Different rate-dependent responses between J waves and the notches on an epicardial local electrogram in a patient with idiopathic ventricular fibrillation

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Introduction
Notching or slurring on the terminal end of the QRS has been noted on standard 12-lead electrocardiograms (ECGs) with and without ST-segment elevation or T-wave alteration, which had long been considered a normal variant with benign outcomes.1-3 In recent decades, case-control studies have shown an association between such end-QRS notches or slurs and sudden cardiac arrest from ventricular fibrillation (VF) in patients without structural heart disease,4-7 and these ECG findings are called J waves.8 Patients who display early repolarization patterns such as J waves in the inferior and/or lateral leads presenting with aborted cardiac arrest, documented VF, or polymorphic ventricular tachycardia are diagnosed as having early repolarization syndrome (ERS); a diagnostic score system is proposed in the recent consensus report.9 ERS and Brugada syndrome are now thought to be the 2 major forms of J wave syndromes.

One of the striking features of J waves in idiopathic VF (IVF) is the pause-dependent augmentation,4,7,8,10 and bradycardia-dependent augmentation of J waves can be confirmed in approximately half of IVF patients.11 As the underlying mechanism, augmentation of transient outward currents at a slower rate is the most likely cause.12

Here, we report a case of an IVF patient, diagnosed as possible ERS by the proposed Shanghai ERS Score,9 in whom the rate-dependent change of the J waves was not evident on a standard ECG but was distinct in the notches in the local electrograms recorded from the epicardial surface of the left ventricle.

Case report
A 37-year-old, apparently healthy male subject developed cardiac arrest due to VF while sleeping at home and was successfully resuscitated by emergency personnel. The patient was transferred to a nearby general hospital. Routine examinations on admission revealed normal findings. No structural heart disease was demonstrated on echocardiography or on cardiac catheterization. Coronary artery spasm was negative on a provocation test.

The patient was transferred to our hospital for implantation of an implantable cardioverter-defibrillator (ICD). After ICD implantation, he had multiple ICD shocks for VF during sleep. VF was triggered by a single premature ventricular complex observed in the records of the ICD. A standard 12-lead ECG showed J waves in the inferior and lateral leads (Figure 1A). To examine the electrophysiological baseline of this patient, an electrophysiological study was attempted after informed consent was obtained. A catheter with 6 electrodes was introduced into the posterolateral vein via the coronary sinus and electrograms were recorded. Local epicardial electrograms were recorded with filter settings at 30-100 Hz for bipolar and 4-100 Hz for unipolar recordings. The electrode located in the inferior vena cava was used as an indifferent electrode for unipolar recordings.

The unipolar epicardial electrograms showed distinct notches of 2.24 to 2.72 mV, which occurred at exactly the same time as the peak of the J waves recorded on the surface ECG (Figure 1B). Premature electrical stimulation was given to the ventricle to induce a prolongation of the R-R interval. When the R-R interval was prolonged from 740 ms to 1180 ms, the notches of unipolar electrograms uniformly increased in amplitude from 2.72 mV to 2.96 mV in the lead revealing maximal amplitude, whereas J waves on the surface ECG remained unchanged at 0.13 mV (Figure 1B).

Then, rapid pacing at multiple cycle lengths was performed from the right atrium, which resulted in a distinct rate-dependent attenuation of the notch amplitude from 2.64 mV to 2.24 mV in the lead revealing maximal amplitude...
Figure 1  The 12-lead electrocardiogram (ECG) on admission and the epicardial electrograms. A: On admission, the standard 12-lead ECG showed J waves (as shown by arrows). Diagnostic J waves were present in leads I (0.12 mV) and III (0.13 mV). B: Epicardial electrograms were recorded in bipolar (Epi 1-2, 3-4, and 5-6) and unipolar (Uni 1, 2, 3) leads. In the unipolar leads, notches (arrowheads) are evident at the timing of the J waves on the surface ECG (arrow). The notches were augmented in the beat next to the premature ventricular stimulation when the R-R interval prolonged from 740 ms to 1180 ms. Epi = electrogram recorded by bipolar leads; RVA = right ventricular apex; Uni = electrogram recorded by unipolar leads.

Figure 2. However, attenuation of the J-wave amplitude was not evident on the surface ECG.

Bipolar epicardial electrograms were accompanied by tiny late potentials that extended out from the QRS complex. The duration of the local electrogram to the end of the late potential was shortened as the paced cycle length shortened (Figure 2).

VF was not inducible by programmed stimulation in this study.

Discussion
The patient illustrates a case of J wave–associated IVF. The rate-dependent change of the J wave was not evident on the standard surface ECG when the R-R interval was changed by electrical stimulation of the heart. Notches were observed in the unipolar epicardial electrograms at the timing of J waves on the surface ECG. The notches revealed distinct augmentation (attenuation) when the R-R interval was prolonged (shortened), which is a characteristic feature of transient outward current–mediated early repolarization.

Notches in the epicardial electrograms
Augmentation of the J waves was observed in IVF patients when the R-R interval was prolonged,4,7,8,11 and this is one of the characteristic features of J waves in IVF patients. Recently, Nakagawa and colleagues13 recorded electrograms from the epicardial surface of the left ventricle and studied the dynamicity of the notches of the unipolar epicardial electrograms from an IVF patient. The notches occurred at the

KEY TEACHING POINTS
- Prominent end-QRS notches (J waves) were observed in the epicardial electrogram recording via coronary sinus in patients with J wave syndromes.
- Owing to the slow recovery of transient outward current (Ito) from the inactivated state, Ito-mediated J-wave amplitude is rate-dependent. In patients with J wave syndromes, J wave is markedly potentiated during bradycardia or after pause.
- In cases with low-amplitude J waves, the rate-dependent changes of J wave may not be evident in surface electrocardiogram and could only be confirmed in the end-QRS notch (J wave) of epicardial electrograms.
- Recording epicardial electrograms could help characterize the underlying mechanism of the J wave.
timing of the J waves on the surface ECG, as was presented here. Distinct augmentation of the amplitude of the notches was observed when the R-R interval was prolonged after the termination of atrial pacing, whereas the notches were attenuated during atrial pacing at a higher rate. These rate-dependent changes were also confirmed in the J waves based on the changes in the surface ECG.

By contrast, our patient in the present study showed no rate-dependent changes of the J waves on the surface ECG, but the epicardial electrograms showed distinct characteristic changes of the J waves as an IVF patient (Figures 1B and 2).

In addition, the epicardial electrograms of the bipolar leads were accompanied by tiny late potentials that extended out from the QRS complex. The duration of the electrograms to the late potentials was progressively shortened as the atrium was paced at an increasingly higher rate (Figure 2). Similar shortening of the epicardial electrograms was observed during atrial pacing at a higher rate in the Nakagawa study. If the late potential is due to a conduction delay, then shortening the duration of epicardial electrograms at a higher rate would be difficult to explain; another mechanism should be considered.

Implications of the epicardial electrogram

Our previous study showed significant (≥0.05 mV) bradycardia-dependent augmentation of J waves in 15 of 27 IVF patients (55.6%), whereas changes were nonsignificant (0.05 mV or smaller) in the remaining 12 patients (44.4%). Clinical profiles were similar between the patients with and without bradycardia-dependent augmentation, and the only difference was the J-wave amplitude at baseline: 0.391 ± 0.126 mV vs. 0.196 ± 0.079 mV (P < .0001), respectively.

From the above study, it could be suggested that when J waves are small in amplitude, it may be difficult to confirm rate-dependent changes. Indeed, the patient in the present study had J waves with a small amplitude—0.13 mV—and only epicardial electrograms revealed the dynamic changes. In a patient from the study by Nakagawa and colleagues, the J waves were larger in amplitude than those of the patient in the present study, 0.38-0.52 mV, and characteristic rate-dependent changes were confirmed in both the J waves and the notches on the epicardial electrograms.

Therefore, when rate-dependent changes of J waves are not evident, recording epicardial electrograms and analyzing them can be helpful to characterize the underlying cause of
the J waves. Different dynamicity of J waves can discriminate the mechanism of J waves. J waves, or more precisely end-QRS slurs or notches, can be observed in middle- and high-aged subjects among a general population, and these J waves may often show an augmentation of the amplitude.

Conclusion
A 37-year-old male patient with IVF exhibited J waves on a standard ECG. During electrical stimulation of the heart, characteristic behavior of the J waves in IVF was not observed on the surface ECG but was distinctly observed in the epicardial electrogram.

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