Current Status and Future Direction of Clinical Electrocardiography: His Bundle Electrogram

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Recordings of His bundle electrogram combined with stimulation method are very useful to diagnose the locations of conduction disturbance, to understand the mechanisms of arrhythmias and to evaluate the electrophysiologic properties of the A-V conducting system. However, there remain some unsolved problems regarding the electrophysiologic interpretations and prognostic implications based on His bundle electrograms.

In this panel discussion, some aspects with regard to the usefulness and limitations of His bundle electrogram will be discussed from our data obtained in 300 patients.

IN 1958, Alanis et al.¹ first recorded extracellular potentials of the His bundle directly in the isolated dog heart using needle electrodes. Recording of its activity in man was first reported by Giraud et al.² in a patient with trilogic of Fallot in 1960. It was Scherlag et al.³ in 1969, however, who were first to report the technic that is currently used by most investigators. Since then, intracardiac electrical recording with or without stimulation method has contributed to us for understanding of many aspects of cardiac electrophysiology, as follows; (1) localization of conduction defects, (2) localization of the site of impulse formation and direction of impulse propagation, (3) the mechanisms of initiation and termination of tachycardia, and (4) the site of actions of drugs.

In this panel discussion on "Current Status and Future Direction of Clinical Electrocardio-

graphy", we would like to discuss some aspects with regard to the usefulness and limitations of His bundle electrogram.

MATERIALS AND METHODS

Electrophysiological studies were carried out in 300 patients ranging in age from 5 to 81 years.

According to Scherlag et al.², 2 to 4 electrode catheters (6F) were introduced percutaneously into both the femoral veins. These electrode catheters were fluoroscopically positioned at the aimed regions in the heart for recording and pacing.

All intracardiac electrograms (filtered at frequency settings of 40 to 500 Hz) and standard electrocardiogram (ECG) Leads, II and V₁ were displayed on a multichannel oscilloscope and recorded on magnetic tape. The records were subsequently reproduced on Mingograf paper at a speed of 100 mm/sec.

Electrical stimuli were provided by a programmable digital stimulator (manufactured by San-ei Sokki, Tokyo) and as pulses of 2 msec in duration at approximately twice diastolic threshold.

Definition of terms⁴

S₁, A₁, H₁ and V₁ represent stimulus artifact,

Key Words:

H-V prolongation
Concealed conduction
Gap phenomenon
Supernormal A-V conduction
Concealed WPW syndrome

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TABLE I CLINICAL AND ELECTROPHYSIOLOGICAL DATA

No.	Name	Age	Sex	ECG	Symptom	H-V (msec)	Pacemaker	Follow-up period (Mo)	Clinical course
1	Y.Y.	20	M	2:1 AVB	+	170	+	120	alive
2	K.Y.	16	F	N	—	65	—	113	alive
3	A.S.	35	M	CHB+af	+	60	+	101	alive
4	H.Y.	51	F	BFB	+	60	+	91	alive
5	M.Y.	66	M	2:1 AVB	+	75	+	85	alive
6	M.M.	23	F	af	—	65	—	65	alive
7	T.S.	31	M	AVJR	+	75	+	61	alive
8	M.H.	48	M	CHB+af	—	60	—	58	alive
9	T.K.	19	F	N	—	65	—	58	alive
10	C.H.	53	F	Wenckebach	—	60	—	50	alive
11	Y.W.	68	M	BFB	—	60	—	46	sudden death
12	F.H.	64	M	BFB	+	60	+	41	alive
13	K.K.	75	M	N	+	60	—	41	alive
14	S.T.	72	M	2:1 AVB	+	180	+	39	alive
15	S.M.	62	F	2:1 AVB	+	95	+	28	alive
16	M.K.	24	F	1° AVB	+	80	—	11	alive
17	Y.K.	26	M	N	—	60	—	10	alive
18	J.O.	40	M	LBBB	+	80	—	10	alive
19	K.Y.	51	M	2:1 AVB	+	265	—	5	alive

Abbreviations:

M = male; F = female; AVB = A-V block; CHB = complete heart block; N = normal; af = atrial fibrillation; AVJR = A-V junctional rhythm; BFB = bifascicular block; LBBB = left bundle branch block; Mo = Month

atrial, His bundle and ventricular electrograms of the basic atrial drive beats, respectively.

S₂, A₂, H₂ and V₂ represent stimulus artifact, atrial, His bundle, and ventricular electrograms of atrial premature beats, respectively.

Functional refractory period (FRP) of the A-V node is the shortest H₁H₂ interval in response to any A₁A₂.

FRP of the His-Purkinje system (HPS) is the shortest V₁V₂ interval in response to any range of H₁H₂ intervals.

Effective refractory period (ERP) of an accessory pathway is the longest A₁A₂ interval at which a delta wave disappears.

RESULTS

1. Property of the HPS as a predictor of high risk

It is natural to assume that patients with H-V prolongation, in particular with bifascicular block, may be at more risk for development of complete heart block than patients without it

may be. However, the value of H-V prolongation as a predictor for future development of A-V block has been controversial^{5,6}

Our data of 19 patients with H-V prolongation (≥ 60 msec) are shown in Table I. Sudden death occurred in only one asymptomatic patient (Case 11). Permanent pacemaker was implanted in 8 of 12 symptomatic patients. Temporary pacemaker was inserted in a patient with A-V block due to acute myocarditis (Case 19). Those patients are all alive.

In addition, we examined the value of the refractory period of the HPS as a predictor of high risk. The FRP of the HPS could be determined by atrial extrastimulus in only 24 patients with 1:1 A-V conduction.

In Fig. 1, the FRP of the HPS (abscissa) is plotted to the FRP of the A-V node (ordinate). The meaning of this plotting is merely to show that such patients with a shorter FRP of the A-V node as seen in Lown-Ganong-Levine syndrome were excluded. Although there was a tendency

Fig.1. Relationship between FRP of the HPS and clinical implication.

that the FRP of the HPS was more prolonged in symptomatic patients, overlap was seen.

2. Clinical verification of electrocardiographic inference on miscellaneous phenomena related to the A-V conduction

A. Dual A-V nodal pathways

In 1956, Moe et al.⁷ suggested that the A-V node could undergo longitudinal dissociation into two pathways (dual A-V nodal pathways). It has been believed that dual A-V nodal pathways are present when sudden prolongation occurs at critical coupling intervals, suggesting a block in a fast A-V nodal pathway, with conduction on a slow A-V nodal pathway. Recently, Denes et al.⁸ stressed the role of dual A-V nodal pathways in the genesis of reciprocal rhythm or tachycardia.

We demonstrated the presence of dual A-V nodal pathways in 51 of 156 patients (32.7%) except Wolff-Parkinson-White (WPW) syndrome. A-V nodal reentrant tachycardia was electrophysiologically induced in only 15 of these 51 patients (30%).

B. Gap phenomenon

The phenomenon of gap in the A-V conduction was also originally described by Moe et al.⁹ in 1965. The term gap defines a time zone within the cardiac cycle during which premature impulse fails to propagate to the ventricles or atria while impulse of greater and lesser prematurity induce ventricular or atrial responses. A total of 6 types of gap phenomenon have been described; 4 types for antegrade conduction and 2 types for retrograde conduction.¹⁰

We demonstrated gap phenomenon in 15 of 300 patients (Table II). Three types for antegrade conduction were seen in Case 5 and 2 types for retrograde conduction in Case 9, in the same patient.

Fig. 2 shows a representative example (Case 3). The atria were driven at a cycle length of 820

TABLE II DATA FROM 15 PATIENTS SHOWING GAP PHENOMENA

No.	Name	Age	Sex	Type	
				Antegrade	Retrograde
1	N.S.	31	F	1	
2	M.E.	35	F	1	
3	M.U.	49	F	2	
4	A.I.	16	F	2, 4	
5	Y.K.	6	F	1, 2, 5	
6	T.H.	60	M	4	
7	F.T.	47	F	4	
8	M.M.	50	M	5	
9	Y.T.	34	M	5	2
10	R.S.	27	F		1
11	Y.O.	68	M		2
12	K.S.	46	M		2
13	M.K.	32	F		2
14	T.D.	28	M		2
15	K.S.	51	F		2

Classification of gap phenomena was adopted from Damato et al.¹⁰

Fig.2. Gap phenomenon.

The recordings from top to bottom are a high right atrial electrogram (HRA), His bundle electrogram (HBE) and standard ECG Leads, V₁ and II. Intervals are expressed in milliseconds.

The above abbreviations will be used for subsequent figures.

msec (S₁). Atrial premature stimulus (S₂) delivered at a cycle length of 430 msec was conducted to the ventricles with slight prolongation of A-H interval, but with a constant H-V interval (A). An earlier stimulus of coupling interval of 390 msec caused a prolongation of H-V interval and right bundle branch block pattern (B). Further earlier stimulus by 10 msec resulted in block in the HPS (C). At coupling interval of 360 msec, A-V conduction recovered due to time-consumption in H-V interval. This finding suggested that the initial block localized in the distal portion of the HPS and recovered with the delay in the proximal portion of the HPS. It is noteworthy, in D of Fig. 2, that critical conduction delay in the HPS allowed impulse to cause reentry to the atria. This finding suggests macroreentry from the right bundle branch to the left bundle branch since the conducted beat showed left bundle branch block pattern.

C. Concealed conduction

The concept of concealed conduction first described by Langendorf¹¹ in 1948 is defined as the effect of a non-propagated impulse.

The recordings of His bundle electrogram have permitted us to localize more precisely the area within the A-V conduction system where the impulse is blocked and also the area where conduction of subsequent beat is prolonged. The prolongation of the P-R interval after interpolated ventricular premature beat is the most obvious example of this phenomenon.

Such an example is shown in Fig. 3. In this case, the first beat in the figure seemed to be of fascicular origin in view of the shorter H-V interval (25 msec) and the slightly deformed QRS complex. It was judged that the prolongation of the A-H interval of the next beat of sinus origin was attributable to retrograde invasion of the impulse from the ectopic focus to the A-V node and that the site of ectopic focus was localized in the right bundle branch because configuration of the beat showed right bundle branch block pattern with constant H-V interval.

D. Supernormal A-V conduction

Supernormal A-V conduction is the term applied to the phenomenon in which conduction of impulse is better than expected.¹² Since the

Fig.3. Concealed conduction.

Note prolongation of the A-H interval.

Abbreviations:

A = atrial potential; H = His bundle potential; V = ventricular potential; H' = retrograde His bundle potential; CRBBB = complete right bundle branch block

Fig.4. Pseudo-supernormal conduction due to dual A-V nodal pathways.

advent of His bundle electrogram, alternative mechanisms have been persuasively applied to explain most of clinical examples of "supernormal conduction".¹³ They include (1) gap phenomenon, (2) peeling back refractoriness, (3) dual A-V nodal pathways, (4) phase 4 block, (5) summation, etc.

As mentioned before, gap phenomenon was the most common mechanism of apparent super-

normal conduction in our cases.

Fig. 4 shows a case of apparent supernormal conduction, in which the mechanism of dual A-V nodal pathways could be easily invoked to explain it. The atria were driven at a cycle length of 710 msec. When an atrial premature stimulus was delivered at a cycle length of 540 msec, conduction time in the A-V node suddenly shortened (middle panel). Therefore, one may assume

Fig.5. A-V conduction curves.

CL signifies basic cycle length.

An arrow shows conduction on a fast pathway. This corresponds with the sudden shortening of A_2-H_2 interval in the middle panel in Figure 4.

the presence of supernormal conduction because the timing of this stimulus coincides with the top of the T wave of the preceding beat. It will be

TABLE III MECHANISMS OF PSVT IN 43 PATIENTS WITH NO VENTRICULAR PRE-EXCITATION

<i>Reentrant</i>	
<i>A-V nodal</i>	15
<i>Intraatrial (sinus nodal)</i>	5
<i>Concealed WPW</i>	12
<i>Automatic</i>	1
<i>Unidentified</i>	10
<i>Total</i>	43

easily understood, however, that mechanism of dual A-V nodal pathways participated in occurrence of this paradoxical phenomenon when A-V conduction curves were constructed as seen in Fig. 5. In this case, it was considered that conduction pathway in the A-V node changed again from a slow pathway to a fast pathway in consequence of a delicate difference of timing.

3. Concealed WPW syndrome

Recent electrophysiological studies have clarified that an accessory pathway capable of only retrograde conduction participates in induction of paroxysmal supraventricular tachycardia (PSVT)^{14,15} and such a case may be more frequent than realized.^{16,17} The syndrome of such kind is designated as concealed WPW syndrome, because any pattern of ventricular pre-excitation

TABLE IV ELECTROPHYSIOLOGIC DATA FROM 12 PATIENTS WITH CONCEALED WPW SYNDROME

No.	Name	Age	Sex	Location of AP	Cycle length of PSVT	Refractory period of AP
1	Y.K.	68	F	S	430	280
2	K.K.	41	M	L	330	270*
3	T.S.	26	M	L	340	200
4	S.M.	52	M	L	370	290*
5	T.N.	46	M	L	320	210*
6	M.K.	43	M	L	330	330*
7	A.M.	58	M	S	320	190*
8	T.S.	36	M	L	305	190*
9	T.O.	39	F	L	325	—
10	K.A.	31	M	L	270	260
11	Y.K.	36	M	L	320	280*
12	T.N.	26	M	R	400	250*

*The ERP of the ventricular myocardium exceeded that of AP.

Abbreviations: S = septal; L = left-sided; R = right-sided; AP = accessory pathway

is not recognized on standard ECGs.

We investigated the mechanisms of PSVT in 43 patients who had never shown ventricular pre-excitation. The results are shown in Table III. Concealed WPW syndrome was identified in 12 patients. The electrophysiological data are shown in Table IV. As seen in the figure, the left-sided accessory pathway was the most common.

DISCUSSION

1. Limitations of His bundle electrogram

It has been expected that His bundle electrogram may provide means for separating patients with high risk from others less so, and deciding indication of pacemaker implantation. Namely, the prognosis of patients with prolonged H-V interval would be worse than ones with normal H-V interval. However, the value of H-V prolongation as a predictor for future development of complete A-V block has been considered controversial up to the present.^{5,6} For example, some investigators have found prolonged H-V intervals in most patients with bifascicular block associated with documented intermittent heart block and have, therefore, recommended permanent pacemaker in all such patients and bifascicular block even when the patients were a symptomatic.¹⁸⁻²¹ In contrast, others have recommended that no pacemaker be implanted in patients without neurologic symptoms regardless of the presence of marked (≥ 80 msec) H-V prolongation.²² Furthermore, Peter et al²³ recently concluded that prophylactic pacemaker insertion in a subset of largely symptomatic patients with chronic bundle branch block and long H-V interval did not protect against sudden death and therefore, presenting neurologic symptom may be an inadequate guides to assess the need for permanent pacing in bundle branch block.

Thus, the controversial results we found in literature have thrown us into confusion, but it seems that the value of recordings of His bundle electrogram was considerably lower than would be expected. Our results also suggested that neither prolongation of H-V interval nor that of FRP of the HPS provided exact indication of any high risk group. This may be attributable to the fact that the conduction property of the HPS is prone to be subjected to the "all or none" law.

2. Usefulness of His bundle electrogram

There are a number of reviews on usefulness of His bundle electrogram in the diagnosis and

management of selected patients.²⁴⁻²⁹

Recently, it has been demonstrated that reentry due to dual A-V nodal pathways is common mechanism of PSVT.³⁰ We as well as others³¹ demonstrated that dual A-V nodal pathways are not uncommon finding. According to Denes et al,³¹ frequency of induction of PSVT in these patients was 41.4% while our results showed figure of 32.7% in incidence.

On the other hand, it is suggested that concealed WPW syndrome, exact diagnosis of which is difficult without electrophysiologic studies, is the second most common mechanism of PSVT. The incidence of the syndrome is reported to be 15 to 30%.³² Our data is also similar to others' (28%). However, popularization of electrophysiologic studies will reveal that concealed WPW syndrome is responsible for a further great number of PSVT. It is noteworthy that an accessory pathway was located in the septal region in 2 patients and between the right atrium and ventricle in one patient, since nearly all of the accessory pathways in hitherto reported cases involved the left atrium and ventricle.^{33,34} In any case, precise understanding of mechanism of PSVT is important, because the drugs should be chosen specially according to the electrophysiologic properties of various antiarrhythmic agents for a given PSVT.

Furthermore, recordings of His bundle electrogram combined with stimulation method have indicated the electrophysiologic explanation for other miscellaneous phenomena, i.e., gap phenomenon, concealed conduction, etc. and forced an interpretative reevaluation of the surface ECGs as mentioned before.

3. Future development of His bundle electrogram

In the future, many of yet unsolved problems will be solved. Furthermore, new technics may be developed to record the sinus nodal and A-V nodal potentials and to evaluate sinus nodal function more accurately.

Future direction of His bundle electrogram may be summarized as follows: (1) solution of problems on the intracardiac events, (2) more accurate prediction of high risk patients, (3) investigation for mechanisms of arrhythmias combined with echocardiography, etc., (4) judgement of effect of drugs including new antiarrhythmic agents on the more localized site of the A-V conduction system, (5) recordings of extracellular potentials of the sinus node, A-V node and

accessory pathways and (6) refinement and clinical application of surface His bundle electrogram.

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