Risk Stratification of Asymptomatic Patients With Brugada Type or J-Wave Type ECG

Akira Fujiki, MD, PhD

Cardiologists may be consulted about the risk of arrhythmic death after an incidental discovery of the Brugada or J-wave type ECG during a routine healthcare examination. The probabilities of induced VF by electrophysiological study (EPS) in patients with Brugada syndrome (BS) are highest for cardiac arrest survivors and lowest for asymptomatic patients. Thus, most cardiologists recommend an EPS for risk stratification of patients presenting with a Brugada-like ECG. However, the reliability of VF induction during EPS as a predictive indicator of a future spontaneous VF episode remains controversial.

There is general agreement on the excellent negative predictive value of EPS but not on the positive predictive value. In the Brugada series, 34% of 263 asymptomatic patients had inducible VF and 12% of them developed spontaneous VF. But in the meta-analysis by Paul et al, 25% of 720 asymptomatic patients had inducible VF and only 7% of them developed spontaneous VF, with a mean follow-up 34 months. The clinical significance of VF induction in asymptomatic patients with triple extrastimuli and shorter coupling intervals is problematic. As the number of asymptomatic patients who have inducible VF increases, many cardiologists have wondered whether or not such patients should undergo implantable cardioverter defibrillator (ICD) implantation. Unfortunately we do not know how to predict the true risk of a future VF episode in asymptomatic BS patients.

In this issue of the Journal, Hiratsuka et al compare the characteristics of induced VF cycle length between symptomatic and asymptomatic BS patients using fast Fourier transform (FFT) analysis. Symptomatic BS had a significantly shorter effective refractory period (ERP) at a basic cycle length (BCL) of 600 ms at the right ventricular outflow tract (RVOT) compared with asymptomatic patients. The mean VF duration induced by EPS was 14.6 s, most of them terminated electrically, and the dominant frequency (DF) of VF significantly increased with the duration of VF. Symptomatic BS patients had a higher DF of VF compared with asymptomatic patients. The authors propose that spectral analysis of induced VF may be a useful marker for risk stratification of BS patients. However, every symptomatic BS patient was asymptomatic before having the first spontaneous VF episode and it is possible that asymptomatic patients may have the same substrate as symptomatic patients. A difference in triggering mechanism may modify the induced VF frequency and may be responsible for the different electrophysiological characteristics between symptomatic and asymptomatic BS patients.

Most mapping studies have revealed that VF is characterized by multiple wave fronts circulating throughout the myocardium. The constant formation of new wave breaks is the distinctive feature of VF, because of the finite life span of the individual waves. There are controversies over whether these new wave breaks are themselves responsible for the continuation of fibrillation or are secondary phenomena caused by a rapidly firing focal source. The former is the multiple wavelet type and the latter is the spatially and temporally organized focal source type. FFT analysis has revealed multiple peaks in multiple wavelet type VF and stationary DF in focal source type VF.

In the present study, FFT analysis of idiopathic VF revealed stationary DF, which suggested VF of a focal origin. A rapidly firing focal source is the fundamental driver of VF and the multiple wavelets are caused by fibrillatory conduction, because the frequency of the focal source is too high to maintain 1:1 conduction to the surrounding myocardium. If a single mother rotor underlies the mechanisms of VF, catheter ablation of the single focus should cure the arrhythmia. Some idiopathic VF cases actually can be cured by focal ablation of the VF substrate at the RVOT. In the present study, the DF of the VF at the RVOT was expected to be higher than that at the right ventricular apex (RVA), but the DF at the RVOT was similar to that at the RVA. It is possible that the bipolar endocardial electrogram at RVOT did not represent the electrophysiological abnormalities of transmural myocardium.

The onset of VF immediately leads to global ischemia as a
result of circulatory arrest. Acute ventricular ischemia results in flattening of action potential duration (APD) restitution and reduced excitability. Chen et al. proposed that flattening of APD restitution induced by acute ischemia could prevent perpetuation of VF and promote spontaneous termination of VF. Patients with BS are known to have repeated aborted sudden cardiac death as a result of spontaneous defibrillation. The absence of associated structural heart disease might contribute to the self-defibrillation.

In our previous study, we analyzed Holter ECGs recorded just after VF episodes and found that idiopathic VF patients had lower QT/RR intervals, an impaired prolongation of the QT interval at longer RR intervals compared with healthy subjects from a control group, although there was an overlap. These repolarization characteristics of idiopathic VF patients during sinus bradycardia may be related to the nocturnal occurrence of spontaneous VF episodes. In the present study, symptomatic patients showed significant shortening of the RVERP at a BCL of 600 ms, but not at BCL of 400 ms compared with asymptomatic patients, similar to our previous study using Holter ECG.

Several studies have suggested that the presence of a prominent J on the ECG and decreased Na and Ca currents in the ventricular epicardial layer may play a key role in the characteristic ECG pattern of idiopathic VF. It can be speculated that these abnormalities of the ionic currents affect not only the configuration of the ST segment pattern but also ventricular repolarization dynamics. At rest, an increase in J on the ECG may limit the prolongation of the APD, especially at slower heart rates and also produce a prominent J wave on the ECG. During exercise, both a faster heart rate and an increase in adrenergic tone may offset the excessive J and make the difference in the ECG insignificant.

In idiopathic VF patients, the most reliable therapy for preventing sudden death is an ICD. However, not all patients can undergo ICD implantation and frequent episodes of VF reduce the quality of life because of frequent shock therapy. Patients with idiopathic VF should be given the option of undergoing ICD implantation and frequent episodes of VF reperpetuation of VF and promote spontaneous termination of idiopathic VF.

References