially sectioned and every fortieth section was retained. (The inferior pathway was examined with the approaches to the A-V node.) The remainder of the heart was cut into blocks and two sections were taken of each block. The slides from blocks containing the SA and A-V nodes, the A-V bundle, the main LBB and the first part of the RBB were consecutively stained with hematoxylin eosin, Weigert-van Gieson, and Gomori trichrome stains. The remainder of the slides were alternately stained with hematoxylin-eosin and Weigert-van Gieson stains. In this manner 1758 sections were examined. These findings were controlled as in the previous case by the studies of aging of the conduction system.⁵, ⁶

**Findings**

- **SA Node.** There was marked fatty infiltration, hemorrhage, and arteriolosclerosis mostly in the peripheral portions. A moderate loss of cells was present and there was a slight infiltration with mononuclear cells.

- **Approaches to SA Node.** Marked fibroelastosis with arteriolosclerosis was present. In the region where the catheter had been, there was thickening of the endocardium with an organized thrombus. There was also hypertrophy of some cells with acute degeneration of cells.

- **Approaches to A-V Node.** Marked fibroelastosis with arteriolosclerosis was present. The ramus septi fibrosi was moderately thickened. Calcification was noted at the base of the mitral valve. There was a moderate infiltration of mononuclear cells. The aortic portion of the central fibrous body was thickened.

- **A-V Node.** There was a marked increase in fibroelastic tissue (fig. 10) with an infiltration of mononuclear cells. Arteriolosclerosis was marked. The cells showed moderate difference in stainability with increased eosinophilia of some cells.

- **A-V Bundle, Penetrating Portion.** The beginning showed marked fibrosis. There was moderate irregular staining of cells and an increase in spaces.

- **A-V Bundle, Branching Portion.** There was slight fibrosis and arteriolosclerosis. A scar was present on the right side. There was slight hemorrhage.

- **LBB.** This was almost completely replaced at its origin by fibroelastic tissue, more posteriorly (fig. 11, left) than anteriorly (fig. 11, right). In addition, there was marked irregularity of staining of cells in the periphery with moderate fibrosis.

- **RBB.** There was a scar at the beginning of the RBB. The latter divided into two parts. A small twig was given off early which showed fibroelastosis and vanished. The main RBB showed marked fibrosis in the first part, severe fibrosis in the second part (fig. 12), and moderate fibrosis in the third part.

- **Preferential Atrial Pathways.** Marked fibroelastosis was present, with arteriolosclerosis and focal degeneration of cells.

- **Myocardium.** The summit and the remainder of the ventricular septum showed marked fibroelastosis and arteriolosclerosis. The left ventricle showed marked fibroelastosis with arteriolosclerosis, with marked irregular stainability of cells with increased eosinophilia of some cells. These findings were more
marked in the anterior wall than the posterior. The right ventricle showed only slight fibroelastosis with small scars. Both atria were the seat of marked fibrosis with arteriolosclerosis.

Pericardium. Chronic pericarditis with fibrosis was present.

The extent of the pathologic findings in each part of the conduction systems in case 1 and case 2 are given in the accompanying table 1. These findings were not found in the control normal hearts.

Discussion

Previous studies from our laboratories demonstrated good correlations between electrophysiological studies and pathological findings regarding sites of conduction disease. These studies have been concerned with atrial standstill, complete heart block, bundle branch block with intact A-V conduction, and split-His potentials. In the present study, we describe the pathologic substrate of two patients with chronic 2° A-V block and bundle branch block, in whom electrophysiological studies were performed. In case 1, electrocardiograms revealed type I A-V block with long P-R intervals and LBBB suggesting that the major conduction disturbance was in the A-V node or its approaches. This was confirmed with His bundle recordings which demonstrated a site of block proximal to the His bundle. However, electrophysiological studies also demonstrated a prolonged H-V interval. This latter finding, in combination with the electrocardiographic diagnosis of LBBB, also suggested the presence of bilateral bundle branch disease.
In case 2, electrocardiograms revealed Mobitz type II block with LBBB suggesting that the major conduction disturbance was in the His-Purkinje system. This was also confirmed with His bundle recordings, which revealed type II and 2:1 block distal to the His bundle. However, electrophysiologic studies also revealed prolonged A-H intervals suggestive of A-V nodal conduction disease.

Pathologically we were dealing with hypertensive and arteriosclerotic heart disease in the first case and hypertensive heart disease in the second case. In case 1 there was concomitant arteriolosclerosis, limited to the conduction system and the summit of the ventricular septum. In case 2 the entire myocardium was the seat of arteriolosclerosis, including the conduction system. The changes in the conduction system in both cases were related to ischemia, due to narrowing of the large and small arteries in case 1 and of only of the small arteries in case 2. These ischemic changes were reinforced by the mechanical changes related to sclerosis of the left side of the cardiac skeleton in case 2, and the old infarct at the summit of the ventricular septum in case 1. Both the arteriolosclerotic effects and the mechanical effects are related to the hypertension present in both cases.

There was good but not perfect correlation between electrocardiographic, electrophysiologic and pathological findings. In both cases, the electrophysiologist would have predicted the presence of diffuse conduction disease involving both the A-V node and His-Purkinje system. In case 1, he would have suggested that proximal conduction disease was more extensive than that in the distal portion, while in case 2, the distal conduction disease would have predominated. The diffuse involvement of the conduction system in both cases was confirmed pathologically. Likewise in case 2 it was confirmed that the findings in the bundle branches were more severe than the findings in the approaches to the A-V node and the A-V node. In case 1, however, the findings were of about the same severity both proximal and distal to the His bundle.

The slight divergence in the electrophysiologic and pathologic findings in case 1 might be explained by the difficulty for the pathologist to evaluate exactly the extent of pathologic involvement of the approaches to the A-V node, because of the large area involved in the approaches, in contrast to the more discrete structures situated more distally. This might also explain the fact that even though there is considered to be more pathologic change in the approaches to the A-V node and the A-V node in case 2 than in case 1, yet there is second degree block proximal to the His bundle in case 1 and only delayed conduction proximal to the His bundle in case 2. Still another factor which cannot be evaluated pathologically is the nerve control affecting the A-V nodal region, which is probably not present in the branching bundle and bundle branches.

It is to be noted that the electrophysiologic studies gave a more precise account of the pathologic changes than did surface electrocardiographic data alone. However, we recognize that there are pathologic changes in the conduction system which have no clinical, electrocardiographic, and electrophysiologic representation.

The above findings in chronic 2° block are in contrast to previous correlations in acute 2° A-V block. In acute type I 2° A-V block related to ischemia, lesions may be especially localized in the approaches to the A-V node and A-V node, and in acute type II 2° block lesions may be especially prominent in the distal bundle and the bundle branches. Thus, pacing is universally recommended in type II acute A-V block, but not in type I acute A-V block.

In chronic 2° block with bundle branch block, a more diffuse involvement of the conduction system may be anticipated, as demonstrated in the present cases. This is in keeping with the recent work of Dhingra and co-workers who suggested that most patients with chronic 2° block and bundle branch block would need permanent pacing including some cases with type I. In this work, however, 2° block distal to the His bundle was associated with a more malignant prognosis than 2° block proximal to the His bundle.

The pathogenesis of the lesions in the left bundle in our cases corresponds to that reported in our previous work. We are dealing in both cases with ischemia related to vascular changes which are the concomitants of hypertension. This factor is enhanced by

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### Table 1

**Extent of Pathologic Findings**

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<tr>
<th>Case no.</th>
<th>SA node</th>
<th>SA approaches</th>
<th>AT</th>
<th>A-V approaches</th>
<th>A-V node</th>
<th>A-V bundle penetrating</th>
<th>A-V bundle, branching</th>
<th>LBB</th>
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**Symbols:** 1+ = minimal; 2+ = moderate; 3+ = marked; and 4+ = severe or total.

**Abbreviations:** SA = sinoatrial node; AT = atrial tracts; A-V = atrioventricular node; LBB = left bundle branch; RBB = right bundle branch.
the mechanical factor abetted by the hypertension and evident pathologically as fibrosis or scar formation at the summit of the ventricular septum. The lesions of the RBB however are considered to be only ischemic.

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