A Post-QRS Potential in Brugada Syndrome
Its Relation to Electrocardiographic Pattern and Possible Genesis

To the Editor: Brugada syndrome (BS) has been established as a clinical entity of idiopathic ventricular fibrillation (VF) and is characterized by the peculiar electrocardiographic (ECG) pattern in V1 and/or V2 (1). The characteristic ECG patterns, coved- or saddleback-type ST-segment elevation, have now been verified to be caused by the transmural voltage gradient created via a loss of the dome of the action potential of the epicardial myocardial cells (2).

Signal-averaged electrocardiogram (SAECG) may be employed in BS, and many patients have been found to have late potential (LP), which has been considered to represent local conduction delay (3). However, whether the LP in BS is related to local conduction or other mechanism has yet to be elucidated (4). In the present study, we analyzed the data of SAECG for a potential appearing separate from the QRS complex in patients with BS (4) and studied its nature.

All patients met the diagnostic criteria of BS (5), and 18 patients showed spontaneously developing coved-type ST-segment elevation. Except for 1 patient, all were men, and the mean age was 55 ± 12 years; the range was 33 to 74 years. Eleven patients had syncopal episodes, and 4 had aborted cardiac arrest. Ventricular fibrillation was not recorded, and all patients denied a family history of sudden cardiac death. Electrophysiologic study was performed in all patients except 1, and VF was induced in 16 patients. An implantable cardioverter-defibrillator (ICD) was implanted in these 16 patients after informed consent.

In these 18 patients showing spontaneous coved-type ST-segment elevation, SAECG was recorded 2 to 3 times (2.4 ± 0.5 on the average in a standardized manner), and 2 or more of the criteria of LP (3) were met in all patients. The fQRS complex was 160 ± 16 ms, LAS40 was 53 ± 10 ms, and RMS40 was 10.7 ± 3.3 μV. Of these 18 patients, 5 (27.8%) showed a discrete delayed potential in SAECG among the repeated studies, whereas V1 and V2 showed some variations in amplitude and/or morphology. The potential was separated from the terminal of the QRS complex by an isoelectric line. Quinidine abolished both the potential and coved-type ST elevation, and when the drug was discontinued, the potential reappeared (Fig. 1).

The other group consisted of 11 patients showing saddleback-type ST elevation who were referred to us for further evaluation. All were men, and the mean age was 54 ± 14 years. Two patients had episodes of presyncope, but none had a history of cardiac arrest or a family history of sudden cardiac death. Ventricular fibrillation was induced among 8 patients who underwent electrophysiologic study, and 5 received ICD implantation.

After admission, all patients received a pharmacological test for diagnosis of BS using pilsicainide. The drug was given at 1 mg/kg in 5 min, and at the final dose of 46.5 ± 15.6 mg, coved-type ST-segment elevation was induced in all patients. The conventional LP was positive in 8 of 11 patients before pilsicainide, whereas all patients showed saddleback-type ST elevation. When coved-type ST-segment elevation was induced, LP became positive in all. The fQRS complex prolonged from 158 ± 12 ms to 190 ± 12 ms, LAS40 from 31 ± 12 ms to 52 ± 11 ms, and RMS40 from 17.7 ± 7.7 μV to 8.9 ± 4.1 μV.

During coved-type ST-segment elevation, the post-QRS complex was found in 2 patients. In 4 patients, the low amplitude potential was marginally separated from the main body of the QRS complex. The RMS20, which covered the post-QRS potential or the latest part of LP, was not altered by pilsicainide (3.3 ± 2.0 μV vs. 3.0 ± 2.1 μV).

Brugada syndrome is often accompanied by a conduction abnormality—a prolonged HV interval (1) or a prolonged intraventricular conduction at electrophysiologic study—and LP is often positive (4). Nagase et al. (6) recorded a discrete potential from the epicardium, but not from the endocardium, at the outflow tract of the right ventricle. The timing of the potential was identical to the latest part of LP, and its onset was delayed by pilsicainide.

In the present study, LP was positive during coved-type ST-segment elevation, and some patients showed a discrete potential separated by an isoelectric line from the QRS complex in SAECG (Fig. 1).

The potential is characterized by the low-frequency components, and our previous study (4) showed that the so-called LP measured at 40 Hz of the low-cut filter disappeared when measured at 100 Hz in BS, whereas LP in the patients with right ventricular cardiomyopathy was not affected by such filter settings. The potential or the latest part of the LP (RMS20) remained unchanged even after pilsicainide in BS. In addition, quinidine abolished the potential concomitantly with a change in ST-segment (Fig. 1). These characteristics seemed to differ from those expected in the classical LP owing to conduction delay (7).

The characteristic ECG pattern of BS can be produced by a transmural voltage gradient (2) and, when large enough, the second upstroke of the dome of the action potential may be detected by SAECG. The post-QRS potential in the present study and that recorded by Nagase et al. (6) may represent such an abnormal potential of repolarization (6).

As a limitation, the number of patients is small, and the incidence of the post-QRS potential is relatively low. However, repeated SAECG recordings during coved-type ST-segment elevation would reveal higher incidence. In summary, some patients with BS may show a discrete potential after QRS complex, and the nature of the potential suggests a mechanism other than conduction delay.

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REFERENCES

Letters to the Editor

Human Ventricular Action Potential Duration Restitution

We read with interest the paper by Narayan et al. (1) that related right ventricular (RV) action potential duration (APD) restitution slopes to the presence of T-wave alternans (TWA) and ventricular tachycardia (VT) inducibility in patients with left ventricular (LV) dysfunction (LV ejection fraction [EF] 28 ± 8%). Standard restitution was measured by plotting APD_{90} as a function of the preceding diastolic interval (DI) during extrastimulus testing (S1S2). The prevalence of steep restitution slopes (>1) was similar between patients with positive or negative TWA, and mean restitution slopes were no different between those with inducible or noninducible VT.

Both these findings are contrary to a study reported by our group in a similar patient population (LVEF 30 ± 8%) (2). We feel this discrepancy can be accounted for by considering differ-