Inappropriate Discharges From an Intravenous Implantable Cardioverter Defibrillator Due to T-Wave Oversensing

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This report describes the clinical management of 2 patients with ventricular fibrillation (VF) who received inappropriate shocks from an implantable cardioverter defibrillator (ICD) due to T-wave oversensing. Cardiac sarcoidosis was confirmed as the underlying heart disease in 1 patient and idiopathic dilated cardiomyopathy in the other. Within 2 months after ICD implantation, both patients received several inappropriate shocks during sinus rhythm. Stored electrograms showed decreased R-wave amplitudes and increased T-wave amplitudes. The ICD sensed both R- and T-waves as ventricular activation, which met the rate criteria for VF treatment. Reprogramming the sensing threshold in association with administration of a drug to slow the heart rate decreased the incidence of the inappropriate shocks in both patients, but these palliative measures did not completely suppress the inappropriate shocks. To avoid T-wave oversensing, the repositioning or adding of a sensing lead is required. The potential risk of T-wave oversensing in ICD patients who have small R-wave amplitudes should be recognized. (Jpn Circ J 2001; 65: 685–687)

Key Words: Implantable cardioverter defibrillator; Inappropriate shock; T-wave oversensing

Case Reports

Case 1

A 65-year-old female was referred for further treatment of cardiac arrest caused by VF. She had been diagnosed as having cardiac sarcoidosis and a daily 50-mg oral administration of prednisolone (PSL) had been started in another hospital. However, VF developed during tapering of the PSL dosage. When she was subsequently admitted to hospital, her chest X-ray showed mild cardiomegaly and bilateral hilar lymphadenopathy, but no pulmonary congestion. The 12-lead ECG during sinus rhythm showed complete right bundle branch block. Two-dimensional echocardiography revealed no abnormality of left ventricular wall motion or chamber size, and 99mTc-pyrophosphate revealed no abnormal uptake. After obtaining written informed consent, an electrophysiological study (EP) was performed using the standard technique. Although only non-sustained VT were induced by programmed stimulation, ICD therapy was indicated because VF had been documented clinically.

In April 1998, a transvenous ICD system (Medtronic Micro Jewel II, 7223Cx, model 6943 right ventricular screw-in lead, Minneapolis, MN, USA) was implanted. At operation, shocks of 20J were twice confirmed to be capable of terminating VF induced by T-wave shock, and 30J was set as the initial treatment of VF. The amplitude of the ventricular electrogram from the tips of the defibrillation lead was 7.5 mV during sinus rhythm and the pacing threshold at the site was 2.0 V at a pulse width of 0.10 ms. One month later, the patient experienced 4 discrete inappropriate shocks during sinus rhythm of 94 beats/min. The stored electrograms showed decreased R-wave amplitude (2.1 mV) and increased T-wave amplitude (2.3 mV). Both R- and T-waves were detected at the ventricular sensing threshold of 0.3 mV and this led to the inappropriate ICD discharge during sinus rhythm (Fig 1). The sensing parameter was subsequently reprogrammed to 0.6 mV, and the VF sensing function at this sensitivity was confirmed by the defibrillation test. In this ICD, after the sensed event, the sensitivity threshold is adjusted to approximately 10 times the pro-
phosphorus revealed an abnormal lead was 8.0 mV during the sinus rhythm, and the pacing recorded intraoperatively from the tips of the defibrillation treatment of VF. The amplitude of endocardial electrograms ICD system was implanted and 30 J was set as the initial documented. In March 1999, the same model transvenous candidate for ICD therapy because VF had been clinically by programmed stimulation, the patient was considered a sustained polymorphic ventricular tachycardia was induced.

**Case 2**
A 26-year-old woman had an episode of aborted sudden cardiac death in October 1998. She had no prior history of syncope, nor was there any family history of sudden cardiac death. She was initially hospitalized at another institution and was diagnosed as having idiopathic dilated cardiomyopathy (DCM). She was referred to us for further evaluation.

Upon admission, chest radiography showed mild cardiomegaly but no pulmonary congestion. The 12-lead ECG during sinus rhythm showed small R-waves in the precordial leads but no ST-T abnormality. An echocardiogram revealed dilatation of the left ventricle and atrium. Although coronary angiography did not reveal any stenotic segments, left ventriculography showed dilatation of the left ventricle and the ejection fraction was 38%.

After obtaining written informed consent, an EP was performed using the standard technique. Although only non-sustained polymorphic ventricular tachycardia was induced by programmed stimulation, the patient was considered a candidate for ICD therapy because VF had been clinically documented. In March 1999, the same model transvenous ICD system was implanted and 30 J was set as the initial treatment of VF. The amplitude of endocardial electrograms recorded intraoperatively from the tips of the defibrillation lead was 8.0 mV during the sinus rhythm, and the pacing threshold at the site was 2.0 V at a pulse width of 0.15 ms.

The patient experienced 2 discrete inappropriate shocks during sinus rhythm of 100 beats/min 2 months later. Stored electrograms showed decreased R-wave amplitude (3.5 mV) and increased T-wave amplitude (2.5 mV). Both R- and T-waves were counted and satisfied the rate criteria of VF (Fig 2). There was no change in the 12-lead ECG. The sensing parameter was subsequently reprogrammed to 0.6 mV, and the VF sensing function at this sensitivity was confirmed by the defibrillation test, as in case 1. Moreover, a p-blocker and diltiazem were used to slow the heart rate. However, these measures did not suppress the incidence of inappropriate ICD discharge completely and during the follow-up period, although the clinical VT did not recur, inappropriate discharge recurred 3 months later. We recommended reoperation, but the patient refused and has been carefully observed since.

**Discussion**
This report describes 2 patients who experienced inappropriate discharge associated with T wave oversensing with a fourth-generation ICD device. In both cases, decreased R wave amplitudes and increased T wave amplitudes caused the T wave oversensing, and although the precise mechanism of T-wave oversensing remains unclear, the incidence of inappropriate discharge was not completely suppressed by reprogramming the device and/or administering drugs to slow the heart rate.

The mechanism responsible for the double counting of R- and T-waves includes electrolyte abnormality, drugs, changes in sympathetic tone and alteration of the shape of the intracardiac electrogram. The T-wave oversensing described here is clearly different from the hyperacute T-wave changes sometimes seen during implantation. The T-wave oversensing in the present patients was associated with marked diminution of R-wave amplitude and increased T-wave amplitude, although the exact mechanism of these alterations is unclear. A diseased myocardium would be associated with an alteration of the R-wave amplitude, and the activity of the disease might have affected case 1, because increasing the PSL dosage decreased the T-wave amplitude and the incidence of inappropriate discharges.
However, in case 2, a diseased myocardium is less likely to be associated with this problem, because 1 h after the inappropriate discharges when we examined the device we found that the T-wave amplitude had recovered its normal configuration. Lead dislodgment or abnormal mechanical stress on the lead might have caused these alterations, but the position and loop of the lead did not change on the chest X-ray.

The theoretical solution to the problem of T-wave oversensing is repositioning or adding a sensing lead. Because the discharges synchronized to a sensed T-wave during sinus rhythm have the potential to induce VT, we strongly recommended reoperation to both patients, but they refused. Thus, we reprogrammed the sensitivity for the gain control, changed the number of intervals to detect VT and/or slowed the heart rate to a levels that would not satisfy the rate criteria for VF treatment, even if a T-wave was sensed. However, in both cases these palliative measures did not suppress T-wave oversensing completely. However, raising the sensing threshold to prevent T-wave oversensing during sinus rhythm imposes potential risk on the delayed confirmation of arrhythmic events and the accuracy of detecting VF. Thus, it the palliative measures will be insufficient to avoid T-wave oversensing completely.

In conclusion, we have described 2 cases of T-wave oversensing. The potential risk of T-wave oversensing must be recognised in patients undergoing ICD treatment who have small R-wave amplitude. To completely suppress T-wave oversensing, the repositioning or adding of a sensing lead is essential.

References