

Conduction Delay-Induced J-Wave Augmentation in Patients With Coronary Heart Disease



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Electrocardiogram records were surveyed for the presence of an atrial premature beat (APB) and J waves in patients with coronary heart disease and patients with noncardiac diseases. The prevalence and response of J waves to sudden shortening of the RR interval on the conducted APB were determined and compared between the 2 patients groups. The change in the QRS complexes on the APB was also determined. Among 17,013 patients, 243 patients who underwent percutaneous coronary intervention for acute myocardial infarction or angina pectoris had an APB, and J waves were observed in 16 patients (6.6%). In an additional 729 patients with noncardiac diseases and APB, 19 patients showed J waves (2.6%; $p = 0.010$). The clinical features were almost similar between the ischemic and nonischemic groups. J waves were located more often in inferior and high lateral leads in the ischemic group. When the RR interval shortened from 942 ± 228 to 621 ± 175 ms and 869 ± 158 to 570 ± 118 ms at baseline and in the conducted APB ($p < 0.001$ for both), the J-wave amplitude increased from 0.16 ± 0.04 to 0.19 ± 0.06 mV ($p < 0.001$) and 0.21 ± 0.07 to 0.24 ± 0.08 mV ($p = 0.010$) in the ischemic and nonischemic groups, respectively. J waves in patients with chronic coronary heart disease and in patients with noncardiac diseases were augmented at short RR intervals together with distinct changes in the QRS complexes, and an augmentation of J waves at short RR interval may represent a conduction delay.

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Slurs or notches at the terminal part of the QRS complex on the surface electrocardiogram (ECG) are currently defined as J waves¹ and categorized under J-wave syndrome.² J waves can be observed in apparently healthy patients,³ in patients with idiopathic ventricular fibrillation,^{4–6} as well as in patients with coronary heart disease,^{7–13} and have been reported to be a risk for cardiac events. However, the mechanism of the J waves in ischemic heart disease has not been fully investigated. In this study, we investigated the prevalence and response of J waves and QRS morphology to sudden change in RR intervals in the conducted atrial premature beats (APBs) in patients with and without coronary artery disease.

Methods

The ECG records of 17,013 patients (aged 60 to 89 years) who visited Toda Central General Hospital from

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January 2015 to January 2017 were surveyed for the presence of APB and J waves. The patients with coronary heart disease had undergone percutaneous coronary intervention (PCI) for myocardial infarction (MI) or angina pectoris 3 or more months ago. The patients with nonischemic heart disease had noncardiac general diseases. A history of sudden cardiac death was denied in all family members, as well as in the patients in each group. None revealed overt sign of heart failure.

Patients with atrial fibrillation, Wolff-Parkinson-White syndrome, QT interval prolongation or shortening, Brugada syndrome, bundle branch block, or intraventricular conduction delay were excluded. Patients with idiopathic dilated, or hypertrophic cardiomyopathy, valvular heart diseases with more than mild severity, congestive heart failure (NYHA >2), previous coronary artery bypass grafting, and implantable devices, such as a cardioverter-defibrillator or a device for cardiac synchronization, were also excluded.

J waves were defined as notches or slurs at the terminal portion of the QRS complexes with an amplitude >0.1 mV above the isoelectric line in at least 2 contiguous leads^{1,5} and were measured in the lead that exhibited the maximum amplitude in the baseline beat, and in the APB and the beat next to the APB.^{14–16} The location of the J waves was classified as inferior (II, III, or aVF), left precordial (V4 to V6), right precordial (V1 to V3), or high lateral (I, aVL). The R- and S-wave amplitudes and ventricular activation time (VAT) were measured in the baseline and the APB (Figure 1). The VAT was measured from the onset of the QRS complex to the peak of the R wave. The changes in the R-/S-wave and VAT were considered to represent an alteration of the depolarization process over the ventricle.

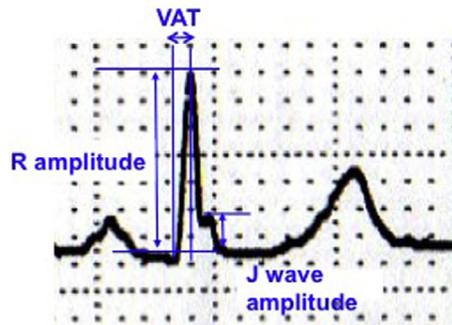


Figure 1. Measurements of the amplitudes of J waves, R waves, and the ventricular activation time (VAT).

The parameters were measured in the lead to identify the maximum amplitude of J waves.

The changes of the J waves and the R- and S-wave amplitudes were defined as augmented if the amplitude increased by ≥ 0.05 mV, unchanged for a change from -0.05 mV to $+0.05$ mV, and decreased for a change of -0.05 mV or more. The ECG parameters were measured after twofold magnification by 2 cardiologists who were blinded to the clinical findings. The type of J waves, notching or slurring, and the ST morphology after the J waves were determined according to the original report.¹⁷

The prevalence and dynamic changes of J waves to varying RR intervals were determined in the 2 patient groups. The responses of the J-wave amplitude to a change in the RR intervals were analyzed by comparing J waves among the baseline beats, on the conducted APBs, and on the beats immediately next to the APB as reported earlier.^{14–16} The maximum changes in the R- or S-wave amplitudes and VAT were analyzed in relation to the changes of the J-wave amplitude.

The numerical values are presented as the mean \pm SD, and the categorical variables are expressed as absolute numbers or percentages. The differences between the groups were analyzed by ANOVA for continuous variables and the Pearson's chi-square test for categorical variables. The J-wave amplitudes among the baseline beats, APB, and post-APB beats were compared by *t* tests in each group. *p* Values less than 0.05 were considered significant. The statistical analyses were performed using IBM SPSS software (SPSS 19; IBM Corporation, Chicago, Illinois). This study was approved by the Institutional Review Board of Toda Central General Hospital.

Results

APBs were observed in 972 of 17,013 ECG records, and 243 patients had ischemic heart disease who underwent PCI for MI ($n = 89$, 36.6%) or angina pectoris ($n = 154$, 63.4%) 3 or more months ago. Of these patients, J waves were observed in 16 patients (6.6%). The vessel treated was the left anterior descending artery ($n = 10$, 62.5%), the right coronary artery ($n = 4$, 25.0%), the left circumflex artery ($n = 3$, 18.8%), and the left main trunk ($n = 1$, 6.3%). One patient (6.3%) was treated for multivessel diseases. No patients developed ventricular tachycardia or ventricular fibrillation during the acute phase of MI and during follow-up periods.

Hypertension, diabetes mellitus, and dyslipidemia were identified in 16 (100%), 4 (25.0%), and 14 (87.5%) patients, respectively. A past history of MI was observed in 4 patients (25.0%), and 4 (25.0%) were current or past smokers. The patients were treated with an angiotensin-converting enzyme inhibitor, angiotensin II receptor blocker, calcium antagonist, or β blocker. Diabetes mellitus was treated with oral antidiabetic drugs or insulin (Table 1).

An additional 729 nonischemic patients with general diseases had APBs, and J waves were identified in 19 patients (2.6%), which was lower than the frequency in the ischemic group ($p = 0.010$). In the nonischemic group, hypertension and dyslipidemia were less frequent, and treatment with angiotensin-converting enzyme inhibitor /angiotensin II receptor blockers, calcium antagonists or statins occurred less often (Table 1).

There was no abnormality in the RR, PR, QRS, and QTc intervals in the ischemic and nonischemic groups, and the parameters were not different between the 2 groups (Table 2). The J-wave amplitude was 0.16 ± 0.04 mV in

Table 1
Clinical characteristics of patients with atrial premature beat and J waves

Variable	Ischemic group (n = 16)	Nonischemic group (n = 19)	p value
Age (years)	75 \pm 7	73 \pm 10	0.602
Men	11 (69%)	11 (58%)	0.844
Ejection fraction (%)	66 \pm 8	68 \pm 7	0.607
Hypertension	16 (100%)	7 (37%)	0.003
Diabetes mellitus	4 (25%)	3 (16%)	0.582
Dyslipidemia	14 (88%)	4 (21%)	0.002
Prior myocardial infarction	4 (25%)	0 (0%)	0.086
Smoker	4 (25%)	4 (21%)	0.602
Medications			
Angiotensin-converting enzyme inhibitor/angiotensin 2 receptor blocker	12 (75%)	3 (16%)	0.002
Ca blocker	12 (75%)	3 (16%)	0.002
Statin	14 (88%)	4 (21%)	0.002
β blocker	5 (31%)	2 (11%)	0.248
Oral diabetes drugs	1 (6%)	2 (11%)	0.566
Insulin	3 (19%)	1 (5%)	0.713

Table 2
Comparisons of ECG findings between ischemic and nonischemic patients showing J waves and premature atrial beat

Variable	Ischemic group (n = 16)	Nonischemic group (n = 19)	p value
RR interval (ms)	942 \pm 228	869 \pm 158	0.216
PR interval (ms)	173 \pm 28	173 \pm 24	0.948
QRS width (ms)	99 \pm 8	100 \pm 9	0.713
QT interval (ms ^{1/2})	443 \pm 27	423 \pm 35	0.370
Amplitude (mV)	0.16 \pm 0.04	0.22 \pm 0.07	0.008
Notch type	4 (25%)	7 (37%)	0.357
Horizontal/downward ST, Location	4 (25%)	6 (32%)	0.524
Inferior	6 (38%)	12 (63%)	0.035
Left precordial	2 (13%)	4 (21%)	0.418
Right precordial	0 (0%)	1 (5%)	0.304
High lateral	8 (50%)	2 (11%)	0.036

Table 3
Responses of J wave related to RR intervals

Variable	Ischemic group (n = 16)	Nonischemic group (n = 19)	p value
Baseline interval (ms)	942 ± 228	869 ± 158	0.216
RR of atrial premature beat (ms)	621 ± 175	570 ± 118	0.246
Postatrial premature beat RR (ms)	1097 ± 287	1073 ± 189	0.737
J-wave amplitude			
Preatrial premature beat (mV)	0.16 ± 0.04	0.21 ± 0.07	0.004
Atrial premature beat (mV)	0.19 ± 0.06	0.24 ± 0.08	0.018
Postatrial premature beat (mV)	0.15 ± 0.07	0.20 ± 0.07	0.005
Changes of J waves			
>0.05 (mV)	6 (38%)	6 (32%)	0.736
-0.05 to 0.05 (mV)	10 (63%)	13 (68%)	0.736

the ischemic patients and 0.22 ± 0.07 mV in the nonischemic patients ($p = 0.008$). The notched type was identified in 4 patients (25.0%) and 7 patients (36.8%) in the ischemic and nonischemic groups, respectively ($p = 0.357$). ST morphology with horizontal/downward displacement was found in 25.0% and 31.6% in the ischemic and nonischemic group. J waves were localized more frequently in the inferior or high lateral in the ischemic group ($p = 0.035$ and 0.036 , respectively).

In the 2 groups, the RR intervals shortened significantly on the conducted APBs and prolonged after the APBs (Table 3). At the short RR intervals, the J-wave amplitude was increased in 6 patients (37.5%) and unchanged in 10 patients (62.5%), whereas no patients showed a decrease. Overall, the J-wave amplitude was augmented from 0.16 ± 0.04 mV to 0.19 ± 0.06 mV at baseline and on APB, respectively ($p < 0.001$). In the nonischemic group, the J-wave amplitude was increased in 6 patients (31.6%) and unchanged in 13 patients (68.4%), whereas no patients showed a decrease on the conducted APBs. Overall, the J-wave amplitude was significantly augmented at the short RR intervals, 0.21 ± 0.06 mV to 0.24 ± 0.08 mV ($p = 0.010$). The J-wave amplitude returned to the baseline

in the beats immediately after the APBs in the 2 groups (Figures 2 and 3).

On the conducted APBs, the absolute changes in R- and S-wave amplitudes were 0.14 ± 0.15 mV and 0.18 ± 0.15 mV the ischemic and nonischemic group, respectively, with the exception of 2 patients: 1 patient each for the ischemic and nonischemic groups. There were no significant changes in the R- and S-wave amplitudes in the patients with and without augmentation: 0.14 ± 0.15 versus 0.11 ± 0.13 mV ($p = 0.4731$) and 0.10 ± 0.05 versus 0.15 ± 0.13 mV ($p = 0.2058$), respectively. The VAT did not exhibit a change in the conducted APBs.

Discussion

J waves were present in the patients with coronary heart disease who underwent PCI more than 3 months ago, more frequently than those with noncardiac diseases. The J-wave amplitude was significantly augmented at the short RR intervals of the conducted APBs, and returned to the baseline level in the beat next to the APBs that occurred with prolonged RR intervals. Concomitant changes of the R- or S-wave amplitudes may represent an alteration of the activation pattern over the ventricle due to a conduction delay induced by short RR interval. A conduction delay seems the mechanism of J-wave augmentation.

J waves may appear in association with acute MI⁷⁻¹³ or vasospasm angina,¹⁸ and have been reported as a risk for developing ventricular tachycardia/fibrillation or sudden cardiac death during acute MI. In MI, J waves were observed more frequently (14.4% to 26%) in patients with ventricular tachycardia/fibrillation or sudden death compared with those without ventricular tachycardia/fibrillation or sudden death (4% to 13%).⁷⁻¹⁰ J waves observed in early or late post-MI were also reported to be a predictor for ventricular and atrial tachycardia,^{9,10} sudden cardiac death,¹³ or shock delivery of an implantable cardioverter-defibrillator.¹¹ In the present study, J waves were present in ischemic and nonischemic patients, 6.6% versus 2.6%, respectively

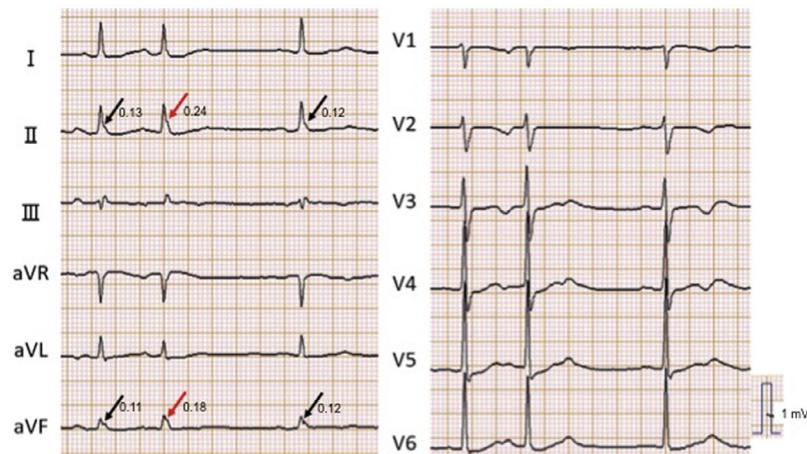


Figure 2. Changes in the J-wave amplitude with varying RR intervals in the conducted atrial premature beat.

The patient is a 70-year-old woman treated for inferior MI by percutaneous coronary intervention. J-wave amplitudes were present in the inferior leads (black arrows) and increased in the conducted atrial-premature-beat (red arrows). The J-wave amplitude after an APB was not augmented. Color version of figure is available online.

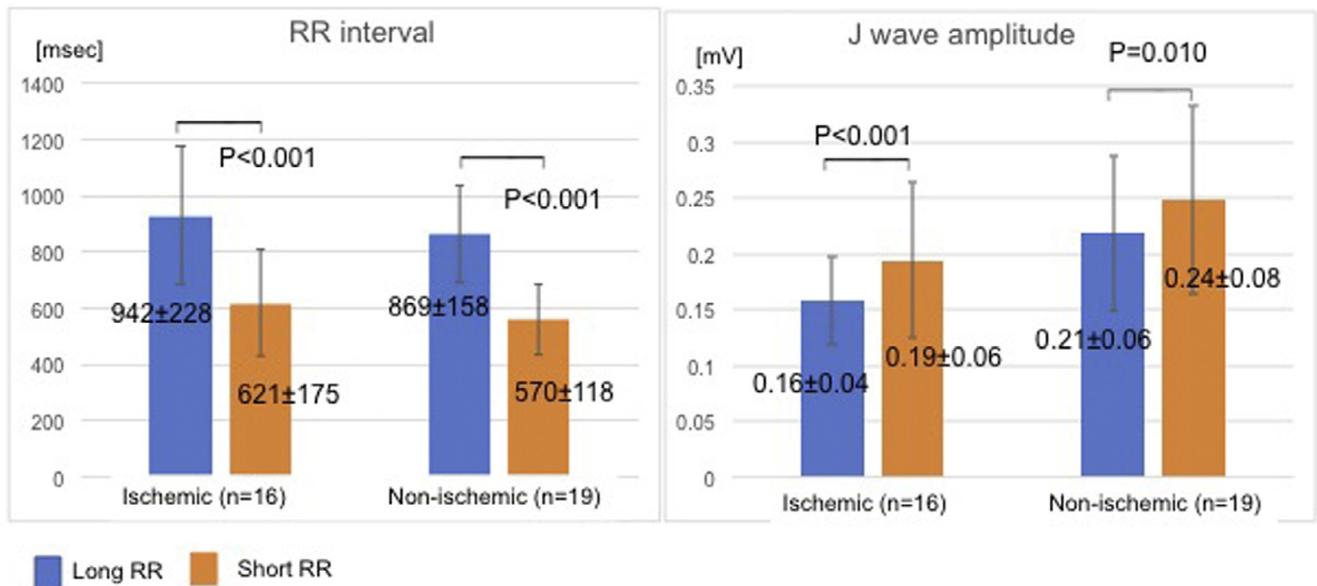


Figure 3. Changes in the J-wave amplitude at varying RR intervals. Left: The baseline RR intervals during sinus rhythm and those of the conducted atrial premature beats (APBs) with shortening.

Right: As the RR intervals shortened, the J-wave amplitude increased (augmented) in the ischemic and nonischemic groups.

($p = 0.010$), however, there was no evidence that J waves are associated with the occurrence of ventricular tachycardia/fibrillation or sudden cardiac death because of a short follow-up period.

Most J waves categorized in the J wave syndrome are considered to be mediated by transient outward currents (I_{to}), and an augmentation of the J-wave amplitude after a prolonged RR interval is the most striking features of I_{to}-mediated J waves.^{2,19} Clinically, J waves have been shown to indicate a pause-dependent augmentation in patients with idiopathic ventricular fibrillation.^{4,14,16} In contrast, J waves may be observed in patients without a history of VF or cardiac arrest, and these J waves may show augmentation of the J-wave amplitude at short RR intervals.¹⁵ Such tachycardia-induced augmentation of J waves can be observed after short RR intervals of the conducted APBs¹⁵ or during atrial pacing at a higher rate,¹⁶ and proved in patients with ischemic heart disease,^{12,20} in patients with noncardiac diseases and in general population.^{14–16} The different response patterns of the J-wave amplitude to varying RR intervals may be used to differentiate the underlying mechanism of J waves.^{14–16}

Distinct changes in the QRS complexes induced at short RR intervals can represent an alteration of the activation sequence over the ventricle from a tachycardia-induced conduction delay, but alteration in orientation of the heart in the pericardial sac due to variation of intracardiac pressures and timing intervals with changes in the RR intervals can be another mechanism.

The study included a rather small number of patients in a single center. However, the behaviors of the J waves on varying RR intervals are compatible with the results from previous studies.^{15,16} Drugs such as isoproterenol or quinidine might be used to differentiate the mechanisms of J waves; however, this approach was not attempted in the present study. Moreover, genetic analysis might provide

deep insights into the underlying mechanism of J waves; however, it was not performed in the present study. J waves could manifest as negative deflections, but such J waves were not focused in the present study but they should be investigated.

In conclusion, J waves of patients with coronary heart disease and those with noncardiac diseases showed an augmentation in amplitude at short RR intervals together with alterations in the QRS complexes. A conduction delay seems the mechanism for J-wave augmentation at short RR intervals; however, further confirmation is required.

Disclosures

The authors have no conflicts of interest to disclose.

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