Electrophysiological and Histological Correlations in Two Cases of Complete Heart Block

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SUMMARY

Electrophysiological and histological correlation was documented in 2 cases of complete heart block (CHB).

Case 1 (77 yrs. female): CHB continued for 1.5 years with normal QRS in axis and duration. The His bundle electrogram (HBE) showed A-H block with normal H-V interval (40 msec). Ten months later she died of bronchopneumonia and paralytic ileus. Histological study of the conduction system revealed a giant nodule of calcification in the mitral ring and central fibrous body, which compressed the end of the A-V node and most part of the penetrating portion of the A-V bundle. Above-mentioned findings were thought to be compatible with A-H block in HBE.

Case 2 (82 yrs. male): His ECG showed CHB with QRS configuration of right bundle branch block (RBBB) with left axis deviation (LAD), with transient appearance of RBBB with right axis deviation (RAD) or left bundle branch block (LBBB) patterns. The HBE revealed normal PH interval (120 msec), and the site of block was distal to the A-V bundle. In addition to the H potential, spike 'X' was also recorded 20 msec before QRS wave. He died suddenly after 1.5 years of hospitalization. Histological examination revealed that both anterior and posterior fascicles of the left bundle branch showed severe destruction at their origins, and the right bundle branch also showed marked fibrosis. These histological findings were so-called 'trifascicular block' and compatible with H-V block in HBE. And the meaning of the 'X' spike in HBE was discussed.

Additional Indexing Words:
Complete heart block  His bundle electrogram  A-H block  H-V block  Mitral ring calcification  Trifascicular block

RECENT development of clinical application of His bundle electrography has been making it possible to interpret various degrees of atrio-
ventricular (AV) block with more accuracy. But there have been only a few cases, who were examined both by the HBE and the histological study of the conduction system. In this communication we reported 2 cases of CHB, one of whom showed A-H block and the other H-V block, and tried to correlate the electrophysiological and histological findings.

CASE REPORTS

Case 1: A 76-year-old female entered the hospital with chief complaints of edema and dyspnea. Her ECG showed normal sinus rhythm and no axis deviation at the age of 74 (Fig. 1a), and the ECG on admission showed complete heart block with ventricular rate of 40/min and QRS pattern of RBBB+RAD (Fig. 1b). Two weeks later QRS complex changed to the pattern of normal width with no axis deviation (Fig. 1c), which was a predominant pattern during her clinical course. Auscultation of the heart on admission revealed a systolic murmur (Grade 2/6) and cannon sounds. Phonocardiogram confirmed these findings and further revealed a diastolic murmur (Fig. 2). X-ray films of the chest revealed a calcification of mitral ring of 'J' shape and a cardio-thoracic ratio of 58%. Five months

![Fig. 1. Electrocardiograms of Case 1, a) taken at 74 years of age, showing normal sinus rhythm and no axis deviation, b) taken at 76 years of age, showing complete A-V block with a ventricular rate of 40/min and QRS pattern of RBBB+RAD, c) showing CHB with QRS pattern of normal width and no axis deviation, d) taken during Adams-Stokes' attack, showing a ventricular rate of 16/min, e) showing CHB with QRS pattern of LBBB, f) taken after the implantation of a permanent pacemaker, and g) showing normal sinus rhythm even after the implantation of a pacemaker.]
after admission she developed a syncope with heart rate of 16/min (Fig. 1d). Administration of isoproterenol and temporary pacing were effective for this episode. A bipolar electrode was inserted to the right heart and the HBE was recorded on the direct-writing recorder according to Scherlag’s method (Nihonkoden rectigraph RGJ 3004, electronic amplifier AVB-2 with a time constant of 3 msec and

Fig. 2. Phonocardiogram of Case 1, showing ejection type systolic murmur (SM) and diastolic murmur (m) following atrial sound (A).

Fig. 3. His bundle electrogram and its schema of Case 1, showing A-H block with normal H-V interval.
paper speed 25–100 mm/sec). Also 4-channel tape system was utilized for further analysis. H-V interval was 40 msec and A-H block was diagnosed (Fig. 3). After 8 months a permanent pacemaker of demand type (Medtronic 5942) was implanted (Fig. 1f). But thereafter her ECG occasionally showed 2:1 A-V block or normal sinus rhythm (Fig. 1g). After 1 year and 5 months of CHB she suffered from bronchopneumonia and paralytic ileus, and died.

Autopsy showed the heart weight of 330 Gm and moderate coronary sclerosis without myocardial infarction. The calcification of posterior mitral annulus was found, which was spreading over the ventricular septum, proved by soft X-ray films (Fig. 4). The conduction system was examined as previously reported according to Lev’s method. The end of A-V node was compressed by a large nodule of calcification encompassing from the summit of the ventricular septum to central fibrous body (CFB) (Fig. 5a). The penetrating portion of A-V bundle was remarkably compressed by this giant calcification of the septum (Fig. 5b), causing severe degeneration of conducting cells. The effect of calcification was slight in the branching portion of A-V bundle, showing slight fibrosis. Left posterior fascicle of the left bundle branch was severely fibrotic (Fig. 6a) but lesion of the left anterior fascicle was slight. Right bundle branch revealed slight fibrosis in the first and second portions but third portion of the right bundle branch was markedly interrupted by

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Fig. 4. Soft X-ray film of postmortem heart of Case 1, showing mitral ring calcification (MRC) and calcification of the top of the septum (arrows).
Fig. 5. Case 1, a) the end of atrioventricular node (N) and b) the penetrating portion of the A-V bundle (B), both compressed by giant nodule of calcification of ventricular septum (arrows).
subendocardial fibrosis and endocardial fibroelastosis of the right ventricle (Fig. 6b).

Case 2: An 81-year-old male was admitted to the Yoiku-in Hospital with a chief complaint of occasional exertional vertigo. He had never experienced any syncopal attacks and at the age of 78 he was diagnosed to have hypertension. Physical examination on admission revealed a well-developed aged man. The blood pressure was 200/94 mmHg, heart rate 42/min, which was CHB with QRS pattern of RBBB+LAD in ECG (Fig. 7a). Laboratory data showed slight anemia, proteinuria and positive serological test for syphilis. The X-ray films of the chest had no signs of congestion with cardio-thoracic ratio of 58%. The cardiac index was 2.40 L/min/M² by the dye dilution method. Treatment was done by antihypertensive drugs and ß-stimulating agent. Two weeks after admission he had bronchopneumonia, when his ECG showed transient 2:1 A-V block (Fig. 7c). About a half year later the blood pressure was maintained at the level of 150/
70 mmHg. At the age of 82, the HBE revealed normal PH interval (120 msec) and another spike (X) with 20 msec before QRS wave (Fig. 8). The site of block was diagnosed to be distal to A-V bundle. After 35 days from the recording of HBE he died unexpectedly.

Autopsy was performed. The heart weight was 400 Gm with hypertrophy of the left ventricle. Coronary arteries showed moderate sclerosis, but there were no

Fig. 8. His bundle electrogram of Case 2, showing PH interval of 120 msec and besides H potential ‘X’ spike was recorded (XQ interval: 20 msec).

Fig. 9. Case 2, the penetrating portion of A-V bundle, revealing about 50% replacement of conducting cells by the fatty tissue.
Fig. 10. Case 2, branching portion of A-V bundle (B) was almost intact but a) the posterior fascicle and b) the anterior fascicle of the left bundle branch disclosed complete interruption of the conducting cells by marked fibrosis (arrows).

abnormalities in the valves. Histological examination revealed scattered myocardial fibrosis but no infarction. The study of the conduction system showed that the S-A node and A-V node were almost intact, with patency of S-A nodal and A-V nodal arteries despite of moderate sclerosis of main coronary arteries. The lower part of penetrating portion of A-V bundle was replaced by fatty tissue in almost 50% (Fig. 9). The branching portion of A-V bundle itself showed moderate changes. Both anterior and posterior fascicles of the left bundle branch revealed complete interruptions of the conducting cells, which were localized in the proximal portion of the left bundle branch (Fig. 10a, b). The right bundle branch showed fibrotic changes in the terminal half of the first portion and the entire second portion (Fig. 11a, b).

DISCUSSION

The histological studies of A-V block have been accumulated since the works by Yater,9) Lev,10) and Lenègre.11) We have also reported the histological studies of the conduction system with various conduction disturbances.7),12)–14) In these works the principal sites of lesions in chronic A-V block have been demonstrated in the branching portion of the A-V bundle
including the beginning of the bundle branches. Despite of newly acquired informations on the A-V block by the His bundle electrograms, there have been only sporadic reports on the comparison of the findings of HBE and of the histology of the conduction system. Rosen, Lev et al\(^1\)-\(^4\) have reported 10 cases from the view point of these correlations. They demonstrated that A-H block was induced by the lesion of the A-V node and its approaches and H-V block resulted from the lesions of the bundle branches. Hunt et al\(^5\) reported 7 cases of heart block complicating acute myocardial infarction, which showed a good correlation between the changes in HBE and histology.

In our 2 cases A-H block in the first case was induced by the lesions of lower part of the A-V node and the penetrating portion of the A-V bundle due to compression by a giant mass of calcification in the ventricular septum and central fibrous body. Remaining cells of penetrating portion of the A-V bundle with degeneration might explain that the case transiently showed normal sinus rhythm. Some cases of CHB were believed to be induced by calcification of central fibrous body and/or septal summit as reported by Nagayyo,\(^15\) Yater,\(^9\) and Korn et al.\(^16\) We previously reported a case of CHB with atrial flutter, resulting from the similar calcification.\(^17\) As in this first
case heart block induced by mitral ring calcification and its involvement of septal summit and complicated apical diastolic murmur was called Rytand's syndrome.\textsuperscript{18,19} Calcified lesions of the A-V bundle resulted in split His bundle potentials in 2 cases of Bharati et al.\textsuperscript{4} Marked difference from our case was the localization of the calcification. In our case it extended from the A-V node to the almost whole penetrating portion of the A-V bundle (A-H block), while the case of Bharati showed localized calcification, leaving intact portions at both the proximal and distal A-V bundle.

In the second case (H-V block) the site of block was located in the beginning of bundle branches, which might be attributed to pathological 'trifascicular block' postulated by Rosenbaum.\textsuperscript{20} We previously reported\textsuperscript{21} a typical case of 'trifascicular block' and correlated the electrocardiographic changes and the histology of the conduction system. In the case presented here there was another spike ('X' spike) in addition to the H potential in the HBE. This apparently suggested 'block within His bundle'.\textsuperscript{22} But, since the interval of X-Q was too short for the potential originating from His bundle and the histological study showed an incomplete lesions (about 50\%) in the lower penetrating portion of A-V bundle, intra-His block seems unlikely. Although the configuration of QRS on a surface electrocardiogram was RBBB + RAD pattern, it is not ruled out that the spike 'X' might be the potential of probably the remaining first portion of the right bundle branch. However, almost complete interruption at the second portion of the right bundle branch seems to be a difficulty for this explanation. It may reflect a retrograde excitation of the A-V bundle evoked by the anterior fascicle of the left bundle. But from the histological changes of the conduction system such hypothesis may be difficult. Although the potential of the left bundle branch has never been recorded by the right heart catheterization, the 'X' spike may be a potential of left bundle branch itself, because the length of complete interruption of the left bundle branch was short and the remaining peripheral fascicles were large and intact and because the interval of X-Q is compatible with that reported by Rosen\textsuperscript{23} on the left bundle potential.

Our study disclosed that the A-H block on HBE indicated the sites of block in not only the A-V node but also in the penetrating portion of the A-V bundle in so far as the lesions continued from the A-V node. Also the lesions in branching portion of the A-V bundle might result in H-V block in so far as the lesions continued to the beginning of both bundle branches. There must be an accumulation of cases to conclude the sites of block from the HBE.
REFERENCES


