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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

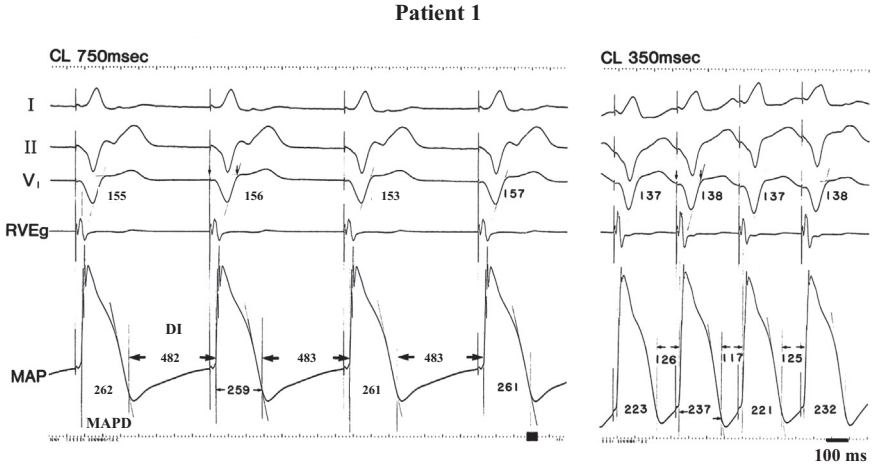

Fig. 1. Effect of the pacing cycle length on the QRS duration. I, II, V1, surface electrocardiogram; RVEg, right ventricular electrogram; MAP, monophasic action potential; MAPD, MAP duration (ms); DI, diastolic interval (ms); CL, pacing cycle length. Note that MAPD alternans (> 10 ms) was observed at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

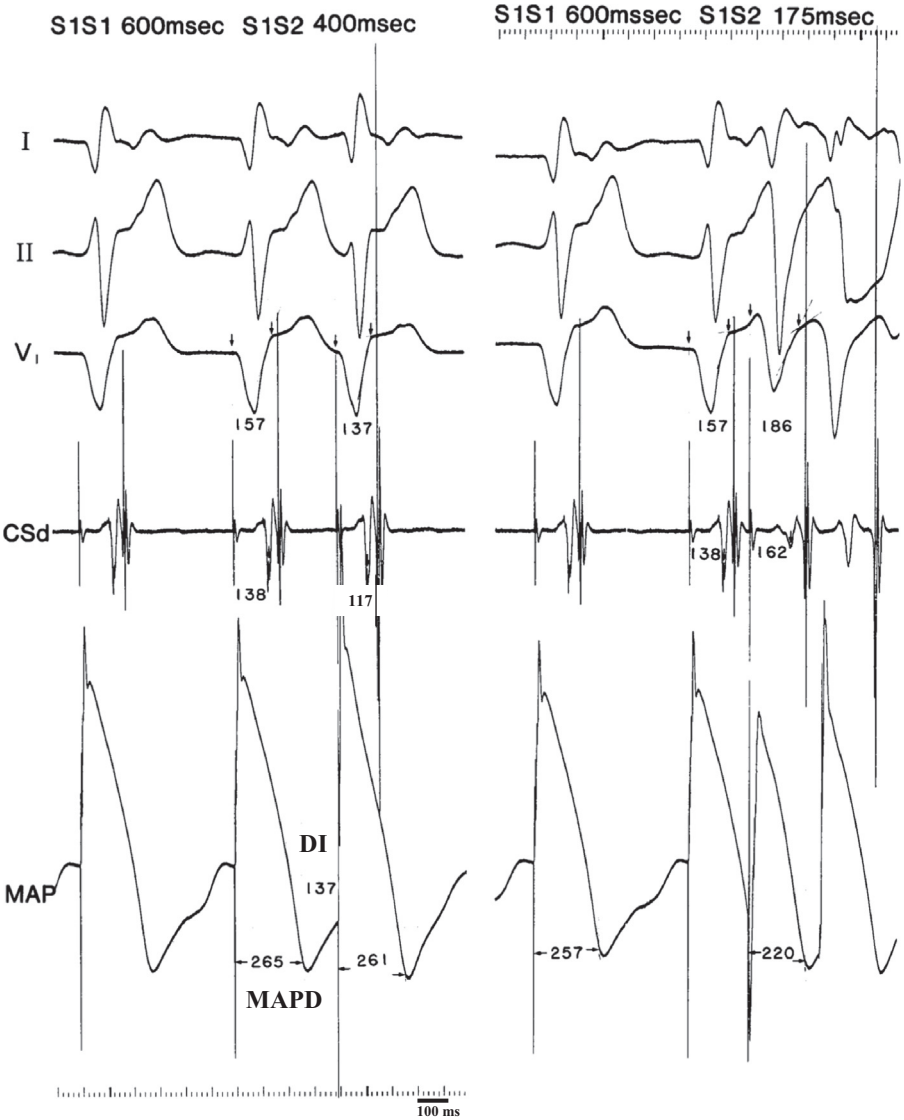

Fig. 2. Effect of programmed ventricular stimulation on the QRS duration and intraventricular conduction time. I, II, V1, surface electrocardiogram; CSd, distal coronary sinus electrogram; MAP, monophasic action potential; MAPD, MAP duration (ms); DI, diastolic interval (ms); CL, pacing cycle length.