Relationship between J Waves and Vagal Activity in Patients Who Do Not Have Structural Heart Disease

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Background: J waves are associated with increased vagal activity in patients with idiopathic ventricular fibrillation in several studies to date. However, the relationship between J waves and autonomic nervous activity in patients without structural heart disease remains under investigation. We investigated whether the presence of a J wave on the surface electrocardiogram (ECG) was related to increased vagal activity in patients without structural heart disease.

Methods: This retrospective study included 684 patients without structural heart disease who had undergone Holter ECG and surface ECG monitoring. Based on the presence of J waves on the surface ECG, patients were divided into two groups: those with J waves (group 1) and those without J waves (group 2). We compared heart rate variability (HRV), reflecting autonomic nervous activity, using 24-h Holter ECG between the groups.

Results: J waves were present in 92 (13.4%) patients. Heart rate (HR) in group 1 was significantly lesser than that in group 2 ($P = 0.031$). The ratio of low-frequency (LF) components to high-frequency (HF) components (LF/HF) in group 1 was significantly lower than that in group 2 ($P = 0.001$). The square root of the mean squared differences of successive NN intervals in group 1 was also significantly higher than that in group 2 ($P = 0.047$). In a multivariate regression analysis, male sex, HR, and LF/HF ratio remained independent determinants for the presence of J waves ($P = 0.039$, $P = 0.036$, and $P < 0.001$, respectively).

Conclusion: In patients without structural heart disease, the presence of a J wave was associated with a slow HR, male sex, and increased vagal activity, independently.

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A J wave on the electrocardiogram (ECG) is defined by positive deflections occurring at the junction between the QRS complex and the ST segment and characterized by a notching or slurring at the terminal part of the QRS complex.^{1,2} The J wave, also known as early repolarization (ER), may be accompanied by an ST-segment elevation in the inferior and/or lateral leads of a standard 12-lead ECG, an ECG feature referred to as an ER pattern. The prevalence of the ER pattern

is 1–15% in the general population,^{2–11} and it is more common in young physically active men and athletes.¹² The pattern of ER and J deflection, which was first reported by Shipley and Hallaran,¹³ has been accepted as a benign ECG variant for decades. However, recent studies have reported that the ER pattern is associated with an increased risk of arrhythmic and cardiac mortality in patients in idiopathic ventricular fibrillation (IVF).^{2,6,14} It also has been reported that the J wave

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amplitude was more significantly influenced by the autonomic balance in IVF than in the controls and J wave elevation was strongly associated with vagal activity in patients with IVF.^{15,16} However, the exact mechanism of J wave augmentation in the general population without structural heart disease remains unclear.

The purpose of this study was to investigate the relationship between J waves and vagal activity in patients without structural heart disease. We compared heart rate (HR) and time/frequency-domain heart rate variability (HRV) components between patients with and without J waves using 24-hour Holter and surface ECG monitoring.

METHODS

Study Population

We performed a retrospective study of 684 consecutive patients without structural heart disease who underwent surface ECG and 24-hour Holter ECG monitoring from 2009 to 2014. Clinical characteristics were assessed, and a surface ECG and transthoracic echocardiography were obtained in all patients. A 24-hour Holter ECG was performed within 5 days after the surface ECG was performed. The patients were divided into two groups based on the presence of J waves on the surface ECG: patients with J waves (group 1) and those without J waves (group 2). Time/frequency-domain HRV components were analyzed on the 24-hour Holter ECG. We compared time/frequency-domain HRV components, including standard deviation (SD) of NN (normal sinus to normal sinus) intervals (SDNN), SD of the averages of NN (SDANN), square root of the mean squared differences of successive NN intervals (rMSSD), proportion of NN50 divided by total number of NNs (pNN50), high-frequency (HF) components, low-frequency (LF) components, and the ratio of LF to HF (LF/HF) components on the Holter ECG between the two groups. We did not include patients with structural heart disease such as acute myocardial infarction, ischemic heart disease, hypertrophic cardiomyopathy, and dilated cardiomyopathy. We also excluded patients with atrial fibrillation, atrial flutter, QRS > 120 milliseconds, and paced rhythm. This study was approved by Inha University Hospital's institutional review board, and written informed consent was obtained from each patient.

J Wave Analysis on the Surface ECG

Standard 12-lead ECGs were digitally downloaded from the GE Marquette MUSE system (GE Medical Systems, Milwaukee, WI, USA) and were also analyzed digitally (Adobe Acrobat X Professional, Adobe Systems Incorporated, San Jose, CA, USA). Three independent cardiologists randomly analyzed the ECGs. On the standard 12-lead ECGs, we defined the J wave as a positive deflection at the junction between the QRS complex and the ST segment manifested through QRS notching or slurring for at least 1 mm (0.1 mV) above the baseline in at least two consecutive inferior or lateral leads. Notching was noted as a positive deflection at the terminal portion of a positive QRS complex (Figure 1A). Slurring was defined as a smooth transition from QRS complex to ST segment with upright concavity (Figure 1B).^{1,2,4} We excluded ECGs of Brugada pattern, which have J waves in the anterior precordial leads (V₁–V₃). Amplitude and location of the J wave on surface ECGs were investigated in all patients. We measured the J wave amplitude at the peak of the positive deflection in notched patterns and at the QRS–ST junction in slurred patterns with software using Screen Calipers 4.0 (Iconico Inc., New York, NY, USA).

Exercise Testing

We analyzed 31 patients with J wave who performed treadmill exercise testing (with a modified Bruce protocol) according to the standard method.¹⁷ Target HR was defined as 85% of the age-predicted HR as assessed by the equation. For the evaluation of functional capacity, metabolic equivalents (METs) were assessed (1 MET = 3.5 mL/kg per min of oxygen consumption) on the basis of the protocol and the total time elapsed in the final stage.¹⁸ An ischemic ST-segment change was defined as horizontal or a down-sloping ST-segment depression of >1 mm occurring 80 milliseconds after the J point. To evaluate exercise capacity between patients with and without J wave, the results of exercise testing were obtained and compared between patients with J wave and age- and gender-matched controls.

Analysis of Holter ECG Recordings

All patients underwent Holter ECG monitoring (MARS & SEER LIGHT EXTEND, V8.0.2; GE

Figure 1. (A and B) Examples of notching morphology J wave (arrows) and slurring morphology J wave (arrows) in inferior leads, respectively.

Healthcare, Milwaukee, WI, USA) within 5 days after surface ECG monitoring. Holter ECGs were continuously recorded for 24 hours during normal activities day and night. HRV was assessed with time/frequency-domain components from 24-hour Holter recording. We considered the time-domain parameters as follows: SDNN, SDANN, rMSSD, and pNN50. The frequency-domain parameters were analyzed by LF, HF, and LF/HF ratios including nighttime (0:00–6:00) and daytime (12:00–18:00) values. We calculated the LF (0.04–0.15 Hz) and HF (0.15–0.40 Hz) power using fast Fourier transform. Averaged values were recorded as the hourly data.

Statistical Analysis

Data are presented as mean $\pm$ SD for continuous variables and as proportions for categorical variables. Continuous variables were compared using the Student's *t*-test between patients with and without J waves. An analysis of categorical variables was performed using the chi-square test. The changes in HF components and LF/HF ratios during the daytime and nighttime were assessed using hourly data obtained in individual subjects. A *P* value <0.05 was considered statistically significant. Statistical analysis was performed using SPSS 21.0 statistical software (SPSS Inc., Chicago, IL, USA). Univariate regression analysis was used to identify relationships between clinical, electrocardiographic characteristics, time/frequency-domain HRV parameters, and the presence of J waves. Independent predictors of the presence of J waves were assessed using

multivariate logistic regression analysis. Figures were created using GraphPad Prism v.5.01 (GraphPad Software, Inc., San Diego, CA, USA).

RESULTS

In total, we enrolled 684 patients with a mean age of 46 ± 13 years, and 364 patients (53.2%) were men. All patients had no structural heart disease such as acute myocardial infarction, ischemic heart disease, hypertrophic cardiomyopathy, and dilated cardiomyopathy. The frequencies of hypertension, diabetes, dyslipidemia, and smoking history of our study population were 161 (23.5%), 46 (6.7%), 65 (9.5%), and 181 (26.5%) patients, respectively. Baseline characteristics, including age, sex, classical cardiovascular risk factors, and history of medication, did not show a significant difference between the two groups (Table 1). The mean value of left ventricular ejection fraction in our study population was $63.3 \pm 3.7\%$, and there were no differences in the two groups.

Prevalence of J Wave Components on Standard 12-Lead ECGs

In our study, we detected J waves on surface ECGs in 92 (13.4%) of 684 patients. Notched-type and slurred-type J waves were 65 (70.7%) and 27 (29.3%), respectively. There were 64 (69.6%) in the inferior lead, 25 (27.2%) in the lateral lead, and 3 (3.3%) in both leads. More men than women had J waves, but the *p* value had a nonsignificant trend [56 [15.4%] vs. 36 [11.3%], *P* = 0.118 for the

Table 1. Clinical Characteristics and Echocardiographic Findings between Patients with and without J Waves

	Total (n = 684)	J Wave (+) (n = 92)	J Wave (–) (n = 592)	P Value
Age, mean ± SD (years)	45.9 ± 12.5	44.8 ± 11.5	46.1 ± 12.5	0.370
Male (%)	364 (53.2)	56 (60.9)	308 (52.0)	0.118
Hypertension, n (%)	161 (23.5)	25 (27.2)	136 (23)	0.428
Diabetes, n (%)	46 (6.7)	6 (6.5)	40 (6.8)	0.933
Dyslipidemia, n (%)	65 (9.5)	13 (14.1)	52 (8.8)	0.124
Smoker, n (%)	181 (26.5)	23 (25)	158 (26.7)	0.733
Medication history				
CCB	115 (16.8)	16 (17.4)	99 (16.7)	0.873
ACEi or ARB	51 (7.5)	6 (6.5)	45 (7.6)	0.952
β-Blocker	10 (1.5)	2 (2.1)	8 (1.3)	0.122
Echocardiographic findings				
LVEDD (mm)	47.9 ± 3.7	47.8 ± 3.6	48.0 ± 3.8	0.761
LVESD (mm)	30.9 ± 3.2	30.8 ± 3.1	30.9 ± 3.2	0.103
LAD (mm)	35.6 ± 4.3	35.2 ± 4.2	35.6 ± 4.3	0.422
E/e'	8.61 ± 2.47	8.49 ± 2.09	8.63 ± 2.53	0.656
LVEF	63.3 ± 3.7	63.5 ± 3.0	63.2 ± 3.8	0.601

^aValues are mean ± SD or number (%).
Abbreviations as in text.

presence of J waves in men vs. women). The mean J wave amplitude was 0.15 ± 0.03 mV, and the frequency of a J wave amplitude ≥ 0.2 mV was 8 (8.7%).

Differences in HR and Time/Frequency-Domain HRV Components between Patients with and without J Waves

Mean HR on surface ECGs and average HR on 24-hour Holter ECGs of group 1 were significantly slower than those of group 2 (66.9 ± 11.8 vs. 73.1 ± 27.6 , $P = 0.031$ for HR on surface ECGs; 70.1 ± 8.3 vs. 73.4 ± 9.2 , $P = 0.001$ for average HR on 24-hour Holter ECGs; Table 2).

Group 1 tended to have higher HF than group 2, although the differences were not significant (5.22 ± 0.72 vs. 5.11 ± 0.91 , $P = 0.244$). The LF/HF ratio of group 1 was significantly lower than that of group 2 (2.12 ± 1.33 vs. 2.65 ± 1.63 , $P = 0.001$). We compared HF and LF/HF ratios during the daytime and nighttime, respectively (Fig. 2A and B). HF tended to have higher values and LF/HF ratios had significantly lower values in both the daytime and nighttime compared with those of group 2. Among time domain HRV parameters, the rMSSD in group 1 was significantly longer than that in group 2 (33.14 ± 11.90 vs. 30.43 ± 12.19 , $P = 0.047$) and group 1 had higher pNN50 values close to the boundary of significance compared to group 2 (11.30 ± 89.01 vs. 9.44 ± 8.66 , $P = 0.058$).

Differences of Circadian Variation in HRV between Patients with and without J Waves

We compared the circadian variations of HF and LF/HF ratios between group 1 and group 2 during the nighttime (0:00–6:00) and daytime (12:00–18:00) using 24-h Holter ECGs. HF increased during the nighttime compared to the daytime, and HF taken every hour in group 1 was also higher than that of group 2 during both the daytime and nighttime (Fig. 3A and B). Although the LF/HF ratios decreased during the night compared with those during the day, those of every hour were also lower than those of group 2 all day (Fig. 4A and B).

Differences of Results in Exercise Testing between Patients with and without J Waves

Only 31 patients had a treadmill test in group with J wave in this study. Additionally, the mean METs in 31 patients with J wave were compared with those of age- and gender-matched controls. Patients with J wave tended to have higher METs, but the differences were not significant (12.2 ± 1.7 vs. 11.9 ± 2.3 , $P = 0.588$). There were also no significant differences in exercise time, peak systolic blood pressure, peak HR, and ischemic ST-segment changes during exercise testing (Table 3).

Table 2. Surface and Holter ECG and Time/Frequency-Domain HRV Components between Patients with and without J Waves

	Total (n = 684)	J Wave (+) (n = 92)	J Wave (-) (n = 592)	P Value
Surface ECG				
HR on surface ECG (HR/min)	72.3 ± 26.1	66.9 ± 11.8	73.1 ± 27.6	0.031
QT (ms)	387.98 ± 32.09	392.63 ± 28.96	387.24 ± 32.52	0.128
QTc (ms)	419.07 ± 28.89	413.73 ± 24.61	419.93 ± 29.45	0.052
Holter ECGs				
Minimal HR (HR/min)	48.8 ± 6.9	47.7 ± 6.5	49.0 ± 7.1	0.091
Average HR (HR/min)	72.9 ± 9.2	70.1 ± 8.3	73.4 ± 9.2	0.001
Maximal HR (HR/min)	128.1 ± 18.8	125.6 ± 19.8	128.5 ± 18.6	0.171
LF (ln ms ₂)	5.93 ± 0.77	5.82 ± 0.71	5.94 ± 0.78	0.170
HF (ln ms ₂)	5.13 ± 0.89	5.22 ± 0.72	5.11 ± 0.91	0.212
Day LF (ln ms ₂)	5.77 ± 0.79	5.69 ± 0.64	5.78 ± 0.81	0.244
Night LF (ln ms ₂)	5.97 ± 0.85	5.88 ± 0.81	5.98 ± 0.86	0.295
Day HF (ln ms ₂)	4.66 ± 0.87	4.75 ± 0.75	4.65 ± 0.89	0.288
Night HF (ln ms ₂)	5.37 ± 0.99	5.49 ± 0.79	5.35 ± 1.01	0.148
LF/HF	2.58 ± 1.60	2.12 ± 1.33	2.65 ± 1.63	0.001
Day LF/HF	3.46 ± 2.00	2.94 ± 1.87	3.54 ± 2.01	0.017
Night LF/HF	2.29 ± 1.76	1.86 ± 1.35	2.35 ± 1.81	0.014
SDNN (ms)	138.73 ± 38.35	143.37 ± 39.49	138.01 ± 38.16	0.212
SDANN (ms)	125.05 ± 38.79	128.77 ± 40.54	124.47 ± 38.51	0.323
rMSSD (ms)	30.80 ± 12.18	33.14 ± 11.90	30.43 ± 12.19	0.047
pNN50 ;)	9.69 ± 8.72	11.30 ± 89.01	9.44 ± 8.66	0.058

^aValues are mean ± SD or number (%);

^bHF, LF, and LF/HF transformed by a natural logarithm. Abbreviations as in text.

Figure 2. (A and B) Comparisons of HF and LF/HF ratios between patients with and without J waves; HF, high frequency; LF/HF, ratio of LF components to HF components.

Comparison of J Wave Amplitudes According to Clinical and HRV Parameters

To determine whether certain parameters could influence J wave amplitude, we compared J wave amplitudes according to age, sex, HR, and HRV parameters. We divided the individuals into two groups by median values of age, HR, HF, LF/HF ratio, rMSSD, and pNN50 in our study population. As shown in Figure 5, the group of younger age (≤47 years) individuals and men had higher J wave

amplitudes compared with the group of older age individuals and women, but those values did not achieve statistical significance (0.159 ± 0.041 mV vs. 0.146 ± 0.029 mV, $P = 0.090$ for age ≤47 years vs. >47 years; 0.152 ± 0.043 mV vs. 0.146 ± 0.040 mV, $P = 0.517$ for men vs. women). The group with HR ≤65 had higher J wave amplitudes than the group with HR >65 (0.152 ± 0.043 mV vs. 0.146 ± 0.040 mV, $P = 0.150$ for HR ≤65 vs. >65). Groups with higher HF or lower LF/HF ratios had higher J wave amplitudes compared with those with lower HF or higher LF/HF ratios, although

Figure 3. (A and B) Changes in HF during the daytime and nighttime between patients with and without J waves; HF, high frequency; data are mean $\pm$ SD.

Figure 4. (A and B) Changes in LF/HF during the daytime and nighttime between patients with and without J waves; LF/HF, ratio of LF components to HF components; data are mean $\pm$ SD.

those values did not quite attain conventional levels of significance (0.158 ± 0.046 mV vs. 0.143 ± 0.037 mV, $P = 0.107$ for $\ln HF > 5.2$ vs. ≤ 5.2 ; 0.143 ± 0.037 mV vs. 0.158 ± 0.046 mV, $P = 0.107$ for LF/HF ratio > 2 vs. ≤ 2). J wave amplitudes of the group with higher rMSSD or pNN50 were also higher than those of opposite groups, but the values did not quite reach statistically significant levels (0.154 ± 0.036 mV vs. 0.145 ± 0.046 mV, $P = 0.299$ for rMSSD > 30 vs. ≤ 30 ; 0.155 ± 0.036 mV vs. 0.144 ± 0.047 mV, $P = 0.215$ for pNN50 > 8 vs. ≤ 8 ; Fig. 4).

Predictors of J Waves in Patients Who Have No Structural Heart Disease

Independent predictors of the presence of J waves are listed in Table 4. QTc interval, average

HR on a Holter ECG, and time/frequency-domain HRV components such as rMSSD and LF/HF ratio showed a significant correlation with the presence of J waves on surface ECGs. In multivariate regression analysis, male sex, average HR, and LF/HF ratio remained independent determinants for the presence of J waves ($\text{Exp}(\beta) = 1.695$, 95% confidence interval [CI]: 1.027 ± 2.797 , $P = 0.039$; $\text{Exp}(\beta) = 0.972$, 95% CI: 0.947 ± 0.998 , $P = 0.036$; $\text{Exp}(\beta) = 0.655$, 95% CI: 0.947 ± 0.998 , $P = 0.036$, respectively; Table 4).

DISCUSSION

The major findings are as follows: (1) We found J waves on surface ECGs in 13.4% of patients who had no structural heart disease in this study.

Table 3. Results of Treadmill Exercise Testing between Patients with J Wave and Age- and Gender-Matched Controls

	J Wave (+) (n = 31)	J Wave (-) (n = 31)	P Value
Age	45.6 ± 12.3	45.6 ± 9.9	0.741
Male (%)	20 (64%)	20 (64%)	1.000
Exercise time (min)	10.8 ± 1.7	10.5 ± 1.6	0.562
Metabolic equivalents (METs)	12.2 ± 1.7	11.9 ± 2.3	0.588
SBP at rest (mm Hg)	131.6 ± 18.4	127.6 ± 18.7	0.425
Heart rate at rest (beats/min)	83.4 ± 14.0	84.5 ± 16.2	0.788
Peak SBP (mm Hg)	182.2 ± 27.9	180.0 ± 18.9	0.875
Peak heart rate (beat/min)	175.2 ± 17.6	180.5 ± 20.2	0.301
Ischemic ST-segment changes (%)	3 (9.6)	2 (6.4)	0.669

^aValues are mean ± SD or number (%).

^bSBP, systolic blood pressure.

Figure 5. Comparison of J wave amplitudes according to clinical factors and HRV parameters; HF, high frequency; HR, heart rate; LF, low frequency; LF/HF, ratio of LF components to HF components; pNN50, proportion of NN50 divided by total number of NNs; rMSSD, root mean square of successive heartbeat interval differences.

(2) Patients with J waves had significantly slower HR compared with patients without J waves. (3) There were significant differences in time/frequency-domain HRV components (rMSSD and LF/HF ratio) between patients with and without J waves. (4) Patients with J waves had significantly lower LF/HF ratios in every hour during both the daytime and nighttime as compared with patients who had no J waves. (5) The main predictors of J waves were male sex, slow HR, and lower LF/HF ratio in the present study population of individuals with no structural heart disease.

The prevalence of J waves, also known as ER, in the general population is estimated at about

1–15% based on previous reports.^{2–6,11,19–25} In this study, the prevalence of J waves was 13.4%. The prevalence of J waves varies greatly among previous reports. The variation of J wave prevalence in different studies may be related to differences in definition, measurement method, and study demographics. Although there are great differences in J wave prevalence among previous studies, it is interesting that the prevalence of J waves in this study was similar to the J wave prevalence values in some studies that had similar values to our mean age and male proportion of the overall population.^{6,22,23} This suggested that the J wave is strongly associated with age and sex. Noseworthy et al.²⁴ demonstrated that the prevalence of

Table 4. Predictors Related to the Presence of J Waves

	OR	95% CI	P Value
Univariate regression analysis			
Age, >47 vs. ≤47 (years)	0.799	0.510 ± 1.251	0.326
Male vs. female	1.434	0.916 ± 2.247	0.115
Hypertension	0.899	0.486 ± 1.315	0.378
Diabetes	0.963	0.396 ± 2.339	0.933
Dyslipidemia	1.709	0.890 ± 3.280	0.107
Smoker	0.916	0.552 ± 1.518	0.733
QTc (ms)	0.993	0.985 ± 1.000	0.042
Average HR on Holter ECG	0.960	0.936 ± 0.985	0.002
rMSSD (ms)	1.017	1.000 ± 1.035	0.048
pNN50 (%)	1.023	0.999 ± 1.047	0.059
LF/HF	0.692	0.566 ± 0.846	<0.001
Multivariate regression analysis			
Age (years)	0.695	0.412 ± 1.173	0.054
Male vs. female	1.695	1.027 ± 2.797	0.039
QTc (ms)	0.994	0.986 ± 1.002	0.147
Average HR on Holter ECG	0.972	0.947 ± 0.998	0.036
rMSSD (ms)	1.023	0.946 ± 1.108	0.566
LF/HF	0.655	0.525 ± 0.817	<0.001

^aAbbreviations as in text.

J waves declines from early adulthood to middle age especially in men, which suggests a hormonal influence on the presence of J waves. The mean age was 46 ± 13 (range, 17–65) years and 53.2% were men in the present study. This might in part account for the modest impact of the difference of J wave prevalence between patients with and without J wave in this study. Wu et al.²⁶ estimated a very low absolute incidence rate of arrhythmic death in the middle-age group with an ER pattern (70 cases per 100000 subjects per year). Although there is a relatively high prevalence of J waves in the middle-age population and men, there is an absolutely very low ventricular fibrillation (VF) event rate in that population. Thus, the presence of J waves alone should not be considered a high risk for sudden cardiac death in patients with J waves who have no structural heart disease, especially in middle-age subjects and men. However, recent studies have demonstrated that J waves on the surface ECG have been associated with ventricular arrhythmia in an experimental model consisting of canine ventricular wedge preparations.²⁷ The concept has now expanded to include other structural heart disease such as acute myocardial infarction, variant angina and even some forms of cardiomyopathy.^{28–30} These findings suggest that the J wave may play a role as a predictor of poor prognoses in ischemic disorders.²⁷

Jerome et al.¹⁷ reported that subjects with ER had higher aerobic capacity than age- and gender-matched controls, which was associated with higher vagal tone. Our study also suggested that increased vagal activity was one of the major predictors of J waves in patients without structural heart disease. However, our data did not show significant differences of exercise capacity such as METs between patients with and without J wave although patients with J wave tended to have higher METs. This might in part account for a small number of patients who underwent treadmill test.

Some experimental studies have proposed that different action potential morphologies across the ventricular myocardium related to more prominent transient outward potassium current (I_{to}) result in a transmural voltage gradient, generating the J wave.^{31–33} The J wave also has been associated with increased vagal activity in patients with IVF in several studies to date.^{15,16} J wave augmentation was confirmed in about half of the populations with IVF after sudden RR prolongation in a previous report.³⁴ Haïssaguerre et al.³⁵ demonstrated that isoproterenol is effective in correcting the J wave pattern and suppressing VF recurrence. It has also been reported that HF components reflecting vagal activity increase at night and decrease during the day, and LF/HF ratios reflecting global sympathovagal balance show inverse patterns in patients with IVF.¹¹ Although several reports

about the relationship between J waves and autonomic balance in IVF patients exist, data for J waves associated with vagal activity in the general population are lacking. We compared vagal activities using HRV from 24-hour Holter ECGs according to the presence of J waves in patients without a history of IVF. In the present study, patients with J waves had significantly slower HR, increased rMSSD, and lower LF/HF ratio reflecting increased vagal activity as compared with patients who had no J waves. Additionally, HF and LF/HF ratios also showed a circadian variation and inverse pattern. The patients with J waves showed significantly lower LF/HF ratios in every hour during both the daytime and nighttime compared with patients who had no J waves. This suggests that increased vagal activity is also strongly associated with the presence of J waves in patients without structural heart disease.

It has been reported that male sex was significantly associated with the J wave compared to female sex.^{9,36} The prevalence of J waves declines in men from early adulthood until middle age.²⁴ J wave amplitude modulation is also affected by HR and autonomic tone.^{15,16} In our multivariate analysis to discriminate independent predictors for the presence of J waves, we found that male sex, slow average HR, and lower LF/HF remained significant determinants for the presence of J waves in patients without structural heart disease.

Study Limitations

This study had several limitations. First, this study was a retrospective analysis and was performed at a single center. Larger, prospective studies need to evaluate the relationship between the presence of J waves and vagal activity in the future. Second, our study consisted of a small number of patients who underwent treadmill test. We need more data of exercise testing to better understand exercise capacity in subjects with J wave. Third, the prevalence of J waves on surface ECGs and 24-hour Holter ECGs may differ. Holter ECG recordings and surface ECG recordings were not repeated in all patients because of the limitation of a retrospective study. Day-to-day variability in surface ECGs and reproducibility of the indices of HRV should be evaluated. However, this study showed that increased vagal activity is also associated with the presence of J waves in the general population.

CONCLUSIONS

Our study demonstrated that the presence of J waves in patients without structural heart disease was associated with a slow HR and increased vagal activity, independently. Our study also suggested that male sex and increased vagal activity might be the major predictors of J waves in patients without structural heart disease.

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