Association of premature ventricular complexes with central aortic pressure indices and pulse wave velocity

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Background Although premature ventricular complex (PVC) occurs frequently, its predisposing factors have rarely been studied. We examined the connection between PVC and aortic stiffness.

Methods We recruited 200 consecutive patients (<50 years, 95 men, mean age 36 ± 10 years) who received a 24-hour ambulatory electrocardiography examination for palpitation and PVC loads. Muscular artery pulse wave velocity (PWVm) and 4 main aortic pressure indices—augmented pressure, augmentation index (AIx), AIx corrected for a steady heart rate of 75 beat/min, and the extra workload—were measured, and atherosclerosis risk was evaluated.

Results Eighty-three (42%) patients had no PVC loads; 58 (29%) patients had low loads (<24 beat/d), and 59 (29%) had high loads (≥24 beat/d). Only age and hyperlipidemia were significantly associated with PVC loads. Using a multivariate logistic regression model adjusted for potential confounders, we found that AIx (odds ratio [OR] 1.88, 95% CI 1.20-2.91, P = .005); augmented pressure (OR 1.57, 95% CI 1.02-2.43, P = .042); AIx corrected for a steady heart rate of 75 beat/min (OR 1.82, 95% CI 1.18-2.82, P = .007); and PWVm (OR 1.53, 95% CI 1.07-2.19, P = .021) were independent factors for PVC loads.

Conclusion Increased central aortic pressure indices as well as PWVm were associated with increased PVC loads in young patients undergoing 24-hour ambulatory electrocardiography. Central aortic properties probably contributed to the occurrence of PVC. (Am Heart J 2008;155:500.e1-500.e6.)

Premature ventricular complex (PVC), common both in healthy and in diseased patients, is associated with an increased risk of ventricular arrhythmias or sudden death in patients with myocardial infarction or congestive heart failure. The possible mechanisms for PVC are myocardial ischemia, myocardial infarction, hypertensive cardiomyopathy, left ventricular systolic or diastolic dysfunction, and left ventricular remodeling. However, the predisposing factors for PVC in apparently healthy individuals have not been fully elucidated.

Many risk models for cardiovascular disease, such as the Framingham coronary risk model, have been used in an attempt to identify those who would benefit most from preventive treatment. Such models are based primarily on age, sex, blood pressure, smoking status, cholesterol, and diabetes mellitus. Still, a large proportion of individuals are not identified before they have developed cardiovascular disease. Noninvasive tests to detect individuals with atherosclerosis or arterial stiffness, preferably before they develop cardiovascular disease, may improve the selection of candidates for preventive treatments.

High PVC loads may be associated with Cornell voltage estimates of left ventricular mass and the left ventricular mass index derived from echocardiography. This effect is independent of many of the known causal predictors of PVC. We also know that left ventricular hypertrophy (LVH) detected using conventional echocardiography is associated with atherosclerotic plaque in the aorta in patients with previous stroke or transient ischemic attack. It was recently shown that aortic augmented pressure (AG) is associated with left ventricular mass. In hemodialysis patients, atherosclerosis is associated with LVH, and the presence of aortic plaques is an independent factor related to the left ventricular mass index. Pulse wave analysis, a noninvasive test and expressed as a central augmentation index (AIx), suggests that atherosclerosis in the aorta and peripheral arteries may be detected as high aortic stiffness, even before patients develop cardiovascular disease.

We hypothesized that high PVC loads might be associated with aortic stiffness in young individuals. To study the connection between aortic stiffness and PVC load, we recruited 200 consecutive patients (<50 years, 95 men, mean age 36 ± 10 years) who received a 24-hour ambulatory electrocardiography examination for palpitation and PVC loads. Muscular artery pulse wave velocity (PWVm) and 4 main aortic pressure indices—augmented pressure, augmentation index (AIx), AIx corrected for a steady heart rate of 75 beat/min, and the extra workload—were measured, and atherosclerosis risk was evaluated.

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loads, from February 2005 through December 2005, we evaluated 200 consecutive individuals who underwent 24-hour ambulatory electrocardiographic (ECG) recordings because of palpitation and measured their central aortic pressure indices and muscular artery pulse wave velocity (PWVm) using applanation tonometry.

**Methods**

**Study population**

We prospectively recruited 200 consecutive young patients (95 men, all <50 years [mean age 36 ± 10 years]) who received an ambulatory 24-hour ECG examination to evaluate palpitation. All the patients were apparently healthy and without peripheral vascular occlusion diseases, finger deformities, coronary artery disease, myocardial infarction, left ventricular dysfunction, congestive heart failure, critical valvular heart diseases, or chronic atrial fibrillation. The traditional risk factors for coronary artery disease—diabetes mellitus, hypertension, hypercholesterolemia, and current smoking—were all carefully evaluated in each patient. Diabetes mellitus was diagnosed if the fasting plasma glucose concentration was >125 mg/dL on 2 separate occasions or if the patient was being treated with insulin or oral hypoglycemic agents. Hypertension was diagnosed if blood pressure was >140/90 mm Hg on 3 occasions or if the patient was taking any antihypertension medication. Hypercholesterolemia was defined as a total serum cholesterol concentration ≥200 mg/dL or as using lipid-lowering therapy. Smokers were defined as those who habitually smoked cigarettes (≥20 cigarettes per day) at the start of this study. A family history of premature coronary artery disease (CAD) was defined as patients whose parents, siblings, or grandparents had premature CAD before the age of 55 years in men and 65 years in women. Antihypertension drugs were also carefully reviewed in hypertensive patients. All patients were asked to refrain from taking their medications for 1 day before each test and consuming coffee, smoking, and eating at least 2 hours before data collection. All patients gave written informed consent for this study, and the study protocol was approved by the human research committee of our hospital.

**Pulse wave analysis using applanation tonometry**

Before any testing, all measurements (including blood pressure and heart rate) were made with the patient supine for 20 minutes in a quiet, temperature-controlled laboratory at 26°C ± 1°C. The right radial and carotid pulse waves were detected directly using an ambulatory electrocardiograph. Twelve-lead electrocardiography

Twenty-four-hour Holter recordings were taken using a standard 3-channel flash card recorder (RZ153; Rozinn Electronics, Glendale, NY). The ECG signal was digitized and stored using a commercially available personal computer–based system. All recordings were visually scanned and analyzed using the RZ153. Low and high PVC loads were defined as < or ≥24 beats of premature atrial contraction per day, respectively, detected using an ambulatory electrocardiograph.

Twelve-lead electrocardiography

Patients underwent a standard 12-lead ECG recorded at 25 mm/s and 1 mV/cm standardization with equipment (Page Writer 200; Hewlett-Packard Company, Palo Alto, CA), whose frequency response characteristics met the recommendations of the American Heart Association. The Sokolow-Lyon voltage criteria were used to assess LVH in electrocardiography. The voltage amplitude...
sum (S-wave in V1 + max [R-wave in V5 or R-wave in V6]) was calculated, and ECG LVH was defined as a sum ≥ 3.5 mV.

**Statistical analysis**

Differences between patients with and without PVC loads were compared using Student t test for continuous variables and the χ² test for categorical variables. The differences between different PVC load groups were tested using 1-way analysis of variance and post hoc tests. Multiple logistic regression analyses were used to assess the independent factor for the occurrence of PVC loads. Statistical significance was set at P < .05. All analyses were done using SPSS software, version 11.5 for Windows (SPSS Inc, Chicago, IL).

**Results**

**Correlation between PVC loads and aortic stiffness indices**

Of the 200 patients, 117 (59%, 53 men, mean age 37 ± 10 years) had PVC loads. Age (37 ± 10 vs 34 ± 11 years, P = .010); PWVm (8.6 ± 1.1 vs 8.2 ± 1.0 m/s, P = .002); AG (6.0 ± 5.6 vs 3.4 ± 6.0 mm Hg, P = .002); AIx (18.0% ± 14.7% vs 7.7% ± 14.7%, P < .001); AIx75 (15.3% ± 15.0% vs 7.7% ± 14.7%, P < .001); and Ew (3321 ± 3130 vs 1947 ± 3416 dyne-s/cm², P = .004) were significantly higher in patients with PVC loads (Table I). More patients with PVC loads had a history of hyperlipidemia (26.5% vs 9.6%, P = .003). There were no differences in sex, other risk factors, ECG findings, or drug history between patients with or without PVC loads (Table I).

We further divided the 117 patients with PVC loads into low- and high-load groups. We took the median number (24 beat/d) as a cutoff point. The low-load group (≤24 beat/d) contained 58 (29%) patients and the high-load group (≥24 beat/d) 59 (29%) patients. Muscular artery pulse wave velocity (8.2 ± 1.0 vs 8.6 ± 1.1 vs 8.7 ± 1.1 m/s) (Figure 2); AG (3.4 ± 6.0 vs 4.9 ± 5.1 vs 7.0 ± 5.9 mm Hg); AIx (10.1% ± 15.2% vs 15.8% ± 15.5% vs 20.0% ± 13.5%) (Figure 3); and Ew (1947 ± 3416 vs 2772 ± 2849 vs 3860 ± 3320 dyne-s/cm²) were significantly higher with PVC loads (Table II).

We did multivariate logistic regression analyses, adjusted by age, sex, heart rate, mean blood pressure, and a history of diabetes mellitus and hyperlipidemia, to access an independent predictor of PVC loads. Odds ratios (ORs) are expressed for a change in each factor by 1 SD. The AIx (OR 1.88, 95% CI 1.20-2.91, P = .005); AIx75 (OR 1.82, 95% CI 1.18-2.82, P = .007); AG (OR 1.57, 95% CI 1.02-2.43, P = .042); and PWVm (OR 1.53, 95% CI 1.07-2.19, P = .021) were all independent predictors of PVC loads. However, Ew was only marginally statistically significantly (P = .075) associated with PVC loads (OR 1.13, 95% CI 0.98-1.28).

<p>| Table I. Clinical characteristics of patients |</p>
<table>
<thead>
<tr>
<th>PVC loads: factors</th>
<th>No (n = 83)</th>
<th>Yes (n = 117)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (%)</td>
<td>42 (50.6)</td>
<td>53 (45.3)</td>
<td>.459</td>
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<tr>
<td>Age (y)</td>
<td>34.0 ± 11</td>
<td>37.0 ± 10</td>
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<tr>
<td>Body weight (kg)</td>
<td>61.9 ± 12.4</td>
<td>63.6 ± 13.1</td>
<td>.344</td>
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<tr>
<td>Body height (cm)</td>
<td>163.0 ± 9</td>
<td>165.0 ± 9</td>
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<tr>
<td>Body mass index (kg/m²)</td>
<td>23.1 ± 3.8</td>
<td>23.3 ± 3.6</td>
<td>.722</td>
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<tr>
<td>Systolic blood pressure (mm Hg)</td>
<td>117.0 ± 18</td>
<td>117.0 ± 17</td>
<td>.893</td>
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<tr>
<td>Diastolic blood pressure (mm Hg)</td>
<td>68.0 ± 11</td>
<td>71.0 ± 11</td>
<td>.067</td>
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<td>Mean blood pressure (mm Hg)</td>
<td>85.0 ± 11</td>
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<td>Pulse pressure (mm Hg)</td>
<td>49.0 ± 16</td>
<td>46.0 ± 15</td>
<td>.243</td>
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<td>Heart rate (beat/min)</td>
<td>70.0 ± 12</td>
<td>70.0 ± 11</td>
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<td>Background</td>
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<tr>
<td>Diabetes mellitus</td>
<td>0 (0.0)</td>
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<tr>
<td>Hypertension</td>
<td>7 (8.4)</td>
<td>20 (17.1)</td>
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<td>Hyperlipidemia</td>
<td>8 (9.6)</td>
<td>31 (26.5)</td>
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<td>Current smoker</td>
<td>27 (32.5)</td>
<td>31 (26.5)</td>
<td>.354</td>
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<td>Chronic renal failure</td>
<td>1 (1.2)</td>
<td>1 (0.9)</td>
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<td>Hyperthyroidism</td>
<td>2 (2.4)</td>
<td>7 (6.0)</td>
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<td>Family history of CAD</td>
<td>3 (3.6)</td>
<td>6 (5.1)</td>
<td>.827</td>
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<tr>
<td>Angiotensin-converting enzyme inhibitors</td>
<td>2 (2.4)</td>
<td>4 (3.4)</td>
<td>.882</td>
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<tr>
<td>Angiotensin II receptor blockers</td>
<td>3 (3.6)</td>
<td>7 (6.0)</td>
<td>.746</td>
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<td>Statins</td>
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<td>12 (13.3)</td>
<td>.642</td>
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<td>β-Adrenergic blockers</td>
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<td>.671</td>
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<td>Calcium-channel blockers</td>
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<td>4 (3.4)</td>
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<td>Left ventricle hypertrophy in 12-lead ECG</td>
<td>0 (0.0)</td>
<td>8 (6.8)</td>
<td>.054</td>
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</table>

Data are means ± SD or number (percentage).
Reproducibility of pulse wave velocity and the AIx

The intraclass correlation coefficient between the 2 separate measurements of PWVm and the AIx were high ($r = 0.959$, $P < .01$, and $r = 0.978$, $P < .01$). There were no significant differences between the 2 measurements (6.37 ± 1.05 vs 6.29 ± 1.00 m/s, $P = .276$; mean difference 0.09 ± 0.35 m/s and 14.20% ± 11.08%, $P = .396$; mean difference 0.12% ± 0.08%). The coefficient of variation was 5.8%, calculated using a previously reported method.19

Discussion

In the present study, we showed that central aortic pressure indices and PWVm were independently associated with the occurrence of PVC loads in young persons undergoing 24-hour ambulatory ECG recordings for palpitation. To the best of our knowledge, this is the first report showing that central aortic properties in addition to the left ventricle contributed to PVC loads. Although the exact mechanisms for the linkage between PVC loads and aortic stiffness are unknown, one of the possible mechanisms is the association of PVC loads with atherosclerosis.

Many studies have shown that aortic stiffness is a risk factor for cardiovascular events.20,21 Physiologically, the stiffness of the large arteries depends on 3 main factors: structure elements within the arterial wall, such as elastin and collagen; distending pressure; and vascular smooth muscle tone. Noninvasive tests to detect individuals with atherosclerosis or aortic stiffness, preferably before they develop cardiovascular disease, may improve our ability to select patients for preventive treatments. Arterial pulse wave velocity provides a more direct surrogate of arterial stiffness,22,23 and the AIx provides a composite measure of aortic elasticity plus muscle artery stiffness and wave reflection.24 The AIx, measured using pulse wave analysis, is used as a surrogate measure of aortic stiffness. The AIx should be interpreted as a global index of wave reflections and left ventricular additional load but not as a direct index of aortic stiffness. Age, sex, and blood pressure are known determinants of the AIx.25,26 The central AIx is related to arterial properties through changes in pulse wave velocity. Increased aortic stiffness increases pulse wave velocity and causes the early return of the reflected wave from peripheral reflecting sites to the heart during systole, when the ventricle is still ejecting blood.27 Central AIx is related to arterial stiffness of both elastic and muscular arteries.27 This mechanism augments ascending aortic systolic and pulse pressures, an effect that increases aortic wall stress and potentiates the development of atherosclerosis.27 Because both pulse wave velocity and the AIx are influenced by age, sex, heart rate, blood pressure, a history of hypertension, diabetes mellitus, and hypercholesterolemia,25 we had to adjust these factors when doing a multivariate logistic regression analysis to find the independent predictors of PVC loads in the present study (Table III).

In recent years, carotid-radial PWVm has been increasingly measured as an alternative to aortic PWV because it is easier to gain access to the vessels concerned, and the measures are simpler to perform than the standard.
assessment of carotid-femoral PWV. Noninvasive measures of carotid-radial PWVm correlate with the extent of coronary artery plaque volume and may be a useful noninvasive surrogate marker for the extent of coronary atherosclerosis. Because aortic stiffness is greater in patients with CAD, and because it increases with CAD severity, there is a possibility that increased aortic stiffness in turn increases the elevation of left ventricular wall stress and then PVC loads. Pulse wave velocity and the AIx are also related to the extent of coronary obstruction in patients with chronic kidney disease. One investigation of the relation between pulse wave velocity and the quantity of coronary artery calcium in a community-based sample of adults without a prior history of heart attack or stroke found (1) that pulse wave velocity was related to subclinical coronary atherosclerosis independently of conventional risk factors and (2) that it might be a biomarker of cardiovascular risk in asymptomatic individuals. Therefore, our population with high central aortic stiffness indices and pulse wave velocity might have had high PVC loads because of subclinical CAD.

Pulse wave analysis for measuring arterial stiffness was valuable for predicting cardiac hypertrophy in untreated mild-to-moderate essential hypertension. Augmented pressure and the AIx, assessed using radial artery applanation tonometry and pulse wave analysis, were predictors of left ventricular mass index independently of age and blood pressure measurements during 24-hour monitoring. It should be noted that both wave reflection amplitude and the AIx can increase without an increase in central arterial systolic pressure. These changes in pressure components increase left ventricular afterload and myocardial oxygen demand, a combination that causes an undesirable mismatch between the ventricle and arterial system (that is, ventricular/vascular coupling). Indices of pulse wave analysis are better predictors of left ventricular mass reduction than cuff pressure. Furthermore, high central pressure increases circumferential arterial wall stress, which causes a breakdown of medial elastin and increases the possibility of endothelial damage and the development of atherosclerosis. The Ew, due to wave reflection is wasted (pressure) energy the ventricle must generate during ejection and can be estimated as Ew. In the present study, we found, using multivariate logistic regression analyses controlled by age, sex, heart rate, and mean blood pressure, that both PWVm and aortic stiffness indices were still significantly associated with the occurrence of PVC loads in young persons. Extra workload was only marginally associated with PVC loads. A small proportion of patients in our study population had hypertension (27/200, 13.5%). However, LVH according to ECG criteria was only marginally significantly different between different PVC load groups (Table 1). It is interesting that the patients in the present study without LVH according to ECG voltage criteria also had an increased AIx, PWVm, and PVC loads, indicating increased aortic stiffness. The PVC loads might be an earlier marker than LVH for aortic stiffness in young persons.

Angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, β-adrenergic receptor antagonists, and statins effectively lower blood pressure and improve arterial stiffness. Drug impacts on PVC loads and arterial stiffness were insignificantly different between our study subgroups. This result connoted that PVC loads might be a surrogate marker for aortic stiffness in the early stage of atherosclerosis in a young population. Although patients with frequent PVCs may be asymptomatic or mildly symptomatic, high PVC loads might be an important marker for early aortic stiffness, which suggests that PVC load studies might be useful for guiding the aggressiveness of drug therapy in low-risk young persons. Optimal treatment of arterial stiffness and its complications should include a consideration of aortic stiffness, augmentation of aortic pressure, Ew, and the loads of PVCs, all of which should be reduced to the lowest possible level.

Study limitations
First, our study was limited by a cross-sectional rather than a longitudinal design. However, after we used multivariate logistic regression analyses that considered all possible confounders, aortic stiffness indices were still significantly associated with PVC loads in patients undergoing 24-hour ambulatory electrocardiography. Second, we did not measure the left ventricular systolic (by ejection fraction) and diastolic performances (by tissue Doppler) or left ventricular mass and index, using echocardiography. Those measurements might have provided a reason why increased aortic stiffness leads to abnormal left ventricular systolic or diastolic performances that led to PVC loads in this study population. Finally, pulse wave velocity may be associated with sympathetic nervous activity. We did not obtain the measurement of heart rate variability from 24-hour ambulatory ECG recordings.

Conclusions
High aortic stiffness indices may be surrogate markers of PVC loads in young patients undergoing 24-hour Holter electrocardiography due to palpitation. Central aortic properties might be important in PVC.

References


