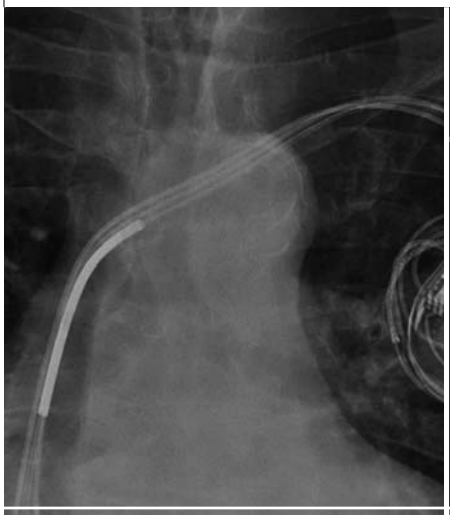

ICD Therapy—ATP

CHAPTER 14

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Introduction

Implantable cardioverter defibrillator (ICD) therapy has been shown to be effective in reducing mortality in both secondary prevention and primary prevention of sudden cardiac death.[1,2] This reduction of mortality by defibrillator shock therapy does, however, come at a cost. Defibrillator shocks can have detrimental effects on the mental and physical well-being of patients leading to a decreased quality of life.[3] ICD shocks may trigger maladaptive psychocognitive responses that can increase the incidence of depression and anxiety.[4,5] In addition, the pain associated with a shock can be significant.[6,7] Rarely, some shocks may also be proarrhythmic.[8–10] Appropriate and inappropriate shocks as compared to no shock at all was associated with a significant increase in subsequent risk of death from all causes in both the Multicenter Automatic Defibrillator Implantation Trial II (MADIT II) and Sudden Cardiac Death in Heart Failure Trial (SCD-HeFT) populations,[11,12] although a causal relationship has not yet been proven. These factors, along with the significant cost of shocks to battery drain and device longevity, would indicate that the reduction of shocks, both appropriate and inappropriate is an important goal.

The current overall shock reduction strategies are antitachycardia pacing (ATP), prolonged time to detection, and the avoidance of inappropriate shocks with reduced oversensing from T wave or electronic noise and improved supraventricular tachycardia discrimination, which is discussed in depth in chapter 16. In this chapter, the evidence for the safety of ATP in general and its mechanisms of action are reviewed along with the success of ATP as a therapy for ventricular tachycardia. The clinical factors that affect the efficacy of ATP are discussed as well as the evidence supporting

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increased time to detection for shock reduction. Finally the two commonest ATP pacing schemes are reviewed with a brief primer on the programmable ATP variables in modern ICDs.

Safety of ATP

In the past, there have been concerns that complicating the ICD with the therapeutic option of ATP and replacing shock as the first therapy with ATP may increase the failure rate by acceleration of the tachycardia (Fig 14.1) and also increasing delay to successful therapy. If true, this effect should be detected by measuring the rate of sudden death, syncope, or near syncope.

Prior to the Pacing Fast Ventricular Tachycardia Reduces Shock Therapies (PainFREE Rx) trials,[13,14] the data on ATP safety was limited. The data came largely from the Bilitch Registry, a voluntary registry of implanted ICDs and pacemakers. The Bilitch Registry measured the effect on mortality by comparing 1553 shock-only ICDs to 550 ATP-capable ICDs. At 2 years' follow-up, the total mortality in the shock group was 89% but was improved in the ATP group to 94%, $p<0.05$.[15] Although the two groups differed in the ATP capability of their ICDs, improved survival in the ATP-capable ICD group may have been secondary to other factors such as broadly improved cardiovascular and other health care. Nonetheless, at the very least, it did

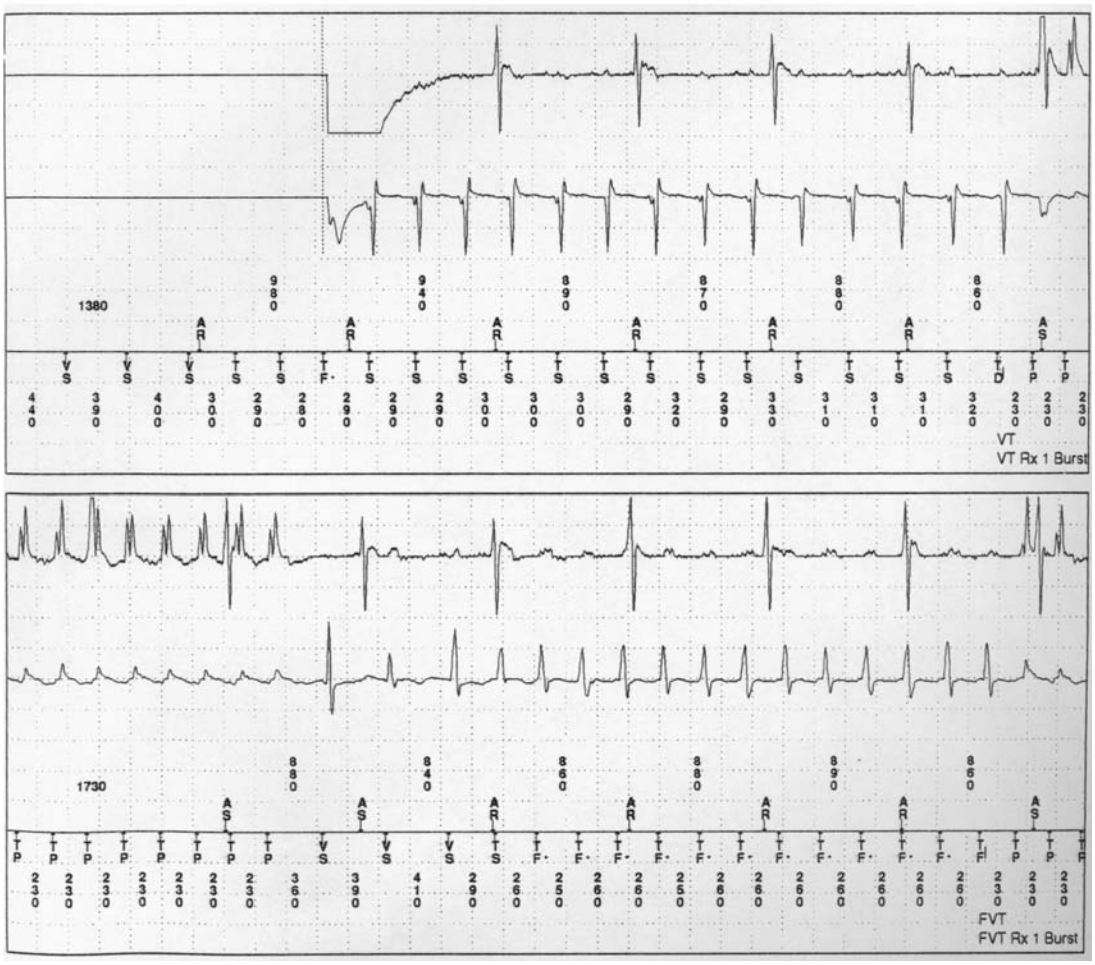

Fig 14.1 Example of burst ATP (annotated by TP on the EGM marker channel) accelerating a VT from a cycle length of 290 to 330 ms to 230 to 260 ms.

not appear that ATP-capable ICDs worsened mortality.

Subsequent nonrandomized studies in the 1990s that assessed the outcomes of ICD patients in ATP therapy trials showed mortality rates in patients with ATP as first therapy to be consistent with prior ICD trials, thus not implicating failed ATP therapy as a contributor to death. The PainFREE Rx II study randomized ICD patients to ATP versus shock. This trial carefully captured and adjudicated all causes of death. There were 2 sudden deaths in each of the shock and ATP arms among the 634 enrolled patients who were followed for a year, again showing that ATP ICDs did not appear to worsen mortality.[14]

In addition, a composite analysis of the Pain FREE Rx, Pain FREE Rx II, and Proven Shock Reduction for Primary Prevention Patients (PREPARE) trials found that in comparison to patients without ventricular tachycardia (VT) or ventricular fibrillation (VF) events, patients with episodes treated successfully with ATP had no reduced survival whereas patients treated with shock had reduced survival (relative risk 1.2, $p<0.001$).[16]

In summary, there is no trial evidence of worsened mortality with ATP. More data will be necessary to fully answer this question.

Mechanism of ATP Efficacy

Most VTs appear to originate within relatively small areas of diseased myocardium and are thought to be largely due to reentry. Although single capture tachycardia termination using critically timed premature extrasystoles falling in the "termination zone" of the tachycardia cycle length can be effective, particularly with slower tachycardias, multiple captures using burst pacing has proven to be more effective.[17–19] Burst pacing from a site not necessarily in the circuit requires distant capture and conduction into the circuit. Essentially, the success of ATP depends on the pacing stimulus penetrating an "excitable gap" in the circuit, and colliding both antidromically with the "head" of the VT wavefront and orthodromically with the VT "tail" at a point of absolute refractoriness, thus extinguishing the tachycardia[20,21] It stands to reason that the faster the reentry circuit, the shorter the time period the critical tissue is available for capture from extraneous pacing. Thus, it has been observed that slower tachycardias are more amenable to pace termination, as supported by evidence from the electrophysiology laboratory.[19,22,23] Not surprisingly there has been an initial reticence to apply ATP to faster VTs and the initial studies to demonstrate clinical efficacy were in slower, presumably hemodynamically tolerated VTs.

ATP Success

Prior studies have demonstrated that ATP was able to successfully treat spontaneous "slow" VTs with success rates of 89% to 94%.[24–28] These studies were limited in that they did not utilize a specific ATP regimen and ATP therapies involving both burst and ramp pacing were evaluated. In addition, the slow VT was variably defined by the investigators from 188 bpm (and even less in some devices) up to 220 bpm. Despite this variation in programmed parameters, ATP seemed to work for slow VT with a low failure rate of 6% to 11%. The rates of VT acceleration by ATP were between 2.0% to 3.7%[29] (Fig 14.2).

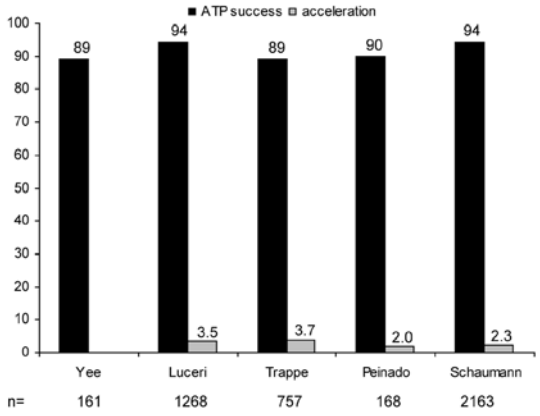

Fig 14.2 Antitachycardia pacing success for "slow" VT < 220 bpm. Abbreviation: n, number "slow" VT episodes. (Modified from Fig 1, Wathen M.[29] Reproduced with permission from Elsevier.)