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Post-Extrasystolic Potentiation as a Predictor of Recovery of Left Ventricular Dysfunction After Radiofrequency Catheter Ablation

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ABSTRACT

OBJECTIVES This study hypothesizes that post-extrasystolic potentiation reflects left ventricle contractile reserve and therefore may predict an improvement of premature ventricular contraction (PVC)-induced cardiomyopathy after PVC ablation.

BACKGROUND Post-extrasystolic potentiation is a physiologic phenomenon of blood pressure accentuation after a PVC beat.

METHODS We performed a retrospective study of patients with a PVC burden of ≥10% PVC/24 h and left ventricular ejection fraction (LVEF) of <50% who underwent successful ablation between January 1, 2009, to June 30, 2015. Subjects were classified as having reversible (a final LVEF ≥50%) or irreversible (final LVEF <50%) LV dysfunction on a follow-up echocardiogram. A reference (control) group with ≥10% PVC but normal LV function was also identified.

RESULTS Sixty-one patients (age 68 ± 11 years, 98% male) were studied: 30 with preserved and 31 with reduced LVEF. During median follow-up of 9.4 months, the LVEF of 17 of 31 reduced EF patients improved (reversible) but 14 did not (irreversible). The post-PVC beat systolic blood pressure (SBP) (mm Hg) increase ranged from 12.1 in control subjects (LVEF >50%) to 11.5 in reversible patients to 5 in irreversible patients. In multivariate analysis, the independent predictors of reversible LV function were post-PVC SBP rise (odds ratio [OR]: 4.61; 95% confidence interval [CI]: 1.45 to 15.83 per 5-mm Hg increase; $p < 0.001$), post-PVC pulse pressure change (OR: 5.2; 95% CI: 2.3 to 18.6 per 5-mm Hg increase; $p < 0.001$), and PVC QRS duration (OR: 2.78; 95% CI: 1.63 to 10.94 per 10-ms increase; $p < 0.001$).

CONCLUSIONS In patients with LV dysfunction and frequent PVC, post-PVC SBP accentuation may be a marker for subsequent recovery of LVEF after ablation in presumed PVC-induced cardiomyopathy. (J Am Coll Cardiol EP 2017;3:1283–91) Published by Elsevier on behalf of the American College of Cardiology Foundation.

Idiopathic premature ventricular complexes (PVC) are generally considered benign and most often are treated conservatively. However, sustained ventricular tachycardia, symptomatic PVC resistant to medical therapy, and PVC thought to contribute to an underlying cardiomyopathy are often treated with radiofrequency ablation.

A high burden of PVC has been associated with left ventricular (LV) dysfunction in prior studies of patients referred for ablation (1–5). A longitudinal study (6) found subclinical deterioration in LV function over 5 years in those with a high PVC burden (≥10% to 20%). Yet many patients with long-standing, frequent PVC have no evidence of LV dysfunction. Furthermore, a

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ABBREVIATIONS AND ACRONYMS

CI = confidence interval

DBP = diastolic blood pressure

IQR = interquartile range

LV = left ventricle

LVEF = left ventricular ejection fraction

MAP = mean arterial pressure

OR = odds ratio

PESP = post-extrasystolic potentiation

PVC = premature ventricular complex(es)

SBP = systolic blood pressure

VT = ventricular tachycardia

significant proportion of patients with frequent PVC and LV dysfunction show no significant improvement in ventricular function despite the elimination of PVC. A few studies have suggested that PVC coupling interval (4), QRS duration of PVC (6), or origin of PVC from epicardial (7) or right ventricular outflow tract (3,5) might predict recovery of LV function post-ablation.

In a normal heart, the post-PVC initiated systolic blood pressure (SBP) is typically higher than the preceding sinus rhythm-initiated SBP. This phenomenon has been termed post-extrasystolic potentiation (PESP). Although the mechanisms are not entirely clear, and a longer diastolic filling period might contribute to the "potentiation," most of the evidence suggests that calcium movement within the sarcoplasmic reticulum is responsible for the increase in myocardial contractility associated with PESP. We hypothesized that the relative change in SBP and mean arterial pressure (MAP) may indicate the LV contractile reserve, and this response in patients before PVC ablation might identify patients in whom PVC-associated LV dysfunction is reversible or irreversible following ablation.

Hence, the purpose of this study was to examine whether post-PVC blood pressure rise is a predictor of the reversibility of LV dysfunction after the successful elimination of PVC by ablation.

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METHODS

PATIENT POPULATION. A total of 150 consecutive patients underwent successful first-time ablation of frequent PVC at the Minneapolis Veterans Affairs Medical Center from January 1, 2009, to June 30, 2015. After satisfying all the exclusion and inclusion criteria, 61 patients were included in this study (**Figure 1**).

We noted those who had a documented left ventricular ejection fraction (LVEF) of <50% on pre-procedure echocardiography. As existing data suggest that very frequent PVC are associated with LV dysfunction (8), we restricted our cohort to patients with ≥10% PVC on pre-procedure 24-h Holter monitoring. Successful ablation was defined as 80% reduction in the 24-h burden of PVC, verified by 24-h Holter monitoring, 3 months post-procedure (9). Patients were excluded if they had a known cause of LV dysfunction or a history of sustained ventricular tachycardia, appropriate implantable defibrillator discharges, or sudden cardiac death. Only subjects with complete Holter and echocardiographic follow-up were eligible. Subjects with unsuccessful ablation were excluded from the analysis. All antiarrhythmic agents were discontinued after ablation. All patients with LV dysfunction before ablation received standard therapy with beta blockade and renin-angiotensin inhibition prior to ablation. Those patients with ongoing LV dysfunction after ablation continued to receive standard therapy for heart failure.

To evaluate characteristics associated with LV dysfunction in the presence of frequent PVC, we also identified a reference group of patients with ≥10% PVC on pre-procedure Holter monitoring but with preserved LV function (echocardiographic LVEF of ≥50%) who underwent ablation during the study period. These patients had the procedure performed for significant worsening of quality-of-life related to symptoms of shortness of breath or palpitations.

DATA COLLECTION. Baseline demographic, historical, and clinical characteristics were collected, retrospectively, through detailed chart review. All electrocardiographic and blood pressure measurements were performed, using digital calipers at 100 mm/s on CardioLab (version 6.5.4.1858, GE Medical Systems, Waukesha, Wisconsin). To limit heterogeneous within-subject variances, all electrocardiographic and blood pressure measurements were repeated on 3 separate PVC, occurring at least 10 min apart during intracardiac catheterization and arterial blood pressure monitoring (**Figure 2**). The measurements were taken prior to anesthesia induction and administration of inotropes. The mean of the 3 measurements was used for analysis. In the case of multiple PVC morphologies, the dominant/targeted PVC was measured.

The following equations were used:

$$\text{PESP SBP change} = \text{post-PVC SBP} - \text{sinus SBP}$$

$$\text{PESP DBP change} = \text{post-PVC DBP} - \text{sinus DBP}$$

$$\text{MAP} = \frac{\text{SBP} - 2(\text{DBP})}{3}$$

$$\text{MAP change} = \text{MAP}_{\text{post-PVC beat}} - \text{MAP}_{\text{sinus}}$$

$$\text{Pulse pressure change} = \left(\text{SBP}_{\text{post-PVC}} - \text{DBP}_{\text{post-PVC}}\right) - \left(\text{SBP}_{\text{sinus}} - \text{DBP}_{\text{sinus}}\right)$$

As quantitative echocardiographic methods of assessment of LV function may be inaccurate in the setting of frequent PVC, visual estimation was used to determine LVEF. All echocardiographic assessments were blinded to electrophysiologic and procedural characteristics. If the interpreter reported a range, the lower value was used for all analyses.