Ventricular arrhythmias originating from papillary muscles in the right ventricle

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BACKGROUND Premature ventricular complexes (PVCs) and ventricular tachycardia (VT) with origin in the left ventricular papillary muscle have recently been described. There are no prior studies describing the characteristics of the ventricular arrhythmias (VAs) arising from the right ventricular papillary muscles (RV PAPs).

METHODS Among 169 consecutive patients who underwent a catheter ablation of a VA, eight patients with RV PAPs were identified (seven men, mean PVC burden 17.0% ± 20%). A control group consisted of 10 consecutive patients with arrhythmias originating from the right ventricle (10 women, mean PVC burden 13.9% ± 12.8%). All patients underwent cardiac magnetic resonance imaging (MRI). Intracardiac echocardiography was used to identify the site of origin of the RV PAP arrhythmias. The site of origin of a total of 15 distinct PAP arrhythmias was mapped to the following papillary muscles: posterior (n = 3), anterior (n = 4), or septal (n = 8).

RESULTS Postablation echocardiograms did not reveal new tricuspid regurgitation. During a mean follow-up of 8 ± 9 months, there were no adverse outcomes. The PVC burden was reduced from 17% ± 20% preablation to 0.6% ± 0.8% postablation in the RV PAP group and from 13.9% ± 12.8% to 0.3% ± 0.4% in the control group. The QRS complex was broader in the RV PAP group compared with in the control group (163 ± 21 ms vs. 141 ± 22 ms; $P = .02$). RV PAP arrhythmias originating from the posterior or anterior RV PAPs more often had a superior axis with late R-wave transition (>V4) compared with septal RV RAP arrhythmias, which more often had an inferior axis with an earlier R-wave transition in the precordial leads (≤V4; $P < .05$).

CONCLUSION PVCs and VT may originate in the RV PAPs. Radiofrequency ablation is effective in eliminating these arrhythmias.

KEYWORDS Ventricular arrhythmia; Papillary muscle; Radiofrequency ablation; Right ventricle; Ventricular tachycardia; Premature ventricular complexes; Mapping; Ablation

ABBREVIATIONS ECG = electrocardiogram; **LV** = left ventricular; **MRI** = magnetic resonance imaging; **PVC** = premature ventricular complex; **RVOT** = right ventricular outflow tract; **RV PAP** = right ventricular papillary muscle; **SOO** = site of origin; **VA** = ventricular arrhythmia; **VT** = ventricular tachycardia

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Introduction

Papillary muscles play a role in arrhythmogenesis in animal models.[1,2] In humans, arrhythmias originating from the left papillary muscles have been described.[3–5] The purpose of this study is to describe a series of eight patients in whom ventricular arrhythmias (VAs) were mapped to one of the right-sided papillary muscles.

Methods

Patient characteristics (Table 1)

Patients in this study consisted of eight consecutive patients (one woman, age 44 ± 14 years old, ejection fraction 0.45 ± 0.16) with frequent premature ventricular complexes (PVCs; n = 4) or both PVCs and ventricular tachycardia (VT; n = 4) who had been referred for catheter ablation and whose arrhythmia was mapped to one of the right ventricular papillary muscles (RV PAPs). The subjects in this study were drawn from a series of 169 consecutive patients. Among the eight study patients, six had a history of palpitations, one came to medical attention because of congestive heart failure, and one had syncope. An impaired left ventricular (LV) function was present in four patients, all of whom had a high PVC burden. One patient had congenital absence of the left pericardium.

The control group consisted of 10 consecutive patients who were referred for ablation of symptomatic idiopathic arrhythmias originating in the right ventricle; seven of 10 patients had VT. All arrhythmias in the control group originated from the right ventricular outflow tract (RVOT). Nine patients reported palpitations, and three had syncope. Their mean LV ejection fraction was 0.57 ± 0.09 ($P = .11$ compared with the study group).

Before the ablation procedure, all patients underwent echocardiograms and cardiac magnetic resonance imaging (MRI) to assess left and right ventricular function, rule out

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Table 1 Comparison of patients with arrhythmias originating from the RV PAP and RVOT

	Right PAP	Control group	*P*
Patients	8	10	
Age	43.6 ± 14	43.3 ± 9	1
Gender, male/female	7/1	0/10	<.001
Cardiomyopathy	4/8	1/10	.1
LVEF, %	45 ± 16	57 ± 9	.08
Right ventricular EF, %	53 ± 8	57 ± 10	.3
PVC and VT (n)/PVC only (n)	4/4	6/4	1
Patients with pleomorphic ectopy	3/8	3/10	.7
QRS width in sinus rhythm	93 ± 10	90 ± 10	.5
Baseline HV interval	45 ± 6	41 ± 4	.08
PVC burden on Holter monitor before ablation, number/hour	676 ± 638	465 ± 296	.4
ECG during arrhythmia;			
QRS width during arrhythmia	163 ± 21	141 ± 22	.02
No. of distinct PVC morphologies	15	13	
Notches in precordial leads	11/15	5/13	.047
Left bundle superior axis	5	0	.04
Left bundle inferior axis	10	13	.04
RF delivered, minutes	16 ± 9	11 ± 6	.2
Procedure time, minutes	277 ± 49	203 ± 67	.03
Activation time at effective site, ms	23 ± 8	28 ± 4	.1
Purkinje potential identified at the effective ablation site	2/8	0/10	.5
Amplitude of ventricular EGM in SR, mV	2.1 ± 1.4	2.9 ± 2.4	.4

Note: EGM = electrogram; right PAP = right papillary muscle; EF = ejection fraction; Q = Q wave; RF = radiofrequency energy; SR = sinus rhythm.

arrhythmogenic right ventricular dysplasia, and assess for the presence of scar.

Electrophysiologic study and mapping

After informed consent was obtained, several multipolar electrode catheters were introduced into the right ventricle, right atrium, and His position. Programmed right ventricular stimulation was performed using up to four extrastimuli to assess for inducible VT. Programmed stimulation was repeated during infusion of isoproterenol at 2–20 μg/min. Electrograms were filtered at 50–500 Hz. The intracardiac electrograms and leads V1, I, II, and III were displayed on an oscilloscope and displayed at a speed of 100 mm/s. The recordings were stored on optical disc (EP Med, West Berlin, NJ). An electroanatomical mapping system (CARTO, Biosense Webster, Diamond Bar, CA) was used to guide mapping. Activation mapping was performed during ventricular ectopy or VT. In the setting of infrequent ventricular ectopy, pace mapping was used to identify the site of origin. Pace maps were classified as matching (11 or 12/12 leads) or not matching.

Radiofrequency ablation

Radiofrequency energy was delivered via a 4-mm-tip catheter (Navistar, Biosense Webster, Inc.) or a 3.5-mm-tip irrigated-tip catheter (Thermocool, Biosense Webster) at sites with the earliest endocardial activation during the VA and/or at matching pace-mapping sites. Radiofrequency energy was delivered in a temperature-controlled mode with a target temperature of 55°C at a power of ≤50 W. With the irrigated-tip catheter, radiofrequency energy was applied at a power of 30–35 W and a maximal temperature of 45°C. Programmed ventricular stimulation with and without isoproterenol infusion was repeated at the end of the procedure.

Real-time imaging

Intracardiac echocardiography (Cypres, Acuson Inc., Mountain View CA) was used during the procedures to identify the papillary muscles (Figure 1) and to confirm catheter contact with the papillary muscle. Using intracardiac ultrasound the distinction of the site of origin (SOO) was made between the septal PAP (insertion on the septum) and the anterior or posterior PAP (inserting in the RV free wall or apex). A transthoracic echocardiogram was performed after the ablation to assess for tricuspid regurgitation.

Twelve-lead electrocardiogram

Twelve-lead electrocardiograms (ECGs) of the PVCs and VTs were analyzed for bundle branch block morphology, axis, QRS width, presence of notching in V1 to V6 (Figure 2), R-wave pattern in V1 (rS, QS), and the transition point from predominantly negative S-wave to predominantly positive R-wave deflection in the precordium. An early transition was defined as transition in lead V4 or earlier (Figure 2, left panel), and late transition was defined as a transition in V5 or V6 (Figure 2, right panel). Notches were defined as deflections in the QRS complex aside from a triphasic pattern (Figure 2).

Cardiac MRI

MRI was performed with a 1.5 Tesla MRI scanner (Signa Excite CV/i, General Electric, Milwaukee, WI) with a 4- or 8-element phased array coil placed over the chest in the supine position in all patients. Images were acquired with ECG gating during breath holds. Dynamic short- and long-axis images of the heart were acquired using a segmented, k-space, steady-state, free-precession pulse sequence (repetition time 4.2 ms, echo time 1.8 ms, 1.4 × 1.4 mm in-plane