

Detection of Arrhythmogenic Substrates in Prior Myocardial Infarction Patients with Complete Right Bundle Branch Block QRS Using Wavelet-Transformed ECG

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Abstract

Background: It is important to follow up patients surviving acute myocardial infarction (MI), to detect the presence of any life-threatening arrhythmias. Various non-invasive examinations, such as signal-averaged ECG (SAECG), have been reported to predict the fatal ventricular tachycardia (VT); however, these conventional methods have limitations in detecting VT occurring in patients with complete right bundle branch block (CRBBB) QRS. Wavelet transform has been increasingly reported as a superior time-frequency analysis on the surface ECG in detecting abnormal high-frequency components (HFCs), thus suggesting abnormal myocardial conductions; however, it remains unclear whether wavelet-transformed ECG (WTECG) is useful in patients with CRBBB.

Objective: The purpose of this study is to assess the predictive value of WTECG for detecting arrhythmogenic substrates in MI patients with CRBBB.

Methods: Both the WTECG and SAECG were evaluated in 22 subjects with CRBBB, including 10 subjects without cardiovascular diseases (control group), 7 prior MI patients without VT (Non-VT group), and 5 prior MI patients with sustained VT (VT group). A 12-lead ECG (10 kHz sampling) was recorded and the representative QRS complex (300 ms) was transformed at a frequency range of 40–280 Hz using the Gabor function as the analyzing wavelet. In the power curve along a time course, the percentages of the peak power values at each frequency (60, 80, 120, 150, and 200 Hz) in the corresponding power values at 40 Hz (P60/40, P80/40, P120/40, P150/40, and P200/40, respectively) were calculated. ‘The power percentages (P120/40, P150/40, or P200/40) $\geq 50\%$ ’ was defined as an abnormal HFC (AHFC), and the number of the leads in which an AHFC was detected (NL-AHFC) of 8 leads (I, aV_F, V1–V6) was counted for comparison of the two MI groups.

Results: There was no significant difference among the three groups in the SAECG recording. The power percentages of HFCs (P120/40, P150/40, and P200/40) in Non-VT group were significantly higher than those in control group (48.2 ± 36.5 vs. 30.6 ± 7.7 , $P < 0.001$; 47.8 ± 35.5 vs. 26.9 ± 7.1 , $P < 0.001$; 47.3 ± 39.4 vs. 24.9 ± 7.6 , $P < 0.001$; respectively). NL-AHFC (P150/40) in VT group significantly increased more than in Non-VT group (3.2 ± 0.4 vs. 1.4 ± 0.8 , $P =$

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0.001). When 'NL-AHFC (P150/40) ≥ 3 ' was defined as abnormal, the sensitivity, specificity, positive and negative predictive values for detection of VT in MI patients with CRBBB was 100, 85.7, 83.3, and 100%, respectively.

Conclusion: WTECG might be a novel non-invasive method to detect arrhythmogenic substrates in MI patients with CRBBB. (J Nippon Med Sch 2009; 76: 291–299)

Key words: wavelet transform, high-frequency component, ventricular tachycardia, right bundle branch block, myocardial infarction

Introduction

Sustained ventricular tachycardia (VT) is a fatal complication of prior myocardial infarction (MI)¹. It is important that high risk patients are selected non-invasively. Clinical examinations, such as signal-averaged ECG (SAECG)², T wave alternans (TWA)³, and QT-dispersion⁴ have been used to non-invasively evaluate the arrhythmogenic substrates in patients with prior MI; however, all of these methods are either of limited or no value in the assessment of the arrhythmogenic substrates in patients with complete right bundle branch block (CRBBB) QRS. CRBBB is often associated with acute MI. The incidence and clinical follow-up studies of CRBBB in patients with acute MI have been reported. CRBBB occurs in from 4 to 7% of patients with acute MI and it is independently associated with a worse outcome^{5,6}. The SAECG has been recognized to be of value in the assessment of arrhythmogenic substrates in patients with prior MI; however, because of the fundamental problem that the wide QRS complex of CRBBB is due to the late activation of normal myocardium, the late potentials by the SAECG are often considered positive even in the normal subjects with CRBBB⁷.

Wavelet transform is a novel technique for time-frequency analysis. It is a high-resolution frequency analysis with a superior time resolution in comparison to Fourier transform for examining biological signals. The high-frequency components (HFCs) disclosed by this analysis are considered an index of conduction disturbance, which cannot be detected with the standard methods such as SAECG because of being superimposed by the high power of

low-frequency components (LFCs)⁸. Wavelet-transformed ECG (WTECG) can detect electrical abnormalities in the QRS complex on the surface ECG even if a high-gain QRS complex hides a small and significant change⁹. This high-resolution frequency analysis is just beginning to be applied to the surface ECG analysis of various heart diseases, such as hypertrophic cardiomyopathy^{10,11}, heart failure due to triple vessel disease¹², and Brugada syndrome¹³. The availability of WTECG in patients with prior MI has been reported^{8,14,15}; however, it remains unclear whether the arrhythmogenic substrates can be detected in patients with CRBBB using WTECG.

The object of the present study is to assess the predictive value of WTECG for identifying the MI patients with CRBBB at increased risk for fatal ventricular tachyarrhythmias.

Materials and Methods

Study Population

The study population included twenty-two subjects with CRBBB, including ten subjects without cardiovascular disease (control group), seven prior MI patients without VT (Non-VT group), and five prior MI patients with sustained VT (VT group). CRBBB was defined as follows; QRS complex duration ≥ 120 ms with broad and notched R waves (rsr', rsR', or rSR' patterns) in the right precordial leads (V1 and V2), and wide and deep S waves in the left precordial leads (V5 and V6). There was no significant difference among the three groups in the clinical characteristics, such as age, gender, and even measured parameters of SAECG (**Table 1**). All subjects underwent standard twelve-lead ECG,

Table 1 Characteristics of each group

	VT group (n=5)	Non-VT group (n=7)	control group (n=10)
age (years)	67.0 ± 6.8	58.4 ± 17.1	68.5 ± 10.9
male (n)	4	7	8
LP positive (n)	4	7	10
f-QRS (ms)	160.4 ± 17.4	157.3 ± 12.5	165.4 ± 12.7
RMS ₄₀ (μV)	15.3 ± 4.6	11.5 ± 6.1	9.3 ± 5.8
LAS ₄₀ (ms)	56.2 ± 18.3	72.7 ± 22.1	62.8 ± 36.9
EF (%)	43.2 ± 5.4 †	45.6 ± 5.3 †	66.7 ± 8.1
drugs			
beta-blockers (n)	4	6	
calcium antagonists (n)	0	1	—
ACE-I or ARB (n)	4	7	
peak CPK (IU/L)	4,220 ± 1,569	5,171 ± 1,272	—
peak CKMB (ng/mL)	556 ± 165	568 ± 185	—
infarct area (culprit lesion)			
anteroseptum (n)	3 (#6, #7, #7)	4 (#6, #7, #7, #7)	—
inferoposterior (n)	2 (#1, #1)	3 (#1, #1, #2)	

LP, late potential; f-QRS, root mean square (RMS) duration of the filtered QRS complex; RMS₄₀, voltage of the terminal 40 ms of RMS; LAS₄₀, low amplitude signal duration below 40 μV in the terminal RMS; EF, ejection fraction; ACE-I, angiotensin converting enzyme inhibitor; ARB, angiotensin receptor blocker; †, P<0.05 versus control group

transthoracic echocardiography, and exercise stress testing. All control group patients were referred for a routine medical checkup, and cardiovascular diseases were excluded. The diagnosis of MI was confirmed by cardiologists. All patients of the two MI groups underwent coronary angiography and successful percutaneous coronary intervention more than one month before study participation. Two of five subjects in the VT-group experienced a sudden cardiac death outside the hospital and were resuscitated by the bystander cardiopulmonary resuscitation. Their VT was confirmed on their automated external defibrillator monitor recordings. The remaining three patients were treated by implantable cardioverter-defibrillator (ICD)-implantation for the primary prevention. They had a history of non-sustained VT and inducible VT. Cardiologists confirmed the appropriate discharges for sustained VTs. Exercise stress tests or myocardial scintigraphy were performed in all patients in the two MI groups to rule out residual ischemia. All patients were studied before the administration of anti-arrhythmic drugs. There was no significant difference in background between the two MI groups, such as ejection fraction, infarct area, culprit artery, peak blood level of creatine

phosphokinase (CPK) and CKMB (**Table 1**).

Sustained VT was defined as a spontaneous wide QRS complex tachycardia with atrioventricular dissociation lasting for more than thirty seconds. The cases of VT induced by the electrophysiological study were excluded from this study, because it was possible that induced VTs contained non-clinical VTs. And VTs caused by the reversible disorders, such as a residual myocardial ischemia or a high blood level of drugs, were also excluded. High-resolution ECGs were obtained from all subjects during the duration of stable disease. The study protocol was approved by the institutional ethics committee and each patient gave their written informed consent before the recordings.

Signal-Averaged ECG Technique

The SAECG was recorded using a commercially available system (MAC5000, GE Medical Systems, Milwaukee, WI, USA) at the same time with WTECG recording. The X, Y and Z lead-ECG was recorded with a sampling frequency of 1 kHz in a shield room. More than 250 beats of QRS complex were averaged, amplified, and bidirectionally filtered with a bandpass filter (40–250 Hz). The root mean square (RMS, $\sqrt{X^2+Y^2+Z^2}$) was calculated and the

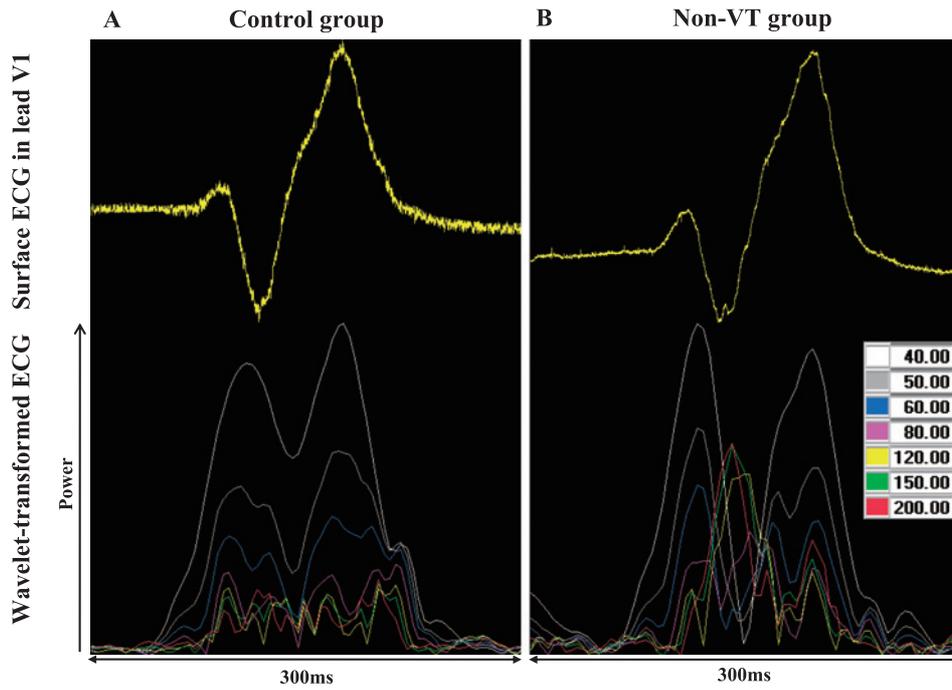


Fig. 1 Typical wavelet-transformed ECG shown in a power curve at each frequency level (40, 50, 60, 80, 120, 150, and 200 Hz) along a time course. In the wavelet-transformed ECG, white, grey, blue, purple, yellow, light green, and red indicate 40, 50, 60, 80, 120, 150, and 200 Hz, respectively. The powers of high-frequency components (120 – 200 Hz) in a Non-VT group patient (B) were higher than those in a control group subject (A). On the other hand, the powers of low-frequency components (40 – 80 Hz) demonstrated no significant difference between the two patients.

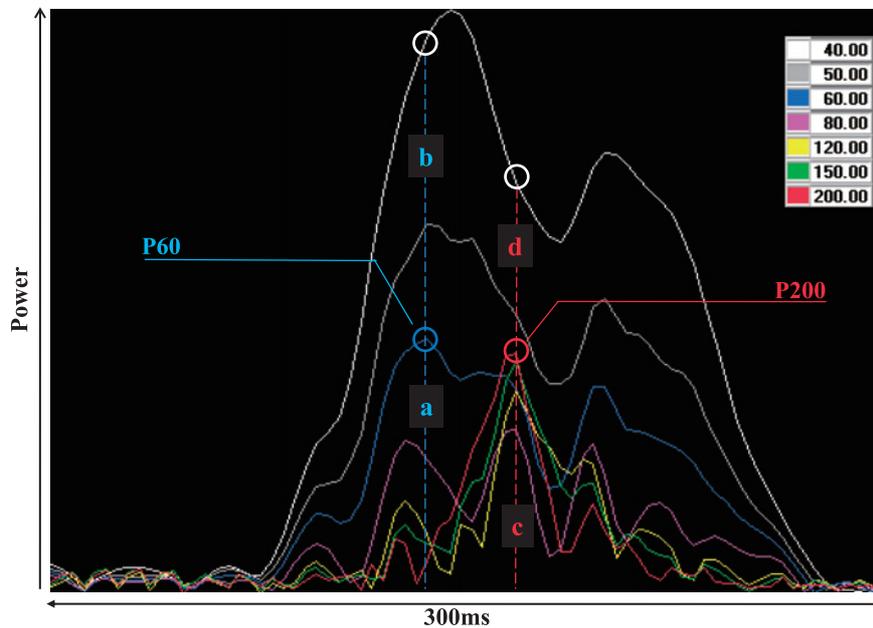


Fig. 2 The measurement methods of the wavelet-transformed ECG. First, the peak power values at 60 Hz (a) and 200 Hz (c) and the corresponding power values at 40 Hz (a+b and c+d) are measured. Next, the percentages of the peak power values at each frequency level (a and c) in the corresponding power values at 40 Hz (a+b and c+d) are calculated as: P60/40 is

$$\frac{a}{a+b} \times 100 \text{ and } P200/40 \text{ is } \frac{c}{c+d} \times 100 .$$

RMS duration of the filtered QRS complex (f-QRS), voltage of the terminal 40 ms of RMS (RMS₄₀), and the low amplitude signal duration below 40 μ V in the terminal RMS (LAS₄₀) were measured. The late potential was considered positive if two of the following criteria were met: (1) f-QRS \geq 114 ms, (2) RMS₄₀ < 20 μ V, (3) LAS₄₀ > 38 ms.

Wavelet Transform Technique

The continuous wavelet transform was performed for QRS complex analysis using a custom-made software package (WAVEROVER, Nihon Bussei, Tokyo, Japan). The modified Gabor function (wave number=4) was chosen as the mother wavelet because of its excellent time and frequency resolution in the high-frequency bands (80–200 Hz). We recorded a low-noise body surface twelve-lead ECG with a sampling frequency of 10 kHz and we randomly selected one representative noiseless QRS complex (300 ms). Digital signals of the QRS complex were transformed at a frequency range of 40–280 Hz using the modified Gabor function as an analyzing wavelet. The transformed signals were displayed in a power curve at different frequency levels (40–200 Hz) along a time course (**Fig. 1**). The peak power values at 60, 80, 120, 150, and 200 Hz (P60, P80, P120, P150, and P200, respectively) were measured, and the percentages of the peak power values at each frequency level in the corresponding power values at 40 Hz (P60/40, P80/40, P120/40, P150/40, and P200/40, respectively) were calculated for comparison of the three groups (**Fig. 2**). A high power value of HFC was considered an abnormal HFC (AHFC) if the power percentage (P120/40, P150/40, or P200/40) was more than 50%. In addition, the number of the leads in which an AHFC was detected (NL-AHFC) of 8 leads (I, aV_F, V1–V6) was counted in each subject. NL-AHFC was counted at 120, 150, and 200 Hz (NL-AHFC (P120/40), NL-AHFC (P150/40), and NL-AHFC (P200/40), respectively) and then compared between the two MI groups.

Statistical Analysis

All measurements are presented as the mean \pm standard deviation (SD). A comparison of

measurements between two groups was assessed using Student's unpaired *t*-test. All statistical analysis was performed using the SPSS statistical software package for Windows 14.0 (SPSS Inc., Chicago, IL, USA). The sensitivity, specificity, and positive and negative predictive values were also evaluated by the standard methods. Differences with $P < 0.05$ were considered significant.

Results

Signal-Averaged ECG

Each parameter of SAECG is shown in **Table 1**. There was no significant difference between the VT group and Non-VT group. The sensitivity, specificity, positive and negative predictive values of SAECG for detection of sustained VT in prior MI patients with CRBBB were low (80, 0, 36.4, and 0%, respectively). In addition, SAECG could not divide the two MI groups even from the control group because the late potentials in all control group patients were considered positive.

Wavelet-Transformed ECG

The power percentages of LFCs (P60/40 and P80/40) were not significantly different between the Non-VT group and control group (**Table 2**). The power percentages of HFCs (P120/40, P150/40, and P200/40) in Non-VT group were significantly higher than those in control group ($P < 0.001$, $P < 0.001$, and $P < 0.001$, respectively). The P150/40 in the Non-VT group and control group were 47.8 ± 35.5 (range, 20–202) and 26.9 ± 7.1 (range, 5–49), respectively. There was overlap of individual values between the two groups; however, 'P150/40 < 50% in all leads (I, aV_F, V1–V6)' distinguished the control group from Non-VT group (**Fig. 3**). Typical examples are shown in **Figure 1**. The power values in the Non-VT group at the high-frequency (120–200 Hz) were more developed in comparison to those in the control group. On the other hand, the power values at the low-frequency (40–80 Hz) were identical in the two groups.

The power percentages of HFCs in the VT group were higher than those in Non-VT group; furthermore, NL-AHFC (P120/40) and NL-AHFC

Table 2 Wavelet parameters

	VT group (n=5)	Non-VT group (n=7)	control group (n=10)
P60/40	59.8 ± 10.9	52.5 ± 9.1	52.3 ± 11.0
P80/40	49.9 ± 18.7	43.3 ± 24.2	39.1 ± 9.9
P120/40	56.7 ± 35.5 †	48.2 ± 36.5 †	30.6 ± 7.7
P150/40	54.4 ± 34.7 †	47.8 ± 35.5 †	26.9 ± 7.1
P200/40	51.1 ± 33.9 †	47.3 ± 39.4 †	24.9 ± 7.6
NL-AHFC (P120/40)	3.0 ± 1.2 ‡	1.0 ± 1.0	—
NL-AHFC (P150/40)	3.2 ± 0.4 ‡	1.4 ± 0.8	—
NL-AHFC (P200/40)	3.2 ± 1.6	1.7 ± 1.4	—

P60/40, P80/40, P120/40, P150/40, and P200/40 were calculated as the percentages [%] of the peak power values at each frequency level (60, 80, 120, 150, and 200 Hz, respectively) in the corresponding power values at 40 Hz as shown in Figure 2. NL-AHFC, the number of lead in which an abnormal high-frequency component was detected of 8 leads (I, aV_F, V1–V6), was counted at 120, 150, and 200 Hz (NL-AHFC (P120/40), NL-AHFC (P150/40), and NL-AHFC (P200/40), respectively) and compared between the two MI groups. NL-AHFC (P120/40) and NL-AHFC (P150/40) in VT group increased significantly more than those in Non-VT group (P=0.01 and P=0.001). There was no difference in NL-AHFC (P200/40) between the two MI groups (P=0.1). †, P<0.05 versus control group; ‡, P<0.05 versus Non-VT group

(150/40) in the VT group increased more than those in the Non-VT group (3.0 ± 1.2 vs. 1.0 ± 1.0, P=0.01; 3.2 ± 0.4 vs. 1.4 ± 0.8, P=0.001; **Table 2**). On the other hand, NL-AHFC (P200/40) did not differ (3.2 ± 1.6 vs. 1.7 ± 1.4, P=0.1).

The leads in which an AHFC was detected at the frequencies of both 120 (A) and 150 Hz (B) are shown in **Table 3**. The leads were not associated with the infarct area in any patient. The leads were confined to the precordial positions (V1–V3) in the patients of the Non-VT group (Pt. No. 6–12). On the other hand, the leads were distributed over a broad area (aV_F, V1–V6) in the patients of the VT group (Pt. No. 1–5).

Predictability for Sustained VT by Wavelet-Transformed ECG

When NL-AHFC (P150/40) ≥ 3 was defined as abnormal, the sensitivity, specificity, positive predictive value, and negative predictive value for detection of sustained VT in the prior MI patients with CRBBB were 100, 85.7, 83.3, and 100%, respectively (**Table 2**).

Discussion

The present study showed that the WTECG should be useful to predict VT occurring in prior MI

patients with CRBBB. In addition, these results suggest that WTECG can distinguish normal subjects with CRBBB from prior MI patients.

Low amplitude (μV level) and fractionated potentials are often recorded in the damaged myocardium in prior MI patients during an electrophysiological study¹⁶. These potentials may reflect the local abnormalities of impulse propagation in the MI-related scar, causing macro-reentrant tachyarrhythmia; however, they are too small to be detected on the surface ECG (mV level). Several approaches, such as frequency and time domain analysis of the SAECG^{17,18} were tried to detect the arrhythmogenic substrates on the surface ECG. SAECG is available in the assessment of arrhythmogenic substrates in prior MI patients without bundle branch block, having a high negative predictive value (over 90%) as reported^{2,19}; however, this yields no useful information in patients with CRBBB. In the present study, the negative predictive value of SAECG for detection of sustained VT in prior MI patients with CRBBB was 0%. That means all Non-VT group patients are considered positive. Furthermore, there is no identifiable difference even between the MI patients and normal subjects without detectable structural heart disease in the SAECG recording. CRBBB is a common ECG abnormality even in normal subjects, which has an

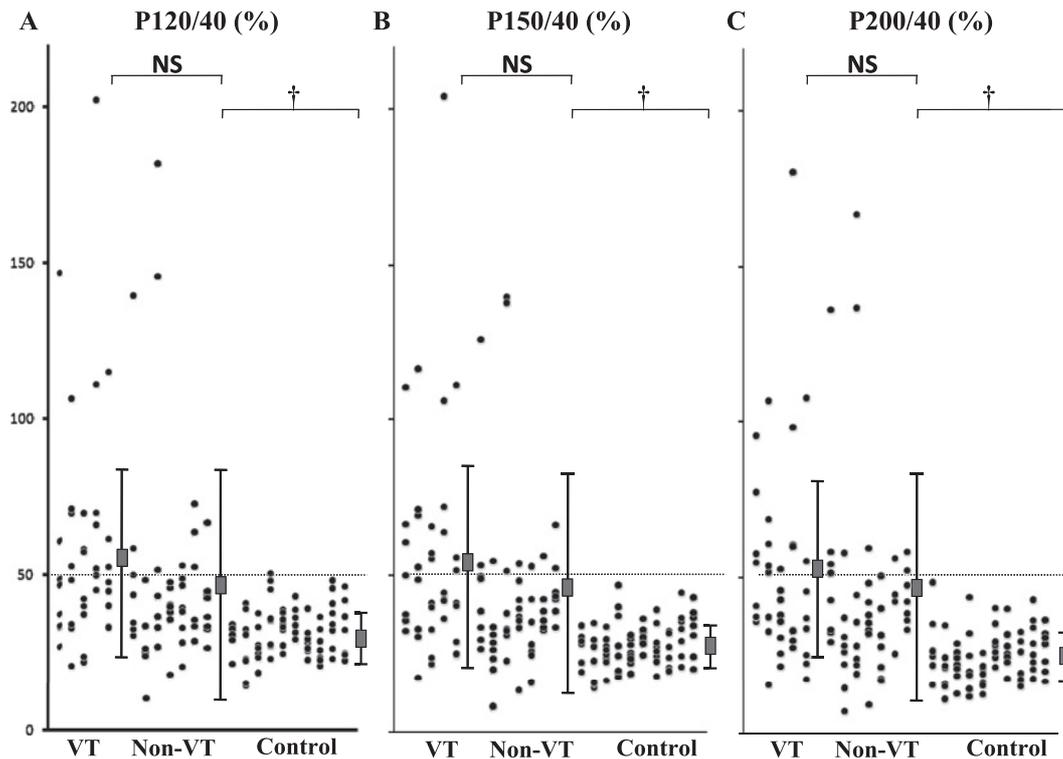


Fig. 3 Scatterplot showing the mean value \pm SD and individual values of the power percentages (P120/40 (A), P150/40 (B), and P200/40 (C)) of each lead in 22 subjects. The power percentages of high-frequency components (P120/40, P150/40, and P200/40) in Non-VT group were significantly higher than those in control group ($P < 0.001$, $P < 0.001$, and $P < 0.001$, respectively). In Non-VT group and control group, P120/40 were 48.2 ± 36.5 (range, 20–202) and 30.6 ± 7.7 (range, 5–49); P150/40 were 47.8 ± 35.5 (range, 8–138) and 26.9 ± 7.1 (range, 13–46); P200/40 were 47.3 ± 39.4 (range, 7–167) and 24.9 ± 7.6 (range, 10–48). There was some overlap of the individual values between the two groups; however, 'the power percentages (P120/40, P150/40, or P200/40) $< 50\%$ in all leads (I, aV_F, V1–V6)' distinguished the control group from the two MI groups. On the other hand, the powers of high-frequency components (P120/40, P150/40, and P200/40) between the VT group and Non-VT group were not significantly different (56.7 ± 35.5 vs. 48.2 ± 36.5 , $P = 0.4$; 54.4 ± 34.7 vs. 47.8 ± 35.5 , $P = 0.4$; 51.1 ± 33.9 vs. 47.3 ± 39.4 , $P = 0.6$). †, $P < 0.05$ versus control group; ‡, $P < 0.05$ versus Non-VT group; NS, no significant difference

incidence of 1 to 2% of the adult population^{20,21}; therefore, a method that can detect abnormalities in patients with CRBBB has been highly anticipated. The abnormal intra-QRS potentials located ahead of the delayed right ventricular activation must be evaluated to detect the conduction abnormalities in the left ventricular activation, because CRBBB is a result of conduction delay in the right-sided intra-ventricular conduction system. This method would preferably have a high time resolution.

Wavelet transform has a superior time resolution in comparison to Fourier transform⁸. Wavelet transform analysis is useful to detect arrhythmogenic substrates in prior MI patients as well as SAECG^{8,14,15}; however, wavelet transform

analysis in patients with CRBBB has never been reported. The present study calculated the percentages [%] of the peak power values at each frequency level in the corresponding power values at 40 Hz for a quantitative evaluation of an abnormal HFC in the QRS complex using the WTECG. A high percentage ($\geq 50\%$), meaning the projection of the HFC from the LFC, was found in the MI groups, even if they had CRBBB QRS morphology.

In addition, the MI patients were clearly differentiated from the normal subjects by the analysis at frequencies between 120 and 150 Hz. In normal subjects, the surface ECG is composed mainly of the frequencies from 20 to 40 Hz, and the frequencies above 80 Hz do not contribute to the

Table 3 Leads in which an abnormal high-frequency component was detected

A P120/40												
Pt. No.	1	2	3	4	5	6	7	8	9	10	11	12
	IP	AS	AS	AS	IP	IP	IP	AS	IP	AS	AS	AS
I												
aV _F	●	●	●	●	●							
V1	●			●	●			●			●	
V2		●	●			●				●	●	
V3		●			●						●	●
V4		●		●								
V5				●								
V6			●	●								
B P150/40												
Pt. No.	1	2	3	4	5	6	7	8	9	10	11	12
	IP	AS	AS	AS	IP	IP	IP	AS	IP	AS	AS	AS
I												
aV _F	●	●	●	●	●	●						
V1	●					●		●				●
V2	●	●	●			●				●	●	●
V3	●	●			●		●		●			
V4		●			●							
V5				●								
V6			●	●				●				

Pt. No., patient number (Pt. No. 1–5 indicates the patient in VT group and Pt. No. 6–12 indicates the patient in Non-VT group); AS, anteroseptal myocardial infarction; IP, inferoposterior myocardial infarction

surface ECG²². The HFCs, shown as small notches and slurs, on the surface ECGs of MI patients were first reported by Langner in 1953^{23,24}. It is thought that an abnormal HFC reflects an abnormal conduction zone, such as patchy necrosis and tissue fibrosis in heart^{25,26}. Popescu et al.¹⁵ suggested that a marker to distinguish prior MI patients with inducible VT from without VT was found between 200 and 300 Hz using the Morlet wavelet as a mother wavelet. In addition, Ota et al.¹² also suggested that HFCs between 120 and 350 Hz developed in patients with coronary heart diseases. In the previous studies^{8,12,14,15}, however, they did not clearly detect VT. The HFCs in the QRS complex of each lead may be too small to compare quantitatively between the MI patients with and without VT. The analysis of only one-lead ECG may have limitations; therefore, the number of the leads in which an AHFC was detected were counted in

each subject and then were compared between the two MI groups. The larger of NL-AHFC (P150/40) probably means that abnormal conductions can be detected in a more broad area, thus suggesting the existence of a large scar area or myocardia with multiple localized lesions.

Study Limitations

In this study, there were some limitations. First, a limited number of patients were studied, although the statistical power of the observations was significant. Secondly, this study was not a prospective study. Therefore, the relationship between the WTECG parameters and fatal ventricular events during the following up period could not be determined. A prospective study in a larger number of patients is therefore necessary in order to verify and confirm these conclusions.

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