HIS BUNDLE ELECTROGRAM IN RHEUMATIC MITRAL VALVE DISEASE WITH SPECIAL REFERENCE TO BACHMANN'S BUNDLE BLOCK IN P MITRALE

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SINCE Scherlag *et al.*¹ first described a simple electrode catheter technique for recording His potential (H) in man in 1969, many studies utilizing this method and its modification have achieved overwhelming clarification of electrophysiological aspects of various cardiac arrhythmias and conduction defects.^{2,3} However, application of His bundle electrogram (HBE) with accompanying surface electrocardiogram (ECG) in assessment of interatrial conduction in patients with mitral valve disease and P mitrale has been limited.⁴

For the intraatrial conduction, there has been described fibers with the histologic characteristics of Purkinje-like tissue extending in three distinct tracts (anterior, middle and posterior internodal tracts) from the sinus node to the atrioventricular node. One of these, the anterior internodal tract branches to the left atrium through Bachmann's bundle (interatrial band).^{5,6} The Bachmann's bundle first described by Bachmann⁷ in 1916, is the shortest and most direct route between the atria, conducting the cardiac impulse from the right to the left atrium.

The notched sinus P wave of 0.12 seconds or longer in mitral valve disease, "P mitrale", has recently been attributed to represent a specific left atrial block--- Bachmann's bundle block.⁸ Utilizing HBE along with simultaneously recorded surface ECG, Bekheit⁴ has found the end-point of atrial activation is altered in P mitrale and the H potential is noted preceding the termination of the atrial activation. Recently, we have found that the left ventricular approach for recording HBE can obtain double

A waves with the terminal one possibly representing left atrial excitation recorded at the catheter position in P mitrale.⁹

It is the purpose of this communication to report our results that have been obtained from a total of 15 patients with mitral valve disease and P mitrale. Electrophysiology of auricular fibrillation in 11 patients with chronic rheumatic valvular disease is also described and discussed.

MATERIALS AND METHODS

Twenty-six patients with chronic rheumatic mitral valve disease, 15 in sinus rhythm and 11 in auricular fibrillation, constitute the object of the present study. There were 12 men and 14 women, ranging from 16 to 49 years of age. In all patients, the diagnosis was confirmed by routine cardiac catheterization with or without angiocardiology and at surgery in addition (Table I). The size of the left atrium was evaluated from the chest roentgenograms with barium-filled esophagogram and was classified arbitrarily into mild, moderate or marked dilatation according to the degree of esophageal compression by the enlarged left atrium and the extent of the left atrial enlargement in relation to the spine shadow at the right anterior oblique view. Pulmonary venous hypertension was graded by pulmonary arterial wedge pressure into mild (15–20 mmHg), moderate (21–25 mmHg) and severe (> 25 mmHg) degrees.

HBE was recorded from either the right (16 cases) or left ventricle (2 cases) or simultaneously from both (8 cases). The method

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TABLE 1 CLINICAL DIAGNOSIS OF 26 PATIENTS WITH CHRONIC RHEUMATIC MITRAL VALVE DISEASE

No. of Cases	Sex	Age (yrs)	Rhythm	Clinical Diagnosis
1	F	24	SR	MS
2	M	40	SR	MS
3	F	20	SR	MS, AI
4	F	37	SR	MS
5	F	34	SR	MS, AI
6	F	22	SR	MS, TI
7	F	45	SR	MS
8	F	38	SR	MS
9	F	16	SR	MI, MS
10	M	48	SR	MI, MS
11	M	36	SR	MS, MI
12	M	16	SR	MS
13	M	16	SR	MS
14	M	49	SR	MS, AS
15	M	26	SR	MS
16	F	38	AF	MS, AS, AI, TI
17	M	41	AF	MS, AI
18	M	22	AF	MS, AS, AI
19	F	24	AF	MS, AI, AS
20	F	21	AF	MS, TI
21	M	21	AF	MS, AI
22	F	26	AF	MS
23	F	52	AF	MS, AI
24	F	35	AF	MS, MI
25	M	35	AF	MS, MI
26	M	41	AF	MS

Abbreviations:

MS: mitral stenosis; MI: mitral insufficiency; AS: aortic stenosis;
 AI: aortic insufficiency; TI: tricuspid insufficiency; SR: sinus rhythm;
 AF: auricular fibrillation; F: female; M: male

of recording electrical potentials from the specialized conduction tissues has been described elsewhere.^{9,10} LB potential can also be recorded from the left ventricular approach. The recorded HBE from the left side was validated by simultaneously recorded HBE from the right heart and atrial pacing. The electrical activity was recorded at frequency setting of 40 to 200 cps on an ECG amplifier. All records were recorded on an 8 channel, oscillographic photographic recorder (Electronics for Medi-

cine, DR-8) at paper speeds of 100 and 200 mm/sec. A standard lead II ECG was also simultaneously recorded. All patients were on digitalis prior to the study.

The following measurements were made in milliseconds:

- 1) P₁-P₂ interval: the duration of the P wave in lead II electrocardiogram (P₁ and P₂ designate the beginning and end of atrial depolarization in the ECG, respectively)
- 2) P₁-A interval: the conduction time from

Fig.1. Schematic drawing of a His bundle electrogram (HBE) with simultaneously recorded surface electrograms (ECG), one with normal P wave (top tracing) and the other with P mitrale (bottom tracing). Note that P_2 follows H and P_2H interval is a negative value in P mitrale. In normal P wave, however, the P_2 precedes H and the P_2H interval is a positive value.

TABLE II INTRAATRIAL AND INTERATRIAL CONDUCTION TIME IN 15 PATIENTS WITH SINUS RHYTHM

No. of Cases	HR (min.)	PR interval (msec)	Duration of P wave (P_1P_2 msec)	Intraatrial conduction time (P_1A msec)	Interatrial conduction time (P_1A' msec)	P_2H (msec)
1	93	166	135	26	—	-23
2	93	145	129	31	66	-36
3	109	160	120	18	—	-32
4	96	180	139	33	—	-13
5	96	154	128	41	82	-26
6	102	163	138	18	—	-40
7	110	151	122	33	47	-14
8	84	179	126	26	53	-5
9	65	179	131	31	67	-27
10	98	209	143	30	68	0
11	107	205	140	15	—	+12
12	79	265	158	73	82	+42
13	93	170	107	23	—	+8
14	70	153	92	13	—	+8
15	72	180	110	26	—	+18

Note: Cases 1-12; Sinus rhythm with P mitrale

Cases 13-15; Sinus rhythm with normal P wave

Abbreviation: HR = heart rate; min = minute; msec = millisecond.

TABLE III COMPARISON OF SINUS IMPULSE CONDUCTION IN P MITRALE AND NORMAL P WAVE

Group	No. of Cases	PR interval (msec)		Duration of P wave (P ₁ P ₂ msec)		Intraatrial conduction time (P ₁ A msec)		A-V nodal conduction time (AH msec)		P ₂ H* (msec)		His-Purkinje conduction time (HV msec)	
		Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean	Range	Mean
P mitrale	12	145-265	180	120-158	134	15-73	31	67-140	96	(-5)-(-40)	-24	40-55	46
Normal P	3	153-180	168	92-110	103	13-26	21	96-118	103	8-18	+11	36-51	43

* 3 patients with first degree A-V block were excluded

sinus node to A-V node or internodal conduction time.

3) A-H interval: approximation of the A-V nodal conduction time.

4) H-V interval: His-Purkinje system conduction time.

5) P₂-H interval: the time interval between the completion of atrial activation (P₂) and the beginning of His bundle deflection (H). This value is negative when P₁-P₂ interval is prolonged and P₂ follows H. Schematic drawing of HBE along with surface ECG is shown in Fig. 1.

In 7 patients, double atrial activities (A and A') could be identified on the HBE recorded from the left ventricular endocardial surface with a catheter positioned at the subaortic region. In these patients, P₁A' interval was measured in msec and designated as interatrial conduction time.

RESULTS

Internodal (or intraatrial) and interatrial conduction disturbances in chronic rheumatic mitral valve disease.

Of the total 26 patients with chronic rheumatic mitral valve disease, sinus rhythm was noted in 15, of whom 12 had P mitrale. The pertinent electrophysiological data including P₁P₂ interval (duration of P wave), P₁A interval (internodal conduction time), P₁A' interval (interatrial conduction time), A-H interval (A-V nodal conduction time), P₂H (time duration between the end-point of P₂ and the H potential) and H-V interval (His-Purkinje conduction time) in the 15 patients are summarized in Tables II and III.

The duration of P wave (P₁P₂ interval) in patients with P mitrale ranged from 120 to 158 msec, with a mean of 134 msec. Patients with normal P wave had invariably the duration of less than 120 msec (range 92 to 110, mean 103 msec). Alteration of P morphology in the same ECG tracing was noted in 3 out of 12 patients with P mitrale.

The PR interval was within normal limits (120 to 200 msec) in all but three patients. The internodal conduction time (P₁A interval) was considered to be prolonged when it exceeded 50 msec (normal range 20 to 45 msec).² Patients with P mitrale had longer interval than those with normal P wave, the mean values were respectively 31 and 21 msec. However, prolongation of the internodal conduction time

Fig.2. Simultaneous recording of the surface electrocardiogram (ECG-II) and His bundle electrogram from the right ventricular endocardial surface (HBE RV) in P mitrale. Note that the His potential (H) precedes the termination of the P wave (P₂). A lead II ECG on the top shows characteristic P mitrale.

Fig.3. His bundle electrogram recorded from the left ventricular endocardial surface (HBE LV) discloses double atrial activities (A and A'). The A' follows A and precedes H and P₁ A' interval is 80 msec.

was observed in only one patient (73 msec). This patient also had first degree A-V block but no rheumatic activity was considered to be present at time of the study.

In 9 of 12 patients with P mitrale but no

A-V conduction disturbance, the H potential was noted to precede the termination of the P wave, giving P₂H interval of negative value (Fig. 2). These values ranged from -5 to -40 msec. In the remaining three patients who had

Fig.4. Simultaneous recording of His bundle electrogram from both sides of the heart (HBE LV and HBE RV). Each atrial activity is identical time onset in both HBES. Note that A' deflection can only be identified in HBE LV (top tracing). A 5 msec time lag between onsets of both H deflections is probably insignificant.

TABLE IV CORRELATION OF THE P_1P_2 INTERVAL AND P_2H INTERVAL WITH THE SIZE OF THE LEFT ATRIUM AND THE HEMODYNAMIC DATA IN 15 PATIENTS WITH SINUS RHYTHM

No. of cases	P_1P_2 interval (msec)	P_2H interval (msec)	LA enlargement	PA wedge Pressure (mmHg)
1	135	-23	mild	17.5
2	129	-36	mild	16.0
3	120	-32	moderate	17.5
4	139	-13	mild	—
5	128	-26	mild	29.0
6	138	-40	mild	28.0
7	122	-14	moderate	31.0
8	126	-5	mild	43.0
9	131	-27	mild	24.0
10	143	0	marked	—
11	140	+12	mild	19.0
12	158	+42	mild	23.0
13	107	+8	mild	22.0
14	92	+8	mild	17.0
15	110	+18	moderate	12.5

Note: Cases 1–12; Sinus rhythm with P mitrale

Cases 13–15; Sinus rhythm with normal P wave

Abbreviation: LA: left atrial; PA: pulmonary arterial; msec: millisecond

Fig.5. HBE recorded by left ventricular approach (HBE LV) at a pacing rate of 133 beats/min reveals significant prolongation of PI-A' interval (93 msec) following pacing as compared with P₁A' interval (80 msec) before pacing (Fig. 3). There is almost no significant difference in conduction time in the right atrium as measured from P₁A (38 msec) and PI-A (36 msec) intervals. Note that A-H interval is also prolonged with H potential following the termination of the P wave.

Fig.6. Simultaneous recording of His bundle electrogram from the left ventricular endocardial surface (HBE LV) and right atrial electrogram (RAE). Note that conspicuous H deflections are recorded and each V is preceded by an H. In the absence of a V there is no H recorded. The HV interval is constant, irrespective of the cycle length of the heart.

first degree A-V block the onset of the H potential either coincided with (P₂H = 0) (one patient), or followed the end-point of the P wave with positive P₂H value (two patients). The values in the latter two patients were +12 and +42 msec, respectively. On the contrary, in three patients with normal P wave the H

potential invariably followed the end-point of the P wave and the P₂H interval disclosed positive value (+8, +8 and +18 msec, respectively).

Both atrial deflections (double A waves) in patients with P mitrale could sometimes be clearly demonstrated in HBE recorded from the

TABLE V ATRIOVENTRICULAR AND INTRAVENTRICULAR CONDUCTION TIMES IN 26 PATIENTS INCLUDING SINUS RHYTHM AND AURICULAR FIBRILLATION

No. of cases	PR interval (msec)	AV nodal conduction time (msec)	His-Purkinje conduction time (msec)
1	166	78	38
2	145	74	40
3	160	87	55
4	180	99	48
5	154	72	41
6	163	101	44
7	151	67	51
8	179	84	42
9	179	79	47
10	209	138	41
11	205	136	54
12	265	140	52
13	170	96	51
14	153	97	43
15	180	118	36
16			38
17			42
18			40
19			46
20			38
21			51
22			37
23			52
24			46
25			47
26			43

Note: Cases 1–15: Sinus rhythm

Cases 16–26: auricular fibrillation

Abbreviation: msec: millisecond

left ventricular endocardial surface with a catheter toward the high ventricular septum. This was accomplished in 7 patients. Fig. 3 illustrates a case. The duration of the P wave (P_1P_2) in lead II ECG was 128 msec. The P_2 followed the H deflection and the P_2H interval was minus 26 msec. Both atrial deflections (A and A'), 42 msec apart, were noted. The first A deflection was thought to represent the right atrial activity and this was confirmed by the simultaneously recorded HBE from the right heart (Fig. 4). The time onset of the A wave recorded from the right lower atrium was

almost the same as that of the A recorded by the left ventricular approach. Another atrial deflection (A') could have been related to the left atrial activity recorded at the catheter position. It followed the A wave and preceded the H potential (A 5 msec time lag observed between onsets of both H deflections is probably insignificant). Furthermore, the $PI-A'$ interval was noted to be lengthened from 80 msec to 93 msec on atrial pacing at a rate of 133 beats/min (Fig. 5). On the contrary, P_1A interval measured from the HBE recorded in the right heart was shortened from 38 to 36

msec at the same pacing rate. This might indicate that further interatrial conduction block (Bachmann's bundle block) had occurred by increased heart rate. The interatrial conduction time measured from P_1A' interval in the 7 patients studied ranged from 47 to 82 msec, averaging 66 msec.

Correlation of PIP_2 interval and P_2H interval with size of the left atrium and hemodynamic parameters.

Dilatation of the left atrium in 15 patients with sinus rhythm was considered to be of mild degree in 11, moderate degree in 4 and of marked degree in the remaining one (Table IV). In these three groups of the patients, P mitrale was noted in 9, 2 and one, respectively. The average P_1P_2 interval in mild left atrial enlargement was 136 msec and in moderate left atrial enlargement was 121 msec. It was 143 msec in patient with marked left atrial enlargement. Thus, there was no correlation observed between the left atrial size and the P_1P_2 interval. The P_2H interval also had no relation to the size of the left atrium in 9 of 12 patients with P mitrale and normal A-V conduction. The average value was -24 msec in mild left atrial enlargement, and -23 msec in moderate enlargement.

Likewise, pulmonary arterial wedge pressure had no correlation to either P_1P_2 interval or P_2H interval (Table IV). The pulmonary arterial wedge pressure was slightly elevated in 4 patients, moderately elevated in 2 and markedly elevated in 4. The average P_1P_2 intervals were 131, 145 and 129 msec, respectively. There was no tendency to have more negative P_2H value as the pulmonary arterial wedge pressure became higher and the P_2H interval in these three groups were -30, -27 and -21 msec, respectively.

Atrioventricular and His-Purkinje conduction times in chronic rheumatic mitral valve disease

A-V nodal conduction time (A-H interval) in 15 patients with sinus rhythm and His-Purkinje conduction time (H-V interval) in all 26 patients as measured in millisecond are shown in Table V. The A-V nodal conduction time (normal range 50 to 135 msec in our laboratory data) was prolonged in three patients in whom there were first degree A-V block and P mitrale. These three patients had A-H interval of 138, 136 and 140 msec, respectively. The His-Purkinje conduction time was normal in all and the H-V interval ranged from 36 to 55 msec.

The normal H-V time by the right ventricular approach was 44 ± 6 msec and that by the left ventricular approach was 45 ± 6 msec in our laboratory.⁹

Electrophysiological aspects of auricular fibrillation in chronic rheumatic mitral valve disease

All H potential was noted to be followed by ventricular activity (V) and concealed conduction that did not invade the His-Purkinje system was quite evident. The H-V interval in individual patient was almost constant, irrespective of the cycle length of the heart (Fig. 6).

DISCUSSION

Over the past six decades, since the first description made by Lewis in 1913,¹¹ notched P wave has frequently been noted to associate with mitral stenosis. Currently, this is known as P mitrale which was first termed by Winternitz in 1935.¹² This P mitrale has been attributed to left atrial enlargement, left atrial strain, and more recently to interatrial conduction disturbance or Bachmann's bundle block.^{8,13,14} There is a tendency for P mitrale to occur more frequently in mitral stenosis with larger left atrium and higher left atrial pressure but the P wave abnormalities are not always related to the size or pressure of the left atrium alone.¹⁴ In fact, the P mitrale may occur with normal size of the left atrium, and moreover it can be noted intermittently in the same ECG tracing.^{14,15} In the present series, there were three patients in whom no constant appearance of P mitrale seen in the same ECG tracing. The etiology of P mitrale seems to be better explained by interatrial conduction disturbance (Bachmann's bundle block) and the present study also supported this view. In normally conducted sinus beat, HBE shows H potential always follows the termination of P wave with positive P_2H value. In contrast, patients with P mitrale frequently shows the H potential precedes the end-point of the P wave, giving negative P_2H value.⁴ In acceleration conduction such as WPW or LGL syndromes,^{16,17} there is also H deflection preceding the end of the P wave. However, in these conditions there are shortened intraatrial conduction as well as A-V nodal conduction time and visible delta waves in the surface ECG. In the present series, 9 out of 12 patients with P mitrale were noted to have P_2H value of -5 to -40 msec.

The interatrial conduction disturbance in P

mitrale is better appreciated by demonstrating double atrial activities in HBE recorded by the left ventricular approach. P_1A' interval was all prolonged in 7 patients studied (47 to 82 msec). It has been stated that the left atrium is activated about 40 msec after the right atrium in the normal heart.¹⁸ It is of interest that one patient in whom atrial pacing was carried out there was further prolongation of the interatrial conduction time as judged from PIA' time. This indicates further Bachmann's bundle block might have occurred by increased heart rate. Possibly more advanced block can be produced by further increase in pacing rate just as A-V block induced by atrial pacing. In animal experiments, second degree (2:1) Bachmann's bundle block can be elicited by rapid atrial stimulation along with potassium infusion.⁶ No complete Bachmann's bundle block has, however, been produced by the experiments.

In the present study the left atrial size as well as the mean pulmonary arterial wedge pressure was noted to have no correlation to duration of the P wave or P_2H interval (left atrial block) in patients with P mitrale. This indicates that the left atrial block is not solely the result of left atrial enlargement. The underlying rheumatic process may play an important role for this block. Prolongation of intraatrial conduction time and A-V nodal conduction time was only rarely observed and His-Purkinje conduction time was within normal ranges in all patients.

It has been stated that the major determinants of irregular ventricular response in auricular fibrillation are entrance block, inherent refractory period and concealed conduction in the A-V junctional region.¹⁹⁻²² The present studies on auricular fibrillation in 11 patients with chronic rheumatic mitral valve disease utilizing HBE confirmed that the A-V node was the major zone of concealment. Any H deflection that was not followed by QRS complex was never observed. Therefore, concealed conduction without involvement of His-Purkinje system in all patients did not influence His-Purkinje conduction time. The H-V interval remained constant throughout the tracing, regardless of cycle length of the preceding heart beat in the same patient.

SUMMARY

HBEs were recorded from either the right or left ventricle or simultaneously from both in 26

patients with chronic rheumatic mitral stenosis alone or in association with other mild valvular lesions during the diagnostic cardiac catheterization. Eleven of the patients had auricular fibrillation. Of the remaining 15 patients with sinus mechanism and P mitrale in the surface electrocardiogram, 12 were noted to have H potential preceded the termination of P wave and gave P_2H interval of negative value — so-called "Bachmann's bundle block". Among these, double atrial activities (A and A' waves) could be identified on the HBE recorded from the left ventricular endocardial surface with catheter electrodes positioned at the subaortic region in 7 patients studied. Interatrial conduction time (P_1A' interval) measured in these patients was prolonged in all and ranged from 47 to 82 with an average of 66 msec. Prolongation of intraatrial (or internodal) conduction time was noted in only one patient who also had first degree A-V block and prolonged A-H interval. There was no correlation of either P_1P_2 or P_2H interval to the degree of left atrial enlargement. The P_1P_2 or P_2H interval also had no correlation with hemodynamic parameters. In patients with auricular fibrillation, all impulses unable to conduct to the ventricle were blocked proximal to the His bundle and concealed conduction was not observed distal to it.

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