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Original Article

A proposal of clinical ECG index "vagal score" for determining the mechanism of paroxysmal atrioventricular block

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ABSTRACT

Background: Paroxysmal atrioventricular block (P-AVB) is a well-known cause of syncope; however, its underlying mechanism is difficult to determine. This study aimed to evaluate a new ECG index, the "vagal score (VS)," to determine the mechanism of P-AVB.

Methods: We evaluated the VS in 20 patients with P-AVB (13 men, 7 women; aged 25–78 years [mean, 59.3 years]). The VS was developed by assigning 1 point each for the following: (1) no AVB or intraventricular conduction disturbance on the baseline ECG, (2) PR prolongation immediately before P-AVB, (3) sinus slowing immediately before P-AVB, (4) initiation of P-AVB by PP prolongation, (5) sinus slowing during ventricular asystole, and (6) resumption of AV conduction with PP shortening, and by assigning –1 point each for (7) the initiation of P-AVB by a premature beat, and (8) resumption of AV conduction by an escape beat. Based on the clinical situations and electrophysiologic findings, we considered the mechanism of P-AVB as vagally mediated or intrinsic conduction disease (ICD).

Results: The VS ranged from 5 to –2 points for each patient. Five patients with a definite vagally mediated P-AVB had high VSs (3–5 points). We observed characteristic ECG findings of ICD consisting of changes in AV conduction by an extrasystole and/or escape beat in only 5 of the 6 patients (83%) with a low VS (1 to –2).

Conclusions: The VS is simple and potentially useful for determining the mechanism of P-AVB. P-AVB with a VS ≥ 3 strongly suggested a vagally mediated mechanism.

1. Introduction

Paroxysmal atrioventricular block (P-AVB) is a well-known cause of syncope. However, it is probably underreported because of poor recognition, limited information in medical literature, its unpredictability, and the lack of a clear marker for AV conduction disease between culprit episodes [1]. In addition, the underlying mechanism of P-AVB is difficult to determine. Identifying the etiology of P-AVB is important because cases of vagally mediated P-AVB are usually benign and do not necessarily require cardiac pacemaker implantation [2]. Although ECG findings are reported to be important for predicting the mechanism of P-AVB [1,2], no clinically useful ECG scoring system for P-AVB is currently available. This study aimed to evaluate a new ECG index, the "vagal score (VS)," for determining the mechanism of P-AVB.

2. Material and methods

2.1. Clinical characteristics of the study population

The study population consisted of 20 patients (13 men and seven women), with a mean age of 59.3 ± 14.7 years (range, 25–78 years). These patients had clinically documented P-AVB with clear ECG recordings in our institutions. P-AVB was defined as a sudden onset of complete AVB with two or more consecutive blocked P waves and ventricular asystole of > 3 s. The clinical characteristics of the study patients are shown in Table 1. Their symptoms were syncope in 13 patients (including 1 patient with convulsion during

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Table 1
Clinical characteristics of study patients.

Case	Age/Sex	Symptom	CD on Baseline ECG	CV Disease	Situation/Triggers	Max Pause (s)	Diagnostic Tool	EPS	PMI	Vagal Score	Mechanism
1	25/M	syncope	none	none	head-up tilt testing	10.5	ECG monitor	not done	No	5	Vagal
2	55/M	syncope	none	none	cough/CSM	7	ECG monitor	not done	No	4	Vagal
3	34/F	syncope	none	none	daytime	3.6	ECG	not done	Yes	4	
4	45/M	syncope	none	CAD, MI	swallowing	4.3	Holter	normal	No	3	Vagal
5	78/M	syncope	1st-degree AVB	CAD, MI, HT	swallowing	5.6	Holter	not done	Yes	3	Vagal
6	74/M	syncope	1st-degree AVB	HT	swallowing/Valsalva	8.1	Holter	AH prolongation	Yes	3	Vagal
7	49/M	none	none	MVP	daytime	4.5	Holter	normal	No	3	
8	74/M	faintness	1st-AVB, RBBB, LAD	CAD, HT	daytime/nighttime	3.8	Holter	not done	Yes	3	
9	75/F	convulsion	none	none	post-abdominal ope.	20	ECG monitor	normal	No	2	
10	57/F	none	none	none	post-abdominal ope.	10	ECG monitor	not done	No	2	
11	71/F	syncope	none	HT	daytime	4.2	Holter	normal	Yes	2	
12	49/M	none	IVCD	HT	daytime	4.7	Holter	normal	No	2	
13	47/F	faintness	none	none	sleeping	4.5	Holter	normal	Yes	2	
14	59/M	syncope	Intermittent RBBB	none	daytime	3.8	ECG	intra-His block*	Yes	2	ICD
15	61/M	syncope	1st-degree AVB	none	daytime	16.5	ECG monitor	AH blodk*	Yes	1	ICD
16	54/M	faintness	1st-degree AVB	HT	walking	4.4	ECG	normal	Yes	0	
17	76/F	syncope	none	cardiac tumor	daytime/nighttime	11	ECG monitor	not done	Yes	−1	
18	69/M	syncope	RBBB	none	on the job	9.4	ILR	not done	Yes	−1	
19	71/M	syncope	RBBB	CAD	daytime	60	ILR	not done	Yes	−2	
20	62/F	faintness	1st-AVB, RBBB, LAD	HT	daytime	6.8	ECG	AH block*	Yes	−2	ICD

CD: conduction disturbance, CV: cardiovascular, EPS: electrophysiological study, PMI: pacemaker implantation
CSM: carotid sinus massage, CAD: coronary artery disease, MI: myocardial infarction, AVB: atrioventricular block, HT: hypertension, MVP: mitral valve prolapse
IVCD: intraventricular conduction disturbance, RBBB: right bundle branch block, LAD: left axis deviation, ILR: implantable loop recorder
ICD: intrinsic conduction disease
* bradycardia-dependent AVB

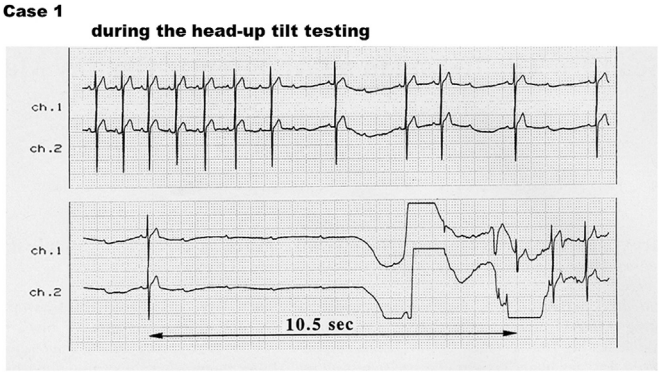

Fig. 1. Episode in case 1. Paroxysmal atrioventricular block (P-AVB) was induced during the head-up tilt test [3]. The vagal score (VS) was 5 points: normal baseline ECG, PR prolongation and sinus slowing immediately before P-AVB, initiation of P-AVB by PP prolongation, and sinus slowing during ventricular asystole.

sleep), faintness in 4, and none in 3. Baseline 12-lead ECG showed PR prolongation (>0.22 s) in 6 patients, right bundle branch block (RBBB) in 4 including 2 with left axis deviation (LAD), intraventricular conduction disturbance (IVCD) in 1, and no AVB or IVCD in the remaining 10. Structural heart disease was observed in 6 patients: coronary artery disease in 4 patients (including 2 patients with a previous myocardial infarction), cardiac tumor involving the left ventricle in 1, and mitral valve prolapse without mitral regurgitation in 1. The ECG-documented maximum ventricular asystole during P-AVB varied from 3.6 to 60 s. The index ECG was recorded by ambulatory Holter ECG monitoring for 8 patients, in-hospital ECG monitoring for 6, standard 12-lead ECG for 4, and an implantable loop recorder for 2. An electrophysiologic study (EPS) was performed for 11 patients. Abnormal AV conduction was recognized in 4 patients. A cardiac pacemaker was implanted in 13 patients.

2.2. Definition of vagal score

The VS was developed by assigning 1 point for patients with each of the following: (1) no AVB or IVCD on the baseline ECG, (2) PR prolongation immediately before P-AVB, (3) sinus slowing immediately before P-AVB, (4) initiation of P-AVB by PP prolongation, (5) sinus slowing during ventricular asystole, and (6) resumption of AV conduction with PP shortening, and by assigning −1 point each for (7) the initiation of P-AVB by a premature beat, and (8) resumption of AV conduction by an escape beat. We copied the index-ECG recording of P-AVB in all 20 patients. ECG scoring was performed in each index-ECG recording by 3 blinded investigators. Scoring discrepancies were resolved by the principal investigator (MS).

2.3. Possible mechanism of P-AVB

Based on the clinical situations and EPS results, we considered the mechanism of P-AVB either vagally mediated or intrinsic conduction disease (ICD).

3. Results

3.1. Diagnosis of vagally mediated P-AVB

The mechanism underlying the P-AVB in 5 patients (cases 1, 2, and 4–6) was definitely a vagal mechanism based on the following reasons:

In case 1, a head-up tilt test was conducted as in a healthy volunteer. The patient had syncope due to P-AVB during the head-up tilt test (Fig. 1) [3].

In case 2, the patient had a history of cough syncope. Syncope with P-AVB was induced by carotid sinus massage. We recommended implantation of cardiac pacemaker; however, the patient rejected the recommendation.

In 2 patients (cases 4 and 5), swallowing induced P-AVB, which was inhibited after an intravenous administration of atropine sulfate (Fig. 2). The baseline EPS was normal in case 3; however, swallowing a solid food repeatedly induced paroxysmal A-H block. In case 6, the patient experienced P-AVB during meals as shown in the Holter ECG recording. The resting ECG revealed a first-degree to second-degree Wenckebach AVB. P-AVB was provoked by Valsalva maneuver; however, it was inhibited after an intravenous atropine injection. A-H Wenckebach block was observed during the EPS; however, it was recovered to the first-degree AVB with an A-H interval of 400 ms after intravenous administration of atropine sulfate.

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