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Proarrhythmic Effect of ICD Function

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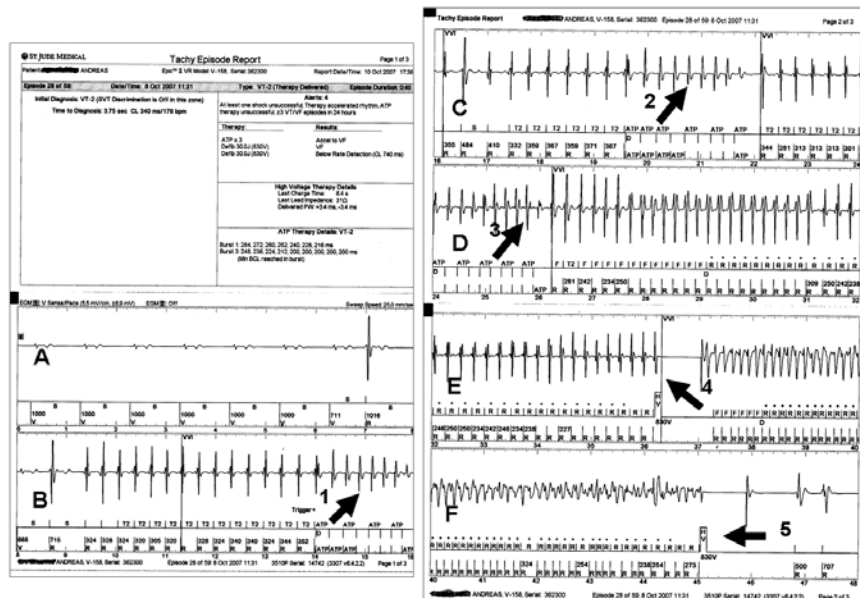
The implantable cardioverter – defibrillator (ICD) is a device that treats ventricular tachyarrhythmias (VT), when they appear in a sustained form. The device’s programming can deliver the following therapies: 1) antitachycardia pacing (ATP), 2) cardioversion and 3) defibrillation. Today’s devices also have the capability of every mode of cardiac pacing, i.e. atrial, ventricular, atrio-ventricular and biventricular pacing.

It is well known that the antiarrhythmic drug therapy can exhibit proarrhythmic effects. Likewise, the antiarrhythmic apparatus can possibly aggravate an existing VT or cause the appearance of a new arrhythmia, attempting to convert the clinical tachyarrhythmia.

A case of proarrhythmic effect related to the therapeutic sequences delivered by an ICD, is delineated in the following continuous recording of an arrhythmic event, as it was stored in the Holter function of the device.

It is about a patient with ischemic cardiomyopathy, low ejection fraction (LVEF=30%), sustained VT recorded on ambulatory monitoring, in whom a single chamber ICD was implanted.

On the upper left quadrant there is the arrhythmia episode report, including the treatment details of the VT detected by the device. The rest of the slide (panels A to F) represents the continuous tracing of the arrhythmic event, recalled from the device’s memory. Each panel consists of the endocardiac ventricular electrogram (EGM) and



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the detected cardiac rhythm cycle length (in ms).

On panel A, the basic paced rhythm (60 bpm) and a premature ventricular contraction (PVC) is recorded. On panel B, it is a PVC that induces a sustained VT at a rate of 185 bpm. The first ATP scheme is released (arrow 1) with no effect. On panel C, the ICD attempts to terminate the VT by a second ATP sequence (arrow 2), resulting in a slight acceleration of the ventricular rate (191 bpm). The third ATP scheme (panel D, arrow 3) is followed by a significant acceleration of the tachycardia rate (240 – 250 bpm), which caused the device to defibrillate (panel E, arrow 4). Consecutively, the VT degenerated into ventricular fibrillation (panel F) and finally, a second shock of 30J (arrow 5) restored the regular rhythm.

In the former example, the ICD caused worsening of the existing VT, before it succeeded in terminating it, in the end. This demonstrates a proarrhythmic effect of the antiarrhythmic device.

Arrhythmogenic effects of the delivered therapies in patients with implanted defibrillating systems have been

recorded at a rate of 5 %. Directly related to the appearance of proarrhythmic effect are in general the following factors: 1) high frequency of clinical VT, 2) rate of aggressiveness of the preprogrammed ATP protocol, 3) degree of myocardial dysfunction, 4) existence of myocardial ischemia, 5) coexistence of electrolyte disorders^{1,2,3}.

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