Efficacy of Implantable Cardioverter-Defibrillators in Treating Slow Ventricular Tachycardia

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Summary

Patients who survive a myocardial infarction frequently develop monomorphic slow ventricular tachycardia (VT). This study aimed to evaluate such arrhythmias and their therapy with implantable cardioverter-defibrillators (ICD) in our patient group with slow VTs. Implantable cardioverter defibrillators were implanted in 112 cases during the duration of the study. Of these patients, 14 met the requirements for having slow, hemodynamically non-relevant (< 150 bpm) VTs. Their mean age was 62 ± 12 years, the underlying heart diseases were coronary artery disease in 13 cases, combined with ventricular aneurysm in 4 cases, and hypertrophic cardiomyopathy in 1 instance. The mean ejection fraction was 34 ± 11%; the patients were classified as NYHA III in 10 and NYHA II in 4 cases. Dual-chamber ICDs were implanted in 5 cases and single-chamber ICDs, in 9 patients. The mean follow-up duration was 16 months. In 7 patients, the indication for ICD implantation was solely slow VT. In these cases, preoperatively tested antitachycardia pacing (ATP) was always effective. With spontaneous VTs, the efficacy was 99% (208/210). The remaining 7 patients had hemodynamically relevant fast VT or ventricular fibrillation (VF) as well as slow VT. In these patients, ATP was effective in only 70%. Because slow VTs were observed quite frequently in this patient group, cardioversion was activated only temporarily or not at all. Acceleration of slow VT after ATP was observed in 3 patients, in these cases ATP had to be switched off. In conclusion, we regard ICD implantation to be indicated in a patient with slow VT only if the efficacy of ATP is nearly 100%. If the patient has fast VT or VF, the electrical treatment of slow VTs should be judged individually.

Key Words

Monomorphic slow ventricular tachycardia, ICD therapy, antitachycardia pacing

Introduction

Implantable cardioverter-defibrillators (ICD) were originally developed and are most widely used for the treatment of patients with malignant ventricular arrhythmias and survivors of cardiac arrest. Nowadays, ICDs are able to prevent sudden cardiac death and to treat sustained ventricular tachycardia (VT) in high-risk patients. Implantable cardioverter-defibrillator therapy emerged as the primary nonpharmacologic option for many patients who are at continuing high risk for fatal arrhythmias. Clinical studies have recorded major improvements in implant risk, system longevity, symptoms associated with arrhythmia recurrences, quality of life, and diagnosis and management of inappropriate device therapy.

Monomorphic slow VT occurs quite often in patients who have survived a myocardial infarction. Radiofrequency ablation and arrhythmia surgery are applicable to a select population of patients with either malignant-hemodynamically relevant-or slow-hemodynamically stable-VTs if the VT are reproducibly inducible and of monomorphic origin, making them suitable for cardiac mapping. The development of radiofrequency catheter ablation techniques might make this method an important therapeutic alternative for these patient groups.

Based on the ACC/AHA ICD Guidelines from 1998, ICD therapy should currently be the first choice in treating sustained VTs (both hemodynamically relevant and stable) [1].

The aim of this study was to evaluate the efficacy of ICD therapy for arrhythmia therapy in our slow VT patients.

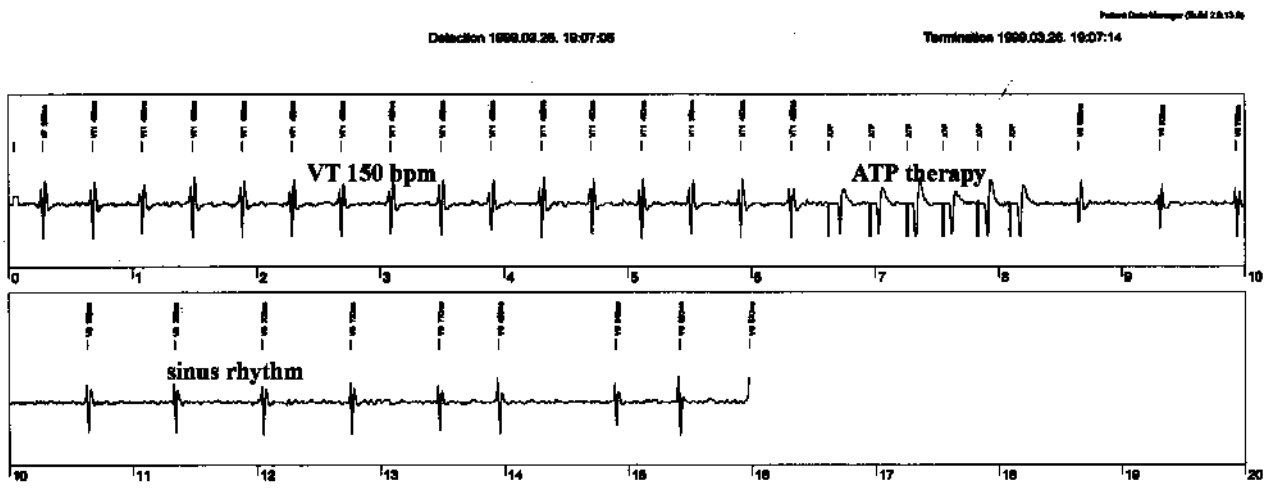

Figure 1. Effective termination of a hemodynamically stable VT (150 bpm) with a RAMP ATP therapy in a Phylax XM patient.

Material and Methods

Implantable cardioverter-defibrillators were implanted in 112 cases between 1995 and 1999. Of these cases, 14 patients met the requirement of having hemodynamically stable (< 150 bpm) VTs. Their mean age was 62 ± 12 years, the underlying heart diseases were coronary artery disease (CAD) in 13 cases, CAD combined with ventricular aneurysm in 4 cases, and hypertrophic cardiomyopathy in 1 instance. The mean ejection fraction was 34 ± 11%. The patients were classified as NYHA III in 10 and NYHA II in 4 cases. Postoperative antiarrhythmic medication of the patients was amiodarone in 5 cases, amiodarone and beta blockers in 4 cases, and d,l sotalol or propafenone in 1 case each. Three patients did not receive any antiarrhythmic medication.

The indication for ICD implantation was exclusively slow VT in 7 cases. In the remaining 7 cases, it was hemodynamically relevant fast VT or ventricular fibrillation (VF), while slow VT also occurred in these patients. In 5 instances, dual-chamber ICDs (Phylax AV, BIOTRONIK), and in 9 cases, single-chamber ICDs (1 Phylax 03, 2 Phylax 06, 4 Phylax XM, 2 MicroPhylax, all BIOTRONIK) were implanted. The mean follow-up duration was 16 months.

Results

In the patients with exclusively slow VTs, the preoperatively tested ATP therapy was always effective, and in cases of spontaneous VTs, the efficacy was 99% (208/210). Figure 1 shows an example for a successfully terminated slow VT. In the patients with hemodynamically relevant fast VT or VF, ATP was effective in only 70%. Because slow VTs were observed quite frequently in this patient group, cardioversion was set only temporarily or not at all. Acceleration of a slow VT after ATP was observed in 3 patients, in these cases the ATP therapy was turned off (Figure 2). After switching off ATP, slow VTs terminated spontaneously in all cases without acceleration or any other complications.

Discussion and Conclusion

While the 1991 ACC/AHA guidelines did not render hemodynamically stable slow ventricular arrhythmia as a class I indication for ICD implantation, sustained slow VT is now regarded a class I indication according to the 1998 ACC/AHA guidelines for ICD implantation [1,2].

Based on our experience, ICD implantation in those patients with slow VT who do not have fatal arrhythmias is indicated and acceptable only if the efficacy of ATP is nearly 100%. If such efficacy cannot be achieved, ineffective ATP therapy and the possible acceleration of arrhythmias, necessitating subsequent cardioversion or defibrillation, are not desirable because the slow VTs are well tolerated, and the shocks have serious psychological effects on the patients. Therefore, if the patient has fast VT or VF, and ATP therapy can accelerate the arrhythmias, the treatment of slow VTs should be judged individually.

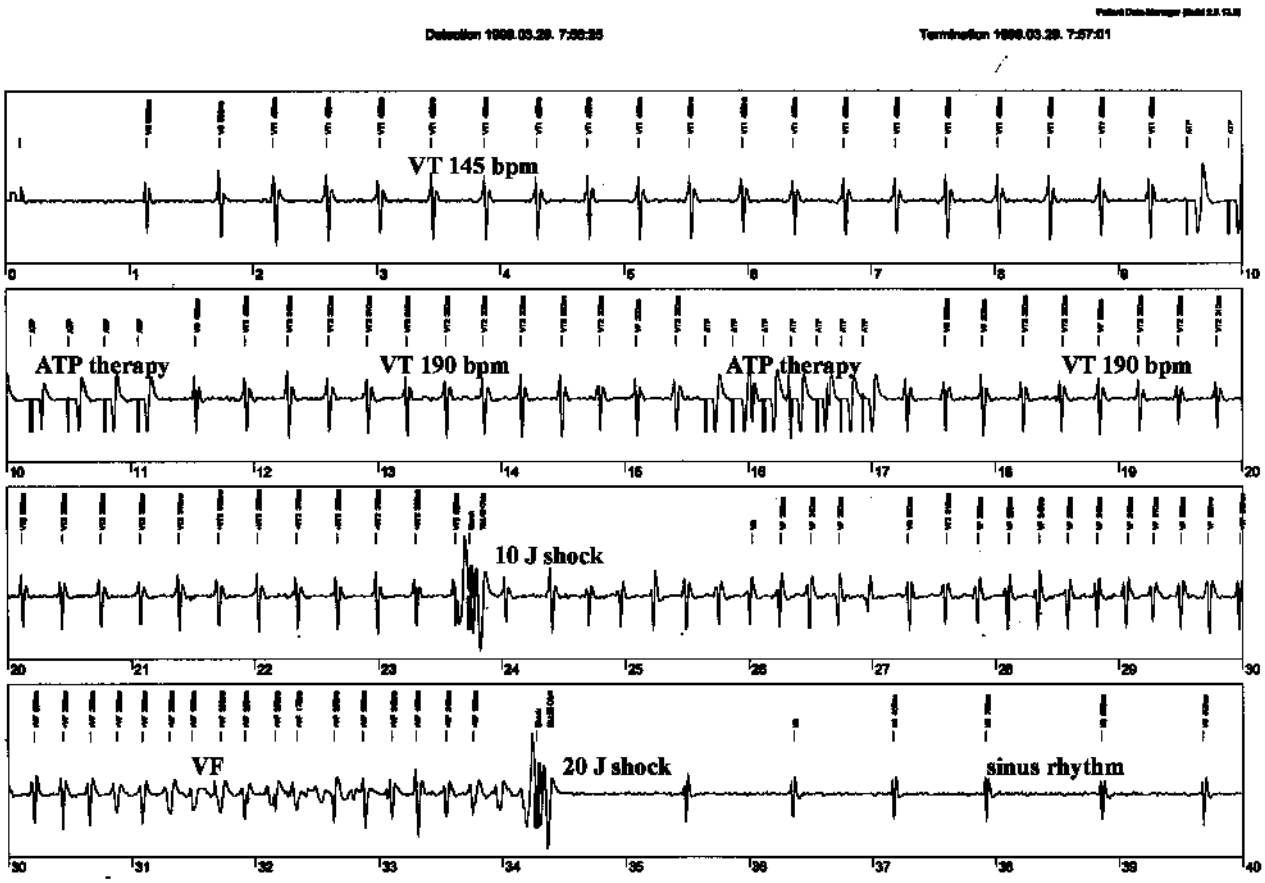

Figure 2. A slow VT (145 bpm) was accelerated by the first RAMP ATP therapy. The next ATP attempt did not treat the accelerated (190 bpm) VT successfully. The following 10-J cardioversion accelerated the VT again, and VF developed. The VF was then successfully treated by a 20-J DC shock.

References

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