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Original Article

Shortening of intraventricular conduction time with rapid ventricular pacing

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ABSTRACT

Background: Supernormal conduction (SNC) of the human ventricular myocardium has been reported, but its mechanism remains controversial.
Methods: We recorded monophasic action potentials during rapid ventricular pacing from the right ventricular endocardium in 24 patients with supraventricular tachyarrhythmias who underwent catheter ablation.
Results: In 7 of 24 patients, shortening of the QRS duration was observed at a pacing cycle length $\leq$400 ms and lengthening of the QRS duration was observed in 3.
Conclusions: Shortening of the QRS duration during rapid ventricular pacing was observed in the patients.

1. Introduction

Programmed stimulation of human atria has shown that there is a period in the cardiac cycle during which premature atrial impulses reach the recording electrodes with less conduction delay (i.e., shorter conduction time) than basic drive impulses [1,2]. Such shortening of the conduction time is related to conduction during a period of "supernormal" excitability and conduction [3–6]. However, few reports have documented shortening of intraventricular conduction time in the human ventricle, and the mechanism is still controversial [7,8]. The purpose of this study was to evaluate intraventricular conduction time in relation to the action potential duration in humans.

2. Materials and methods

2.1. Patients

Twenty-four consecutive patients (15 men and 9 women; mean age, 58.8 ± 12.0 years; range, 34–78 years) with supraventricular tachyarrhythmias (Wolff–Parkinson–White [WPW] syndrome, 4; atrioventricular nodal reentrant tachycardia, 5; typical atrial flutter, 15) were referred to the Nihon University Hospital for catheter ablation from December 2004 to July 2009. The study protocol, comprising catheter ablation followed by an electrophysiological study, was approved by the Clinical Research Committee of Nihon University Hospital on November 1, 2004 (Approval no. 51), and written informed consent was obtained from all the patients.

2.2. Study protocol

Treatment with antiarrhythmic drugs (excluding digitalis, beta-blockers, and calcium channel blockers) was discontinued for at least five half-lives before the electrophysiological procedure. A 7-F Franz combination catheter (EPT Ltd., Sunnyvale, CA, USA) was inserted through the right femoral vein, and monophasic action potential (MAP) was recorded by pressing the Franz catheter against the right ventricular apex. MAP signals were amplified at a filter setting of 0.05–500 Hz. Ventricular pacing was performed from the proximal electrode pair of the Franz catheter at twice diastolic threshold strength and a pulse duration of 2 ms. The MAP duration (MAPD) was measured as the interval along a line horizontal to the diastolic baseline, from the steepest part of the MAP upstroke to the level of 90% repolarization ($MAPD_{90}$) [9]. The right ventricle (RV) was paced at cycle lengths (CLs) of 600, 500, 400, 350, 300, 275, and 250 ms for 120 beats at each CL. RV MAPs were recorded during atrial pacing at a filter setting of 0.05–500 Hz. MAPD at each pacing CL was measured from the onset of the steep upstroke of the MAP and the intersection between the diastolic baseline and a tangent placed on the phase 3 repolarization. Ventricular myocardial conduction time was assessed by measuring the total QRS duration in lead V1

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or lead II, from the onset of the pacing stimulus to the end of the QRS complex, which was defined as the intersection of tangents to the ST segment and the major terminal deflection of the QRS complex at a sweep speed of 100 ms/cm (Fig. 1) [10]. QRS duration shortening and lengthening were defined as $\geq$10 ms shortening and $\geq$10 ms lengthening based on the QRS duration at a pacing

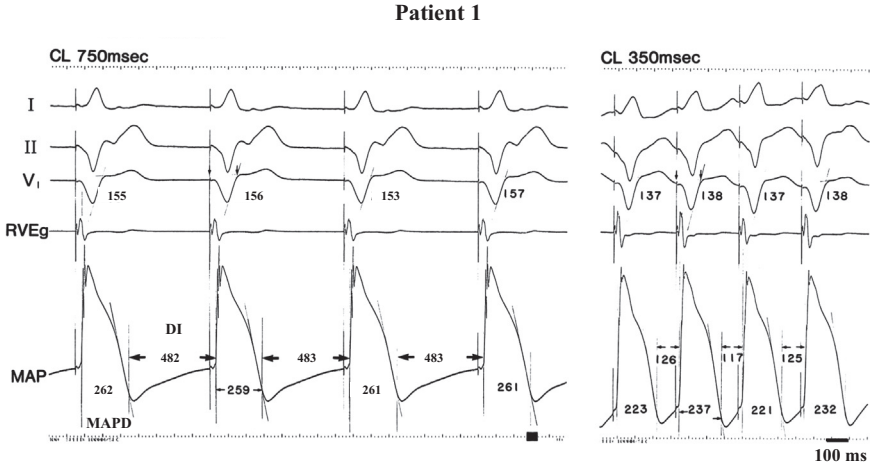

Fig. 1. Effect of the pacing cycle length on the QRS duration. I, II, V1, surface electrocardiogram; RVEg, right ventricular electrogram; MAP, monophasic action potential; MAPD, MAP duration (ms); DI, diastolic interval (ms); CL, pacing cycle length. Note that MAPD alternans (> 10 ms) was observed at a pacing cycle length of 350 ms.

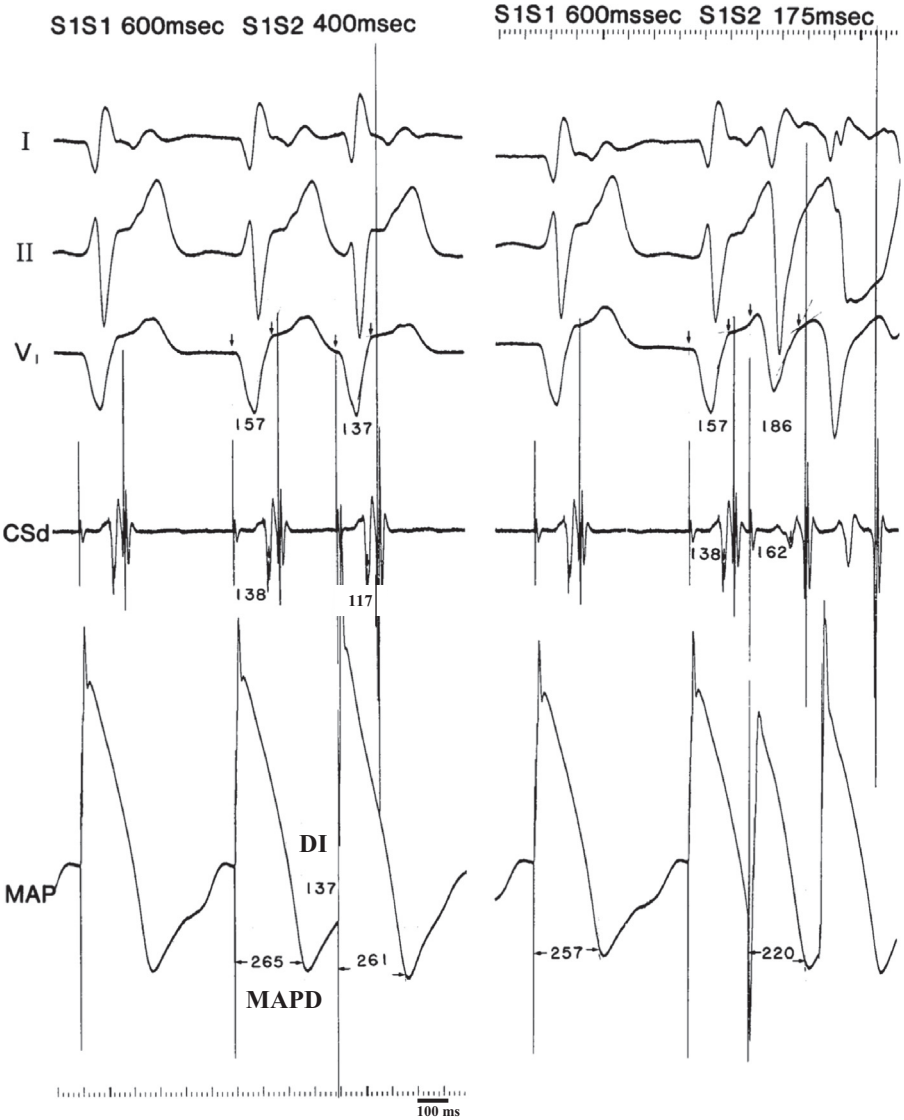

Fig. 2. Effect of programmed ventricular stimulation on the QRS duration and intraventricular conduction time. I, II, V1, surface electrocardiogram; CSd, distal coronary sinus electrogram; MAP, monophasic action potential; MAPD, MAP duration (ms); DI, diastolic interval (ms); CL, pacing cycle length.

Table 1
Clinical, genetic, electrocardiographic, and electrophysiologic characteristics of ERS and ER pattern patients.

	ERS ($n=8$)	ER Pattern ($n=5$)	P value
Age (years)	39.1 ± 17.0	51.8 ± 23.2	0.281
Sex ratio (M/F)	8/0	5/0	0.502
Family history of SCD	2	0	0.223
ECG pattern (1/2/3)	6/0/2	1/1/3	0.008
Late potentials	7/8	3/5	0.254
EPS	8	0	–
Inducible VF/PVT at EPS	6	–	–
ICD implantation	5	0	0.003
Follow-up (months)	51.6 ± 29.6	30.0 ± 20.2	0.185
Arrhythmic event during follow-up	2	0	–

Number of patients is shown unless otherwise indicated.
ER: early repolarization; ERS: early repolarization syndrome; SCD: sudden cardiac death; BS: Brugada syndrome; EPS: electrophysiologic study; VF, ventricular fibrillation; PVT, polymorphic ventricular tachycardia; ICD, implantable cardioverter defibrillator

Table 2
Comparison of spatial and transmural repolarization time and dispersion of repolarization time in ERS and ER pattern patients.

	ERS ($n=8$)	ER Pattern ($n=5$)	P value
Avg. RT-c	201.7 ± 18.8	234.0 ± 25.5	0.0404
Avg. RTe-c	287.1 ± 10.3	325.8 ± 43.8	0.0481
Avg. T(p-e)-c	41.0 ± 9.9	42.6 ± 8.2	0.883
RT-c dispersion	71.9 ± 14.3	84.2 ± 11.9	0.223
Avg. RT-c dispersion	37.2 ± 8.4	39.6 ± 15.7	0.608
RTend-c dispersion	52.7 ± 14.3	52.6 ± 11.3	0.807
Avg. RTe-c dispersion	25.4 ± 9.1	29.0 ± 15.8	0.608
T(p-e)-c dispersion	52.7 ± 14.3	52.6 ± 11.3	0.806
Avg. T(p-e)-c dispersion	26.4 ± 9.3	29.0 ± 15.8	0.144

Average RT-c = average of RT-c from the187-channel ECG, Average RTe-c = average of RTe-c from the187-channel ECG, Average T(p-c)-c = average of T(p-c)-c from the187-channel ECG, RT-c dispersion = RT-c max − RT-c min, RTend-c dispersion = RTend-c max − RTend-c min, T(p-e)-c dispersion = T(p-e)-c max − T(p-e)-c min, Average RT-c dispersion = average of [each RT-c of the 187 channels − RTmin-c], Average RTend-c dispersion = average of [each RTend-c of the 187 channels − RT-min-c], Average T(p-e)-c dispersion = average of [each T(p-e)-c of the 187 channels − T(p-e)min-c].

cycle length of 600 ms. In one patient (16 years old, male, with WPW syndrome), programmed ventricular stimulation was performed during the MAP recording. The QRS duration and conduction time from stimulus spike to the bipolar left ventricular electrogram, recorded from distal coronary sinus electrogram of the premature stimulus, was measured (Fig. 2). Diastolic interval was defined as 90% repolarization of the RV MAP to the next ventricular pacing stimulus artifact.

3. Results

Shortening of the QRS duration in 7 and lengthening of the QRS duration in 3 of the 24 patients were observed during RV pacing at a mean pacing cycle length of 346 ± 37 ms and 258.3 ± 14.4 ms (Fig. 1; Tables 1 and 2). The mean shortening of the QRS duration was −12 ± 3 ms, and the mean lengthening of the QRS duration was 13 ± 3 ms (Tables 1 and 2). Patient 1 demonstrated shortening in QRS duration by 20 ms at a pacing CL of 350 ms and a diastolic interval of 123 ms (Fig. 1). Fig. 2 shows a tracing from a patient in whom conducted programmed stimulation was performed, demonstrating shortening of the QRS duration by 20 ms, and shortening of the stimulus to left ventricular bipolar electrogram conduction time by 21 ms, observed at a diastolic interval of 137 ms. However, the QRS duration and stimulus to left ventricular electrogram conduction time were increased by 29 and 24 ms, respectively, at a diastolic interval of 0 ms (premature stimulation occurred before 90% repolarization of the preceding MAP; Fig. 2). Shortening of the QRS duration was observed at a mean diastolic interval of 110 ± 9 ms, which corresponded to the mean MAP duration [(S1S1 interval − diastolic interval)/S1S1 interval] of 68.5 ± 2.8%. QRS duration did not increase in patients 1, 6, 7, 8, and 9, and increased by 6 ms in patients 2 and 10, but none of the 7 patients showed an increase ≥ 10 ms at shorter pacing cycle lengths.

4. Discussion

We showed that shortening of the QRS duration occurred at a pacing cycle length ≤ 400 ms in 29% of the patients and that shortening of the QRS duration and stimulus to left ventricular conduction time occurred in one patient in whom programmed ventricular stimulation was performed. Supernormal conduction is defined as conduction that is more rapid than expected or as the presence of conduction when block is anticipated. True supernormal conduction may be defined as a shortening of the conduction time in premature beats as compared with the regular conduction of later stimuli in the normal heart. This decrease in conduction time might be caused by the period of supernormal excitability. A previous study of the intact canine heart by Puech et al. [5] showed that supernormal conduction in the RV, measured from pacing spike to the onset of the MAP, occurred at an S1S2/S1S1 ratio between 66.1 ± 0.9% and 77.9 ± 8.2%. Our finding that MAP duration/S1S1 interval was 68.5 ± 2.8% is consistent with that of their study. In contrast, another study reported that intraventricular conduction time and the major axis dimension of the left ventricle were shortened to a similar magnitude and at a similar time in the cardiac cycle. Therefore, shortening of intraventricular conduction time might result, at least in part, from a shortened dimensional pathway length from the site of the stimulating impulse propagation to the recording electrodes [8]. However, Puech et al [5]. showed "supernormal conduction" at the RV epicardium, but we found that only 29% of the patients exhibited shortening of the paced QRS duration during rapid ventricular pacing. Therefore, further studies are needed to clarify the association between true "supernormal conduction" and shortening of the conduction distance in the shortening of the QRS duration in the intact heart.

The limitations of this study were the following: (1) we did not measure intraventricular conduction time from more than 2 intracardiac electrograms; (2) we conducted premature stimulation study in only one patient; and (3) some MAP recordings included the T wave of the electrocardiogram due to insufficient contact between the recording electrode and RV endocardium, which made measuring the diastolic interval difficult; and (4) the relationship between QRS duration, MAP duration, and diastolic interval at each pacing cycle was not measured systematically; therefore, the data was not shown graphically.

5. Conclusions

Shortening of the QRS duration during rapid ventricular pacing can occur in humans.

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Conflict of interest

None.

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