Both Low and High Energy Cardioversion Induced Accelerated Ventricular Tachycardia in a Patient Treated with an Implantable Cardioverter Defibrillator

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SUMMARY

A 72-year old male with an old myocardial infarction who had drug-refractory ventricular tachyarrhythmias received an implantable cardioverter-defibrillator (ICD). The patient did not take his prescribed β -blocking agent for two days, following which he experienced six discrete shocks for spontaneous VT while riding his bicycle. Both 5J and 30J cardioversions were ineffective at terminating the VT and accelerated VT developed following the shocks. After admission, an electrophysiological study was performed while he was taking the β -blocking agent, both low and high energy cardioversions reproducibly terminated the clinical VT without showing any accelerated rhythm. These findings suggest that the increase in sympathetic discharge may enhance the proarrhythmic potential of ICDs. (*Jpn Heart* 1999; 40: 665-669)

Key words: Ventricular tachycardia, Cardioversion, Implantable cardioverter defibrillator

THE implantable cardioverter-defibrillator (ICD) can effectively prevent sudden cardiac death in high-risk patients,¹⁻²⁾ while shock treatments have the potential to induce or worsen cardiac arrhythmias.³⁻⁶⁾ Although acceleration of VT or degeneration to VF is sometimes observed in low-energy cardioversion, in the present case, both low and high energy cardioversion induced accelerated VT. These phenomena were considered to be associated with the enhancement of sympathetic discharge.

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CASE REPORT

A 72-year old male with an old myocardial infarction had an out-of-hospital cardiac arrest. He was hospitalized at another hospital, where electrocardiography (ECG) revealed ventricular fibrillation (VF) and monomorphic sustained VT with two different types of QRS morphology. Class I antiarrhythmic drugs failed to prevent recurrence of the VT and the patient was referred to our hospital for further treatment of his ventricular tachyarrhythmias.

On admission, chest X-rays showed mild cardiomegaly but no pulmonary congestion. Two-dimensional echocardiography revealed dilatation of the left ventricle (LV). Antero-septal wall motion of the LV was aneurysmal and the LV ejection fraction was 22%. After obtaining written informed consent, electrophysiological (EP) study was performed using the standard technique.⁷⁾ Two non-clinical rapid monomorphic VTs and VF were induced by programmed stimulation and DC shocks were needed for termination. Consequently ICD therapy was indicated for this patient.

In March 1998, a transvenous ICD system (Medtronic Micro Jewel II, 7223Cx, Minneapolis, MN, USA) was implanted. During the operation, shocks at 20J were twice confirmed capable of terminating VF, and a shock of 30 J was set as the first treatment of VF. Two weeks after the operation, we attempted an EP study and reconfirmed that pacing induced-VT was terminated by 3J

Figure 1. EGM recordings during clinical VT. **A:** The recorded EGM shows VT at a cycle length of 410 msec followed by low energy (5 J) CV, resulting in acceleration of VT. **B:** Subsequent delivery of high-energy (30 J) CV changed the morphology and slightly accelerated VT. EGM = intracardiac electrogram; CV = cardioversion.

Figure 2. EGM recordings during electrophysiological study. Cardioversion at 5 and 30 J successfully terminated VT after administration of β -blocking agent.

cardioversion and T wave shock induced VF was terminated by 20J DC shocks. Antitachycardia ramp pacing was also effective to terminate the VT. Accordingly, a shock of 30J was set as the first treatment of VF, and ramp pacing and cardioversions were selected for the VT therapy. Prior to discharge, the patient underwent an exercise stress test. As this resulted in sustained VT, he was administered metoprolol. Eight months later, the patient experienced six discrete shocks while riding a bicycle. Reevaluation by conventional techniques showed that neither congestive heart failure nor acute coronary insufficiency were responsible for the episodes of VT, however, the patient had discontinued taking metoprolol for two days.

Interrogation of the device revealed normal pacing parameters, with a pacing impedance of 540 Ohms and a sensed R wave of 11.2 mV. Stored electrograms displayed monomorphic ventricular tachycardia at a mean cycle length of 410 msec. Antitachycardia ramp pacing could not terminate the VT, and cardioversion at 5 J induced accelerated VT (Figure 1A). Though subsequent therapy of defibrillation at 30 J could not terminate VT, the VT rate gradually decreased. Subsequent therapy of cardioversions at 30J also induced accelerated VT with different morphology (Figure 1B). Although ICD could not resume the VT to sinus rhythm, it was terminated spontaneously. After admission, we performed an EP study with the patient in a mild sedated state to confirm the effect of ICD. Cardioversion at both 5 J and 30 J terminated the pacing-induced VT, and VF was successfully defibrillated by a shock of 20 J with oral metoprolol

60 mg/day (Figure 2A and B). An exercise test on treadmill was also performed to simulate the clinical situation. At the second stage of the Bruce protocol, monomorphic VT was induced and cardioversion at 20 J terminated the VT. Thus, the energy of the first shock was set at 20J which had terminated VT without acceleration of the VT.

DISCUSSION

In the present case, both high and low energy cardioversion accelerated VT after discontinuation of the β -blocking agent metoprolol. Cardioversion could not terminate VT under these conditions. Although shock-related acceleration of VT or degeneration to VF sometimes happen, the mechanisms remain unclear. The risk of VT acceleration increases when VT occurs at a heart rate greater than 180 bpm. Acceleration also appears to be more frequent in patients with highly depressed left ventricular function or higher cardioversion thresholds. This increased susceptibility to proarrhythmia during faster rhythm may be related to one or more of the accompanying factors; a concomitant increase in sympathetic discharge, a rate-related decrease in ventricular refractoriness or provocation of myocardial ischemia.⁴⁾ In addition, the effect of cardioversion appears to be blunted by prolonged tachycardia duration.⁸⁾ All of these factors are associated with an increased vulnerability to arrhythmogenesis. In this case, reduced myocardial function and the increase in sympathetic discharge would be associated with the proarrhythmic effect of cardioversion. However, under administration of the β -blocking agent, neither low nor high energy cardioversion caused the acceleration of VT. Moreover, the VT induced by the exercise stress test was not accelerated by cardioversion.

Based on the present results, administration of a β -blocking agent would most likely prevent the cardioversion-related acceleration of VT in patients with low cardiac function and faster VT rates. In these patients, high energy cardioversion should be selected as the initial therapy for VT since the prolonged duration of tachycardia may influence the efficacy of ICD.

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