**Repetitive shock therapy of subcutaneous implantable cardioverter defibrillators in a patient with idiopathic ventricular fibrillation: What is the mechanism?**

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**Introduction**: The efficacy and safety of the subcutaneous ICD (S-ICD) has been demonstrated sudden cardiac death. However, the presence of inappropriate shocks observed.

**Methods**: N/A.

**Result**: A 45-year-old man was admitted to our hospital due to repetitive shock therapies from a S-ICD without syncope during the daytime. When he was 35-years-old, he was resuscitated from ventricular fibrillation (VF) during sleep. He was diagnosed with idiopathic VF (J wave syndrome), and a dual chamber transvenous ICD was implanted in another hospital. When he was 44-years-old, he received repetitive inappropriate ICD shocks due to lead dysfunction, and therefore a S-ICD was implanted. Since he received several appropriate ICD shock therapies at midnight, cilostazol was prescribed to suppress the VF episodes. All previous VF episodes had been successfully terminated by a single ICD shock. As shown in Fig A, repetitive ICD shock therapies were required to terminate the tachyarrhythmia. What was the mechanism of the arrhythmia and what should we do next?

**Conclusion**: As shown in the S-ICD tracing (Fig A), the tachyarrhythmia exhibited a regular tachycardia with a cycle length of 240 ms and the QRS complex did not seem to be relatively wide, which differed from VF episodes. In addition, the polarity of the QRS complex differed during sinus rhythm and the tachycardia. The additional important differences from the previous appropriate shock episodes was 1) the VF was always terminated by a single ICD shock, while the tachyarrhythmia required multiple ICD shocks to terminate it, and 2) VF always occurred at midnight, while the tachyarrhythmia occurred in the daytime without syncope. The possible diagnoses seemed to be 1) supraventricular tachycardia with aberrant conduction, and 2) ventricular tachycardia. We performed an electrophysiological study to clarify the mechanism of the tachycardia. Atrial pacing easily induced a regular tachycardia with right bundle branch block and a superior axis (Fig B). The S-ICD tracing during the electrophysiological study showed that the tracing of the induced tachycardia (Fig C) was exactly the same as that of the clinically observed tachyarrhythmia. The tachycardia exhibited atrio-ventricular dissociation and was easily induced by pacing from both the atrium and ventricle. We diagnosed it as a verapamil-sensitive idiopathic left ventricular tachycardia (ILVT) because 2.5 mg of verapamil prolonged the tachycardia cycle length. Ablation at the mid septum of the left ventricle where a Purkinje potential was recorded immediately terminated and eliminated the tachycardia. Since the ILVT did not lead to a hemodynamic breakdown, this shock therapy from the S-ICD was an inappropriate delivery. To the best of our knowledge, this is the first case in whom verapamil-sensitive ILVT coexisted with idiopathic VF.