Brugada syndrome (BS) is a distinct form of idiopathic ventricular fibrillation (VF) characterized by a unique ECG pattern consisting of ST elevation in the anterior precordial leads with/without right bundle branch block-like morphology. It is generally accepted that patients with the type 1 ECG pattern (coved type) and with ventricular tachyarrhythmias (symptomatic BS patients) must receive an implantable cardioverter defibrillator to prevent a second VF attack. However, in those without syncope, family history or documented VF (asymptomatic BS patients), the best strategy is controversial because the prevalence of Brugada-type ECG change by daily medical check-ups has been reported to be approximately 0.1–1.0% in healthy subjects, but their prognosis is good compared with that of symptomatic patients. However, some asymptomatic BS patients occasionally become symptomatic and sudden cardiac death can occur with the first VF attack. Therefore, a marker that can differentiate high-risk asymptomatic patients from low-risk asymptomatic patients is needed. To date, several invasive and noninvasive parameters have been proposed for identification of patients at risk of VF, including spontaneous type 1 ST elevation, characteristics of the S wave, presence of late potentials, coexisting atrial fibrillation, augmented ST elevation after exercise, fragmented QRS wave, early repolarization pattern in the inferior/lateral leads, third intercostal ECG and inducibility of VF using programmed electrical stimulation, but the usefulness of these indexes remains controversial.

The results of a study by Miyamoto et al published in the Journal indicate the interesting possibility of using new ECG criteria for identifying high-risk asymptomatic BS patients. They performed computer-based ECG analysis for more than 100,000 patients and detected spontaneous type 1 ECG in 185 (0.18%) of the patients. Detailed examination was performed in 31 of these 185 patients, and 16 patients were diagnosed as high-risk BS (syncope: 87.5%, aborted...
sudden cardiac death/document VF: 68.8%). They concluded that a more negative T wave in lead V1 (<−1.05 μV), longer PQ interval (>170 ms) and family history of sudden death are associated with life-threatening events.

ECG patterns associated with BS have been classified into 3 types. Type 1 ECG is characterized by ≥2-mm J-point elevation, coved-type ST-T segment elevation, and inverted T-wave in leads V1 and V2. Type 2 ECG is characterized by ≥2-mm J-point elevation, ≥1-mm ST-segment elevation, saddleback ST-T segment, and a positive or biphasic T-wave. Type 3 ECG is the same as type 2 except that the ST-segment elevation is <1 mm. Among the 3 types, only the type 1 ECG is diagnostic of BS. An experimental study has revealed that the mechanism of ST-T abnormality in the right precordial leads is an outward shift of ionic currents during early repolarization (transmural voltage gradient) causing a marked accentuation of the action potential notch and prolongation of repolarization in the right ventricular epicardial but not endocardial cells (Figure). This discriminating electrophysiological mechanism is thought to be associated with ST-segment elevation (J wave) and T-wave inversion in this syndrome. In a human study using the activation recovery interval (ARI) method, Nagase et al demonstrated that the inverted T wave associated with the type 1 ECG is due to a preferential epicardial ARI prolongation secondary to accentuation of the action potential notch in the right ventricular outflow tract, consistent with the results of the experimental study. The data obtained in previous studies support the results of the present study. However, it has also been reported that this ECG pattern is very dynamic and often concealed during follow-up, and repeated ECG recordings should therefore be performed so as not to miss high-risk BS patients.

In BS, depolarization abnormalities, including prolongation of the P wave, PQ interval and QRS width, are sometimes observed, particularly in patients having severe forms of the gene mutation (SCN5A). Therefore, it would not be surprising that prolongation of the PQ interval is a risk marker in this syndrome, so we also need a careful attention to this marker.

In conclusion, although the number of high-risk patients was small, the follow-up period was short and further study is needed to reach a definitive conclusion, Miyamoto et al have provided important clinical evidence of a simple ECG marker for differentiating high-risk patients with BS-type ECG.

References