

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}.

REFERENCES

- 1 Fischer JD, Kim SG, Matos JA, et al. Comparative effectiveness of pacing techniques for termination of well-tolerated sustained ventricular tachycardia. *PACE* 1983;6:915-920.
- 2 Fischer JD, Johnston DR, Furman S, et al. Long-term efficacy of antitachycardia pacing for supraventricular and ventricular tachycardias. *Am J Cardiol* 1987;60:1311-1316,
- 3 Kappenberger L, Bufalo A, Fromer M. Termination and acceleration of ventricular tachycardia during antitachycardia pacing, in Luderitz B, Saksena S (eds), *Interventional Electrophysiology*, Futura, 1991, p.p. 225-231.