Pathophysiologic Correlations in Two Cases of Split His Bundle Potentials

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SUMMARY
This is a pathophysiological correlation in two cases showing split His bundle potentials. The first case had a history of previous complete heart block and the electrophysiological studies revealed split His potentials with intact A-V conduction. Case two had split His potentials with complete heart block. Serial sections of the conduction system in both cases revealed calcific impingement on, and degenerative changes within the bundle of His with healthy bundle of His proximal and distal to the lesion.

Additional Indexing Words:
Split His potentials  Bundle of His  Heart block  Conduction system

A TRIOVENTRICULAR (A-V) BLOCK may be located proximal, within, or distal to the His bundle, with the use of the His bundle recording technique.1-3 Pathological studies, utilizing serial sections of the conduction system, have been reported in a limited number of patients with conduction disease who had His bundle recordings.4-6 These studies suggested that block proximal to the His bundle is associated with lesions in the A-V node and A-V nodal approaches. In contrast, block distal to the His bundle was associated with significant sclerodegenerative bilateral bundle branch disease.

There are no reported cases where serial section of the conduction system has been performed in patients with suspected block within the His bundle with His bundle recordings. In this report, we detail the pathological findings in two patients with split H potentials, an electrophysiological finding suggesting His bundle block.1-3,7-12 These findings helped confirm the relationship of split H potentials and localized disease within the His bundle.

Materials and Methods
His bundle electrograms were recorded with a tripolar catheter passed percutaneously from a femoral vein.13 Recordings were obtained on a multichannel oscilloscopic photographic recorder at paper speed of 100 and 200 mm/sec. (Electronic for Medicine, DR 16; Minneapolis, Minn.). A-H interval was measured from the first rapid deflection of the atrial electrogram to the first high frequency component of the His bundle electrogram. H-V interval was measured from the first high frequency component of the His bundle electrogram to the earliest QRS deflection recorded on the surface electrocardiogram. The normal values for intervals from our laboratory are as follows (mean ± 2 SD): A-H 92 ± 38 msec and H-V 43 ±12 msec.14 When split His bundle potentials were recorded, the initial (proximal) potential was labeled H and the second potential (distal) labeled H'. When split potentials were recorded with intact A-V conduction, measurements were made of A-H, H-H' and H'-V. When complete block occurred between H and H', only A-H and H'-V intervals could be measured.

In the first patient, single and coupled atrial pacing were performed using a second electrode catheter placed against the lateral wall of the right atrium. Because of difficulty in accurately measuring the onset
of the atrial electrogram during atrial pacing, the interval from pacing spike to H (S-H) was also measured. With a constant stimulus-atrium latency, directional changes in S-H are identical to those of A-H.

The method of histologic study of the conduction system was that of Lev and McMillan. In case I the sinoatrial (SA) node and its approaches were serially sectioned and every tenth section was retained. The A-V node and its approaches, the bundle of His and the bundle branches up to the level of the muscle of Lancisi were serially sectioned and every fifth section was retained. The remainder of the bundle branches were serially sectioned and every tenth section was retained. The atrial preferential pathways and the roofs of both atria were serially sectioned and every forty-fifth section was retained. The remainder of the heart was cut into blocks and two sections were taken from each block. Sections were alternately stained with hematoxylin and eosin and Weigert-van Gieson stains. In addition, every third section from the bundle of His and the main left bundle and the first part of the right bundle branch were stained with Comori's trichrome. In this manner 2,433 sections were examined for case I. In case II, the SA, A-V nodes and their approaches, the bundle of His and the bundle branches up to the region of the moderator band were serially sectioned and every tenth 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 and eosin and Weigert-van Gieson stains. In this manner 1,008 sections were examined. Results of serial sections in both cases were evaluated in the light of previous studies in a group of patients of similar ages without apparent conduction disease.16,17

Case I

Clinical Summary

The patient was a 67-year-old male who was admitted to Evanston Hospital on 7/18/72 with a 24 hour history of dyspnea. Physical examination revealed bradycardia, basal rales, and an ejection murmur at the cardiac base. He was treated with digitalis, diuretics, and temporary transvenous pacing, and became asymptomatic within 72 hours of admission. On 7/30/72 he was transferred to the University of Illinois Hospital for further evaluation. Examination at that time revealed a blood pressure of 180/75 mm Hg and a heart rate of 60 beats/m. The previously described ejection murmur was still present at the cardiac base and was heard over the carotid arteries. The temporary pacemaker was removed following electrophysiological studies. The patient was transferred back to Evanston Hospital and was discharged on 8/10/72. Following discharge the patient remained asymptomatic and was followed closely by his family physician. On 11/12/72 he developed severe substernal chest pain, dyspnea, and collapsed. He was dead on arrival at Evanston Hospital.

Analysis of Electrocardiograms

Only one electrocardiogram taken prior to the current admission was available for review. This ECG taken in 1967 revealed a normal PR interval, QRS axis, and QRS duration. QRS morphology was within normal limits except for counterclockwise rotation. Admission electrocardiogram on 7/18/72 revealed complete heart block with an atrial rate of 104/min and a ventricular rate of 34/min (fig. 1). The escape rhythm was characterized by a wide QRS (0.12 sec) with a pattern of right bundle branch block and an axis of +60°. This QRS was almost identical to that of conducted beats when conduction subsequently returned (fig. 1). The escape rhythm could be either idioventricular arising in the left ventricle or A-V junctional with additional right bundle branch block. On 7/24/72 complete heart block spontaneously reversed. The ECG on 7/24 as well as subsequent cardiograms revealed sinus rhythm, first degree A-V block (PR 0.22 sec), right bundle branch block, and an axis of +80° to +90° (fig. 1).

Electrophysiological Studies

Electrophysiological studies were performed when the patient was in sinus rhythm (7/31/72) The atrial
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rates varied between 60 and 70/min. Spontaneous A-V block was not noted. With the tripolar catheter positioned at the tricuspid valve, two high frequency electrograms were recorded between the atrial and ventricular electrograms of conducted beats (fig. 2). These closely resembled previous descriptions of split H potentials and will be referred to as H and H'.

Control conduction intervals were as follows: A-H 75 msec, H-H' 48 msec, and H'-V 48 msec.

Conduction was stressed with atrial pacing(s). S-H and A-H increased as pacing rates were increased (figs. 3A-C). H-H' also increased when contrasted to control intervals during sinus rhythm (fig. 3A). At a paced rate of 120 beats/min, Wenckebach periods were noted between H and H'. These periods were characterized by progressive increases in H-H' over several beats terminating in block between H and H', then followed by resumption of conduction (fig. 3B). At a paced rate of 130 beats/min, 2:1 block was noted between H and H' (fig. 3C), the H-H' of conducted beats, being 100 msec H'-V remained constant during sinus rhythm and at paced rates.

Coupled atrial stimulation was performed with an atrial extra stimulus introduced after every tenth sinus impulse (figs. 4A-C). The extra stimulus was conducted with prolongation of S-H, A-H, and H-H'. These intervals prolonged further as coupling intervals were shortened (figs. 4A and B). At a coupling interval of 380 msec (measured from sinus P to extra stimulus) conduction failed between H and H' (fig. 4C). The shortest attainable interval between the H potential of the conducted beat and that of the extra stimulus was 480 msec. This could thus be considered the effective refractory period of splitting. Extra stimuli were not introduced at coupling intervals short enough to measure the A-V nodal and atrial effective refractory periods. His bundle pacing was not utilized for validation, for reasons previously discussed.

Autopsy Findings

Aside from the findings in the heart, the pathological diagnoses included: 1) Bilateral severe pulmonary edema and congestion; 2) marked passive hyperemia of liver; and 3) severe generalized atherosclerosis.

Heart

The heart was hypertrophied and enlarged, weighing 729 gm. The right atrium and both ventricles were hypertrophied. The left atrium was hypertrophied and enlarged. The mitral valve was calcific at the base and line of closure and the chordae were thickened. The aortic orifice was narrowed by changes in the valve. Marked calcification of the body, the line of closure, the edge, the base and the upper margins of the sinuses of Valsalva of the aortic cusps was seen. The pars membranaceae was somewhat thickened. All the coronary arteries were calcified and narrowed.

Histologic Examination: Conduction System

SA node. The tail was atrophied and surrounded by fat.

Approaches to SA node. There was considerable fibrosis and fatty infiltration.

Atrial preferential pathways. Fatty infiltration was marked.
Right bundle branch (RBB). The first part showed moderate fibrosis. This became marked in the second portion and was moderate again in the third portion which lay adjacent to a scar.

Myocardium. The posterior walls of both ventricles showed moderate fibrosis with small scars and arteriolosclerosis.

Case II

Clinical Summary

The patient was an 85-year-old female admitted to Cook County Hospital with a two-week history of progressive dyspnea and leg swelling. There was a previous history of hypertension. Physical examination revealed bradycardia, confusion, an audible cardiac third sound and ankle swelling. Emergency transvenous pacing, as well as digitalization were initiated. Despite successful pacing, the patient developed progressive hypoxia, hypotension, and died 12 hours after admission.

Analysis of Electrocardiogram

No previous electrocardiogram was available for analysis. The admission electrocardiogram revealed complete heart block with an atrial rate of 120/min and a ventricular rate of 50/min. The escape rhythm was characterized by wide QRS complexes with beats of both right and left bundle branch block pattern. Multiple premature ventricular contractions were also noted.

Electrophysiological Studies

His bundle recordings were obtained at the time of temporary pacemaker insertion. Complete A-V dissociation was noted with atrial rates of 100-120/min and ventricular rates of 50/min. P waves were followed by H potentials with an A-H interval of 100 msec (figs. 8A and B). These were not conducted to the ventricles. Two escape rhythms were seen, those with right bundle branch block pattern (fig. 8A) and those with left bundle branch block pattern (fig. 8B). Beats with right bundle branch block pattern were not preceded by H' potentials (fig. 8A), suggesting that these were originating within the left ventricle. Escape beats with left bundle branch block pattern were preceded by high frequency potentials (H') with an H'-V interval of 48 msec (fig. 8B).

Autopsy Findings

Aside from the findings in the heart, the pathological diagnoses included: 1) pulmonary emboli, multiple, bilateral; 2) pulmonary edema.

Heart

The heart weighed 532 gms. All the chambers were hypertrophied and enlarged. The mitral annulus presented areas of calcification, which extended to the aortic-mitral annulus. The pars membranacea was somewhat thickened. The coronary arteries were calcific and rigid; however, they were not narrowed.

Histologic Examination: Conduction System

SA node. There was moderately severe arterioloscle-
osis and fatty infiltration. Recent and organizing thrombi were present in the right atrial appendage.

Approaches to SA node. Arteriolosclerosis and fatty infiltration were marked.

Approaches to the A-V node. Fatty infiltration was moderately severe. The A-V nodal artery was moderately thickened.

A-V node. Arteriolosclerosis and fatty infiltration were slight.

A-V bundle, penetrating. There was calcification at the summit of the ventricular septum in the central fibrous body (fig. 9A). The proximal part of this portion of the bundle was intact (fig. 9A). However more distally the calcium impinged upon the bundle (fig. 9B) and produced marked degenerative changes in the distal part of the bundle (fig. 9C).

A-V bundle, branching. There were no changes (fig. 10).

LBB. The main left bundle was separated from the bundle of His by fibrous strands (figs. 10 and 11). The peripheral portion showed Purkinje cells which were smaller than normal.

RBB. Distal to the bifurcation there was moderate fatty infiltration. Still more distally it was normal.

Myocardium. Fibrosis and arteriolosclerosis of the left ventricular septum was moderate (fig. 10) and of the right ventricle, mild. The atrial septum showed slight fibrosis.

Discussion

Electrophysiological studies suggested that both our cases had His bundle disease, with H being recorded proximal to, and H' recorded distal to an area of conduction delay in the His bundle. Validation of the proximal potentials (H) depends upon demonstrating that this potential is not generated by atrium. In case I, the prolongation of S-H and A-H with single and coupled atrial pacing suggested that H was not part of the atrial electrogram. This interpretation is supported by the histologic findings in case I. The approaches to the A-V node and the A-V node show insignificant
changes, a finding which is consistent with this patient's normal A-H interval. At the same time the proximal part of the bundle of His was intact.

Likewise in case I, if one accepts H as being a His bundle electrogram, then H' is presumably a His bundle potential recorded distal to H, or a right bundle branch electrogram. If H' were a proximal right bundle branch potential, then the site of right bundle branch block must be distal to this recording site, since H' is not delayed relative to the QRS. The prolongation of H-H' with single and coupled atrial pacing would then reflect a prolongation of conduction time between the His bundle and the proximal right bundle branch. This would not affect distal complete right bundle branch block. However H' would move into the QRS complex. The fact that H'-V remained constant as H-H' lengthened suggests that H' was recorded from the His bundle. Prominent atrial and ventricular electrograms were recorded from the catheter electrode when H and H' were recorded suggesting that the catheter was in the usual position for recording His bundle and not right bundle branch activity. 21 The H'-V interval was within the normal range for H-V interval despite the severe pathologic change in the left bundle branch (right bundle branch block).

Serial sections were consistent with H' being recorded from either the distal His bundle or the right bundle branch, since the beginning of right bundle branch was intact and the functional integrity of the distal portion of the bundle appeared to be intact in its superior half (away from the calcium).

In case II, the pattern of splitting was somewhat more complex. A proximal potential (H) followed every P and was not conducted to the ventricles. The distal potential (H') was only seen preceding escape beats with left bundle branch block pattern. Escape beats with right bundle branch block pattern were not preceded by these potentials. Several combinations of lesions could explain these electrophysiological findings. These would be as follows: 1) Two lesions, both producing complete block, one located in the His bundle and the other in the left bundle branch (fig. 12A). H could be recorded from the His bundle proximal to the site of block. Two escape rhythms could occur: one arising from the His bundle distal to the site of block; this would be preceded by H' potentials and conducted with left bundle branch block; the other
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Figure 9

Left panel. Case II. Bundle of His, penetrating portion. Note that it is intact. Weigert-van Gieson stain × 17. B = bundle; C = calcific mass; V = ventricular septum; CFB = central fibrous body; A = artifact of microtome knife. This tissue was not decalcified. Middle panel. Case II. Calcium impinging upon bundle of His. Hematoxylin-eosin stain × 17. B = bundle; C = calcific mass; V = ventricular septum; A = artifact of microtome knife. This tissue was not decalcified. Right panel. Case II. Degenerative changes in the A-V bundle. Hematoxylin-eosin stain × 150.

could arise in the left bundle branch system distal to the site of left bundle branch block. This latter rhythm would be characterized by right bundle branch block pattern and QRS would not be preceded by H' potentials. 2) Two lesions, one destroying the His bundle distal to the H recording site and the other producing delays in the right bundle branch system (fig. 12B). Two escape rhythms could be postulated, one arising from the right bundle branch and the other from the left bundle branch. The former would produce left bundle branch block pattern. The H' potential would be a proximal right bundle branch potential. The long RB-V (normal RB-V being 15-25 msec) would have to reflect an additional (third) lesion producing slowing in the distal right bundle branch. 3) Two lesions, one in each bundle branch (fig. 12C). Again H' potentials would be right bundle branch potentials, with additional delay in the right bundle branch (third lesion) accounting for the relatively prolonged RB-V interval.

Of these three possibilities, electrophysiologically the first seems most likely in that it implies that H' was recorded from the distal His bundle and not the right bundle branch. This was consistent with the position of the recording electrodes. This combination of lesions also is consistent with a pattern of lesions previously associated with complete heart block, e.g., the type B lesion described by Davies.22

The pathological findings also were in accord with the first possibility, in that two major lesions were seen with serial section, one in the His bundle and the other in the left bundle branch. It should be noted that there was healthy appearing His bundle tissue both proximal and distal to the His bundle lesion. We would postulate that H was generated from the His bundle proximal to this lesion, and that H' was generated by tissue distal to the lesion.

Clinical and Pathological Implications

Two patterns of splitting are described in this report, one occurring with intact conduction, and the other occurring with complete heart block. The former case did have an episode of complete heart block prior to the electrophysiological studies in which electrocardiograms during block were consistent with a site of block in the His bundle. Both cases had a pathological common denominator, this being the presence of a calcific lesion impinging upon the His bundle, with the appearance of healthy His bundle tissue proximal and distal to the lesion. Thus, this study appears to confirm the
previous speculations that split His bundle potentials reflect the presence of interruptive lesions in this structure.

The natural history of such conduction lesions is not known, and it is difficult to recommend appropriate therapy. Perhaps case I should have been treated with a permanent demand pacemaker. In both of our cases, other pathological findings may be implicated as direct cause of death, e.g., calcific aortic stenosis and pulmonary edema in case I, and multiple pulmonary emboli with congestive heart failure in case II.

Conclusion

The split His bundle potentials as previously postulated is produced by a delay in an area of bundle of His with healthy tissue proximal and distal to it. The healthy tissue proximal is responsible for H and healthy tissue distal is responsible for H'.
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