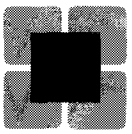

Pacemaker and defibrillator combination therapy for recurrent ventricular tachycardia

MARTIN MASTERSON, MB, BCH; SERGIO L. PINSKI, MD; BRUCE WILKOFF, MD; TONY W. SIMMONS, MD; VICTOR A. MORANT, MD; LEONARD R. GOLDING, MB, BS; LON W. CASTLE, MD; JAMES D. MALONEY, MD

■ The judicious use of antitachycardia pacemakers can enhance the benefits of automatic implantable cardioverter defibrillators in certain patients. Both devices were implanted in 10 highly selected patients with drug-refractory pace-terminable sustained ventricular tachycardia. During the observation period of 8 ± 4.8 months, the automatic pacemakers detected 1,542 episodes of ventricular tachycardia and appropriately managed 1,373. The automatic implantable defibrillator was activated at least once in every patient; on two documented occasions, the defibrillator discharged because the automatic pacemaker accelerated the tachycardia. Concomitant antiarrhythmic drugs could be reduced but not withdrawn. With meticulous device programming and testing, the two systems in combination can work synergistically to manage malignant ventricular arrhythmias in carefully selected patients.

□ INDEX TERMS: CARDIAC PACING, ARTIFICIAL; TACHYCARDIA □ CLEVE CLIN J MED 1990; 57:330–338

THE PROGNOSIS is guarded for patients with atherosclerotic heart disease, left ventricular dysfunction, and monomorphic sustained ventricular tachycardia (VT_S) that is refractory to medical therapy. The 1-year mortality rate is 28%; the 1-year sudden death incidence is 17%.[1]

The automatic implantable cardioverter defibrillator (AICD) reduces the yearly incidence of sudden cardiac death in this patient population to approximately 2%, and total mortality at 5 years is about 30%.[2–6] But this modality has limitations. Current technology precludes satisfactory results for patients at risk of sudden death who have variable-rate or frequent VT_S or both. Even with newer programmable devices, an overlap between normal sinus and slow VT_S rates is unavoidable. Moreover, patient discomfort and the likelihood of early battery depletion limits the frequency of the AICD discharges.

Until recently, antitachycardia pacing has been considered a therapeutic alternative for patients with drug-refractory VT_S.[7,8] The major drawback of antitachycardia pacing is VT_S acceleration, which occurred in 43% of patients in one series.[7]

Automatic antitachycardia pacemakers (ATPs) have had short-term benefits in a few carefully selected patients with drug-refractory VT_S,[9–17] but the long-term benefit of this therapy in the absence of defibrillation backup is questionable[9,18,19]. The use of the two devices in combination is one way to overcome these limitations.[20,21]

Beginning in August 1986, we implanted independent automatic ATPs in combination with AICDs in carefully selected patients. The selected patients had both slow and rapid VT_S or ventricular fibrillation that

From the Department of Cardiology, The Cleveland Clinic Foundation.

Address reprint requests to J.D.M., Department of Cardiology, The Cleveland Clinic Foundation, One Clinic Center, 9500 Euclid Avenue, Cleveland, Ohio 44195.

TABLE 1
PROTOCOL FOR DEVICE INTERACTION TESTING

1. Programming pacemakers in bipolar mode.
2. Reconfirming the optimal pacing protocol for VT termination.
3. Ruling out double-counting.
4. Selecting algorithm for rapid VT sensing and termination without AICD triggering.
5. Testing of both devices together during induced VT.
6. Ruling out of AICD inhibition by pacemaker pulses during induced ventricular fibrillation.

was refractory to drug therapy and were not considered suitable candidates for surgical or catheter ablation of their VT_S.

This report is a descriptive analysis of our clinical investigation. Our objectives were to: (1) evaluate methods for testing device compatibility; (2) determine whether the ATP could terminate both slow and rapid VT_S, thus reducing the requirement for AICD discharges; (3) determine whether combined device therapy in patients previously considered poor candidates for the AICD could eliminate or significantly reduce the need for concomitant antiarrhythmic drug therapy; and (4) determine the efficacy and feasibility of combined device therapy to manage malignant ventricular arrhythmias.

PATIENT POPULATION

AICDs were implanted in 105 patients (mean age, 57 years) between May 1984 and December 1987. These patients had been referred to our institution because of hypertensive VT_S or sudden cardiac death that was refractory to conventional antiarrhythmic drug therapy.

Despite continued drug therapy, 17 patients in this group had frequent, symptomatic, recurrent VT_S at a mean rate of 130 bpm, which is below the tachycardia detection rate (mean, 154 bpm) of the nonprogrammable AICD. The use of an ATP and the AICD was considered as a possible treatment option for these patients.

Ten of the 17 patients had pace-terminable VT_S and were identified as candidates for combination ATP/AICD implantation. The other seven patients, whose VT_S were not reliably pace-terminable, were excluded from consideration for combined device therapy.

Clinical characteristics of the study group

All 10 male patients (mean age 61 ± 8 years) underwent biochemical profiles, electrocardiography (ECG), multiple 24-hour ECG monitoring, coronary angiography with left ventriculography, and cardiac electrophysiologic assessment.

All patients had coronary artery disease with previous myocardial infarctions. Two-vessel disease was present in six patients and three-vessel disease in four. Six patients had had previous coronary bypass surgery and one had had previous antiarrhythmic surgery. Six patients were in New York Heart Association functional class II for heart failure and four were in class III. The mean left ventricular ejection fraction was 32.4 ± 11%.

Arrhythmia characteristics

The patients in this group had responded poorly to a mean of 7.8 different antiarrhythmic regimens. Among the 10 patients, there were 4.3 hospitalizations requiring 6.2 ± 2.6 external cardioversions for spontaneous VT.

All 10 patients had VT_S induced during cardiac electrophysiologic study while they were drug-free, and on repeat testing during medication with a variety of conventional and investigational antiarrhythmic agents; all continued to have spontaneous VT_S despite therapy. The mean VT_S rate was 195 bpm without drugs, and 130 bpm during drug therapy.

METHODS

ATPs were implanted in the 10 patients. The Orthocor II 284A (Cordis Pacing Systems, Miami, Fla) ATP, which was released in July 1985 as a clinical investigational device for the treatment of VT_S, was implanted in four patients beginning in August 1986. The Intertach ATP (Intermedics Inc., Freeport, Tex) was released for clinical investigation in August 1986 to treat VT_S and was implanted in the subsequent six patients.

Devices

The characteristics of the Automatic Implantable Cardioverter Defibrillator (AICD [Cardiac Pacemakers Inc., St. Paul, MN 55164]) have been described previously.[22]

Both ATP devices are single-chamber, multiprogrammable, and bipolar. They are capable of detecting, cataloguing, and treating tachycardias, and can also prevent bradycardia below a programmable rate.

Both pacemakers detect, as "pathologic tachycardia," any ventricular rhythm above a programmable rate (100–220 bpm, Orthocor; 94–226 bpm, Intertach) that persists for a programmable number of intervals. In the Intertach ATP, this basic detection algorithm has been made more specific with the addition of three programmable criteria: (1) suddenness of onset of tachycar-

dia; (2) rate variability, or the maximum allowed variability between R-R intervals that define a rhythm as VT, which helps to distinguish atrial fibrillation from VT; and (3) sustained high rate, which detects tachycardia that persists above the threshold rate for up to 250 programmable intervals. The third variable is used only in conjunction with the first and second and can override them both. This may reveal a persistent VT_S that does not meet the other two criteria, but it does so at the cost of decreased specificity. With this combination of criteria, nine different detection algorithms are possible.

When a tachycardia is detected, termination is attempted by the pacemaker delivering one, two, or three critically timed extrastimuli, or a burst of extrastimuli (Orthocor, up to 30; Intertach, up to 250). These are programmable either as a percentage of the tachycardia cycle length (Intertach) or at fixed intervals after the last sensed event (both Orthocor and Intertach). The pacemaker can be programmed to scan progressively shorter intervals if the first sequence does not effectively terminate the tachycardia. Successful sequences are stored in the device's memory and used first if the tachycardia recurs. The Intertach has primary and secondary response modalities that can be programmed independently.

Both devices have extensive memory capacity for

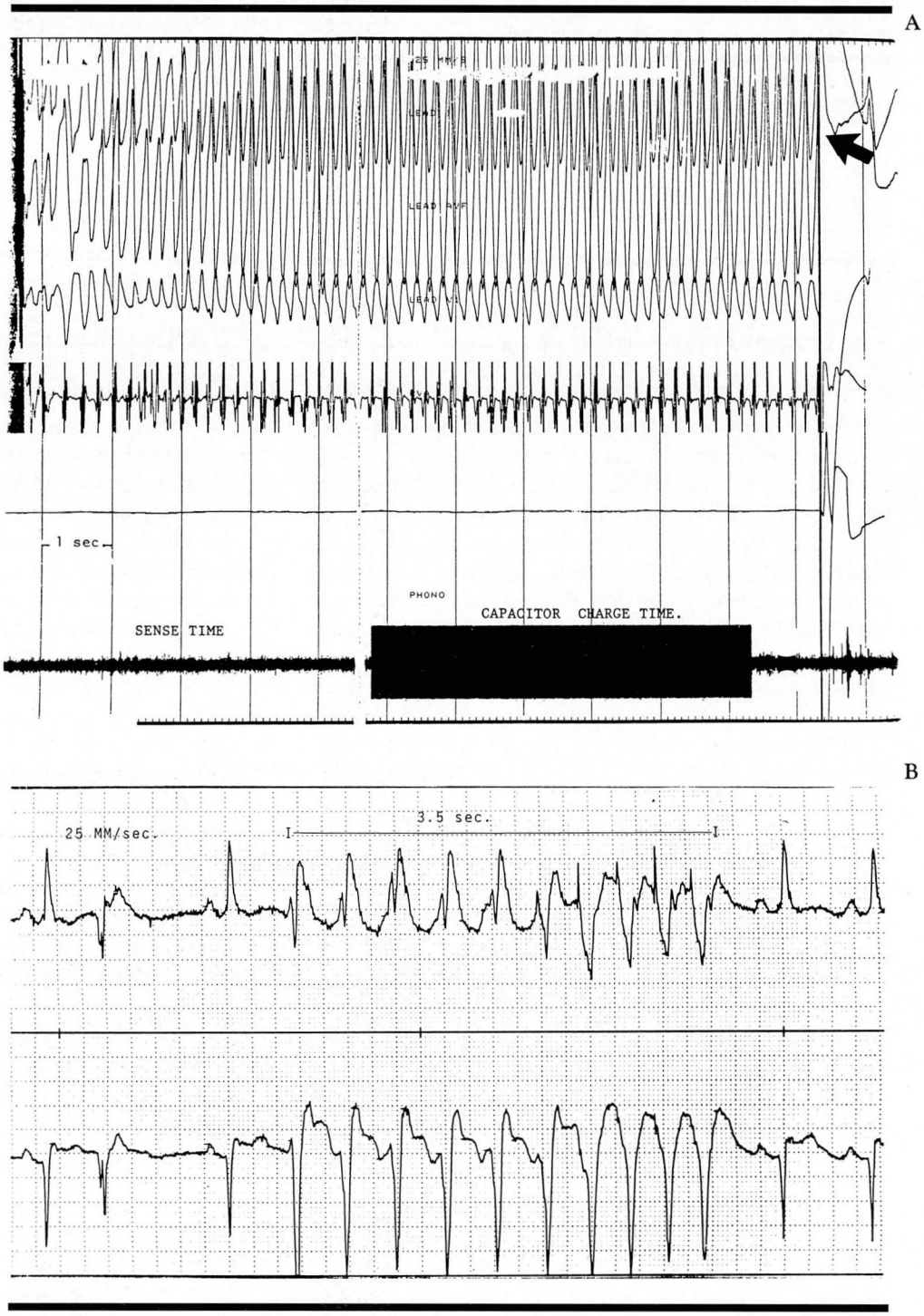

FIGURE 1. Short time frame available for successful combined device therapy. A: Induced ventricular tachycardia and simultaneous phonographic recording of the implantable cardioverter-defibrillator demonstrates the 5 to 5.5 seconds required for tachycardia recognition by the AICD (arrow, AICD discharge). B: Holter recording of spontaneous ventricular tachycardia with automatic detection and termination in less than 5 seconds by the antitachycardia pacemaker; event did not trigger the AICD.

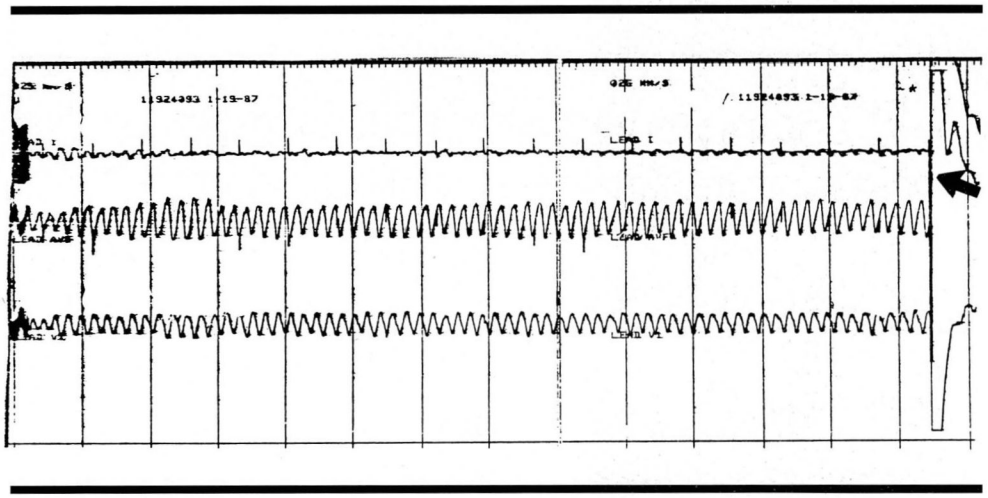

FIGURE 2. ATP and AICD testing to exclude life-threatening AICD inhibition. VOO pacing at maximum energy output during induced ventricular fibrillation (worst-case scenario) demonstrates absence of AICD inhibition.

TABLE 2
ORTHOCOR II DETECTION AND TERMINATION ALGORITHMS

Patient	1	2	3	4
Detection				
Rate (bpm)	130	110	135	135
Number of intervals	4	4	4	4
Termination				
Automatic overdrive	+	+	+	+
Overdrive constant (ms)*	40	100	40	60
Number of pulses	7	3	7	5
Ramp between pulses (ms)	-23	-23	-8	-15
Maximum pace interval (ms)	200	200	200	200
Number of attempts	Infinite	Infinite	10	15

*Overdrive constant = interval in ms less than the ventricular tachycardia cycle length detected

storage of the tachycardia rate and the number of episodes. The Orthocor also records the duration. Noninvasive cardiac electrophysiologic stimulation can be performed using implanted devices by means of telemetry interaction with an external programmed stimulator.

Surgical procedures and device compatibility testing

The AICDs were implanted using general anesthesia. The epicardial sensing leads were placed close to each other on the posterolateral left ventricle. Each patient received one small and one large internal defibrillation patch.

After surgical recovery, ATPs were implanted using local anesthesia. A bipolar endocardial lead was used in every case, placed as far as possible from the epicardial sensing leads (generally in the right ventricular apex).

A comprehensive protocol was followed in order to detect and avoid potentially deleterious device-to-device interactions *(Table 1)*. This included:

1. Programming of the ATP to the bipolar mode.
2. Reconfirmation of the optimal pacing protocol for terminating VT. This was done by placing the AICD in the stand-by "electrophysiologic mode" and then inducing the patient's VT on at least 10 occasions (range 10–16). VVT mode was frequently used during this step to help assess pacemaker sensing function.
3. To assure the absence of double counting, pacing was then initiated above the patient's intrinsic heart rate at maximum energy output for the ATP. The sensing function of the AICD was then assessed with a Beep-o-gram.[23]
4. The ATP detection and termination algorithm was then selected to ensure that the time frame encompassed for successful function would not exceed the time for tachycardia recognition by the AICD (ideally less than 5 seconds *[Figures 1A and 1B]*). The maximal heart rate response attained during previous symptom-limited exercise testing was also taken into account when selecting the tachycardia detection rate.
5. The simultaneous function of both activated devices was then assessed against induced VT. In all patients, the ATP terminated the VT on its first attempt without triggering the AICD.
6. Finally, to ensure that pacemaker pulses delivered during ventricular fibrillation would not be wrongly sensed as a normal rhythm by the AICD (due to its automatic gaining control), special testing was undertaken. The pacemakers were programmed to the VOO mode with maximum energy output and then ventricular fibrillation was induced. This "worst case scenario" did not interfere with normal AICD function, which sensed and terminated the induced fibrillation on its

TABLE 3
INTERTACH DETECTION ALGORITHM

Patient	1	2	3	4	5	6
Detection rate (bpm)	125	130	125	120	125	130
Number of intervals	5	10	15	7	5	6
Sudden onset delta (ms)	154	340	246	246	–	–
Sustained high rate	+	+	+	+	–	–
Number of intervals	100	100	50	100	–	–
Rate stability	+	+	–	+	+	+
Number of intervals	15	10	–	20	10	10
Delta (ms)	26	26	–	26	26	26

first attempt (mean 13.8 seconds) in all patients (*Figure 2*).

If deleterious interactions were detected during any of the testing steps, they were managed by repositioning the transvenous lead-electrode farther from the epicardial leads and repeating the testing sequence.

Since current AICDs are "committed devices" (once they are activated they always discharge), this assessment is critical to ensure that the ATP will detect and terminate VT_S before AICD activation, sparing the patient a needless shock.

ATP tachycardia detection algorithms are compared in *Tables 2* and 3. A composite chest/abdomen radiograph (*Figure 3*) shows both devices in place.

Patient follow-up

The 10 patients underwent monthly evaluations, including history, physical examination, check of AICD status, and telemetry of stored arrythmia data from the memory of the ATPs. Ambulatory ECG monitoring was performed every 2 months.

TABLE 4
FOLLOW-UP* COMPARISON OF ORTHOCOR AND INTERTACH ANTITACHYCARDIA PACEMAKERS

	Orthocor	Intertach
VT episodes detected	442	1100
VT episodes terminated	341	1032
VT rate (bpm, mean)	142 ± 13	140 ± 13
VT duration (s, mean)	5.3 ± 0.6	22.9 ± 15
VT episodes undetected	1 (122 bpm)	10 (107 bpm, mean)
VT episodes detected, not terminated	1	2

*Results at 7.8 ± 4.8 months' follow-up
VT = ventricular tachycardia

TABLE 5
FOLLOW-UP* RESULTS OF AICD

AICD discharges	179 (mean, 18 ± 15)
VT rate (bpm, mean)	190 ± 32
VT duration (s, mean)	22.9 ± 15
Inappropriate AICD discharges	10
VT duration	7.8 ± 0.6
VT rate (bpm)	202 ± 30
Discharges induced by accelerated VT caused by pacemaker	2

*Results at 7.8 ± 4.8 months' follow-up
VT = ventricular tachycardia

RESULTS

Mortality

Five patients died during the observation period. Three of the deaths were sudden. One patient died suddenly 5 days after implantation. ECG monitoring disclosed four episodes of ventricular fibrillation in the minutes preceding his death. Three of these were treated successfully with AICD discharges. The ATP did not induce the arrhythmias or interfere with AICD function.

Two patients died suddenly at home. In one, who died after 11 months, total AICD battery depletion was noted when the device was explanted. In retrospect, this patient had premature battery depletion. His charge time 1 month before his death suggested the need for elective generator replacement. The other, who died after 10 months, had been restarted on amiodarone because of frequent AICD discharges for rapid VT. The reinstitution of amiodarone 2 weeks before his death may have contributed to his death by increasing an already high defibrillation threshold.

Finally, one patient died from hepatitis B at 2 months after implantation, and the other died of heart failure at 11 months.

VT_S termination

Analysis of ATP memories showed that at 8 ± 4.8 months, the ATPs had detected 1,542 episodes of VT_S.

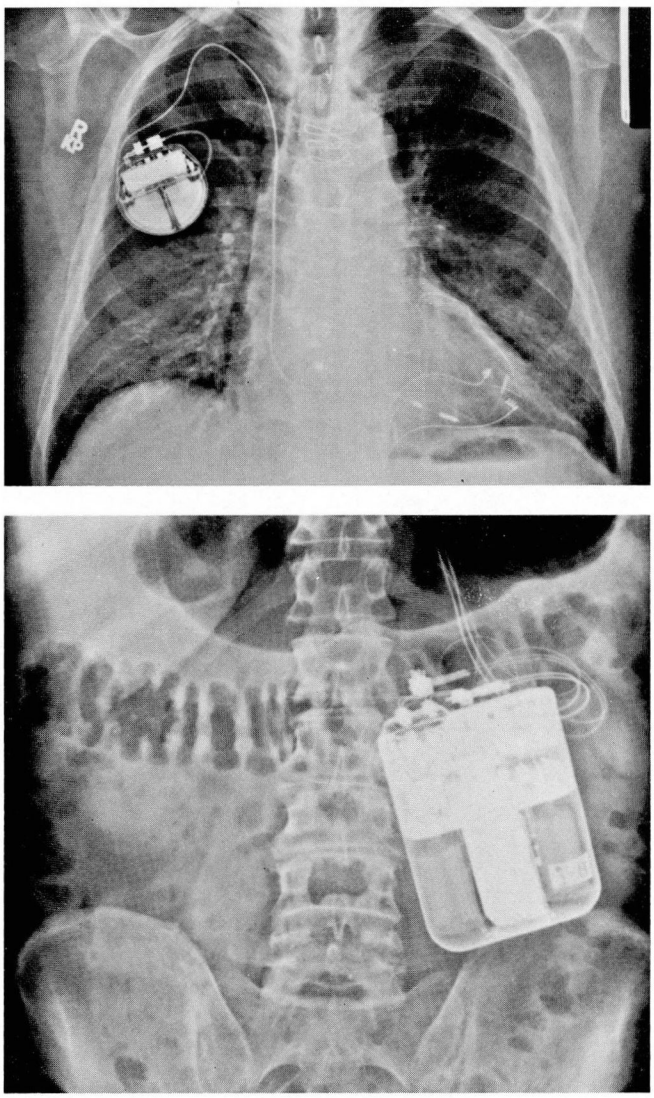

FIGURE 3. Radiographs of chest and abdomen showing placement of antitachycardia pacemaker and implantable defibrillator. Note large separation between AICD sensing electrodes in the left ventricle and the pacemaker lead in the right ventricular apex that minimizes potential for "crosstalk."

The ATP had been used a mean of 154 times by each patient, and each patient had had a mean of 18 AICD discharges (*Tables 4* and *5*).

Holter ECG documentation was available for analysis in 10 spontaneous AICD discharges and 21 ATP treatments. The 10 AICD discharges were preceded by pacemaker-delivered extrastimuli. Seven were for episodes of VT_S that persisted to the time of discharge, including two episodes of slow VT_S that were accelerated by the ATP. Two shocks were delivered for episodes of VT that terminated prior to discharge. One AICD discharge was induced when an episode of sinus tachycardia triggered the ATP. The ATP discharge, in turn, induced VT_S which was terminated by the AICD (*Figure 4*). The patient's ATP was deactivated until his sinus rate response to exercise was controlled pharmacologically.

In the other 11 documented pacemaker treatments, VT_S was expeditiously detected and terminated without triggering the AICD (*Figure 1B*).

Three patients who had toxic reactions to amiodarone (*Figure 5*) had antiarrhythmic drugs withdrawn and were treated solely with the devices. In two of these, it was necessary to restart drug therapy after a mean of 4 months to control the increased frequency of rapid VT_S requiring AICD discharges. The third patient died after 2 months from hepatitis B.

Subsequently, all patients were maintained on full antiarrhythmic drug therapy, although the combination of devices allowed low dosages and minimized the risk of drug toxicity.

Complications

During implantation of the first patient, his tined ATP lead dislodged at the time of an AICD discharge. Active fixation leads were implanted in subsequent patients.

The memory of the ATP disclosed that 10 of the AICD discharges were for episodes of VT that persisted for less than 6 to 8 seconds, but nevertheless activated the discharge of the committed AICD. Holter monitoring showed that in four patients, 11 episodes of slow VT_S remained undetected because their rates were below the programmed detection rate. This was managed by decreasing drug therapy and/or reprogramming of the detection algorithm. Three episodes of VT_S were detected but not terminated by the ATP; these patients responded then to a more aggressive termination algorithm. Two patients required pharmacologic blunting of their sinus rates with beta blockers for successful ATP function.

Three patients were intolerant to VVI pacing backup by their ATP (pacemaker syndrome). This was managed by reprogramming the lower rate or inactivating the antitachycardia backup function.

DISCUSSION

Patients with atherosclerotic heart disease, left ventricular dysfunction, and sustained monomorphic ventricular tachycardia represent a major therapeutic challenge, particularly when the tachycardia is resistant

to therapy or the patient is intolerant of antiarrhythmic drugs. Antiarrhythmic surgery with electrically guided subendocardial resection is successful in selected patients, but the operative mortality and recurrence rates for sudden cardiac death and clinical VT_S remain high.[24]

Pacing as a mode of reliably inducing and terminating VT_S has been reported since 1972.[25,26] Many variations in the pacemaker pulse train have been evaluated to find the safest and most effective mode for VT_S termination.

Pace termination of VT_S with bursts of rapid ventricular pacing[7] and self-adapting, autodecremental extrastimuli[8] have yielded the best results; however, VT_S acceleration occurred in more than 40% of patients tested in one series.[7] This observation, plus published[20,21] and unpublished long-term experience have led to a general consensus that ATP should be avoided in patients with VT_S in the absence of backup defibrillation.

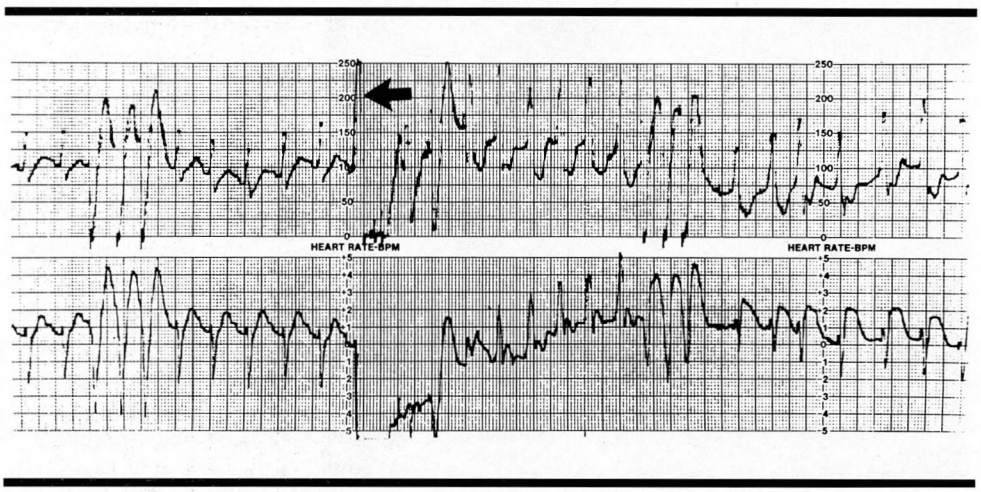

FIGURE 4. Undesirable interaction between devices and patient. Holter recording shows sinus tachycardia (143 bpm) which triggers the ATP, which in turn causes the AICD to discharge (arrow). This induces ventricular tachycardia (176 bpm) that is terminated by the ATP.

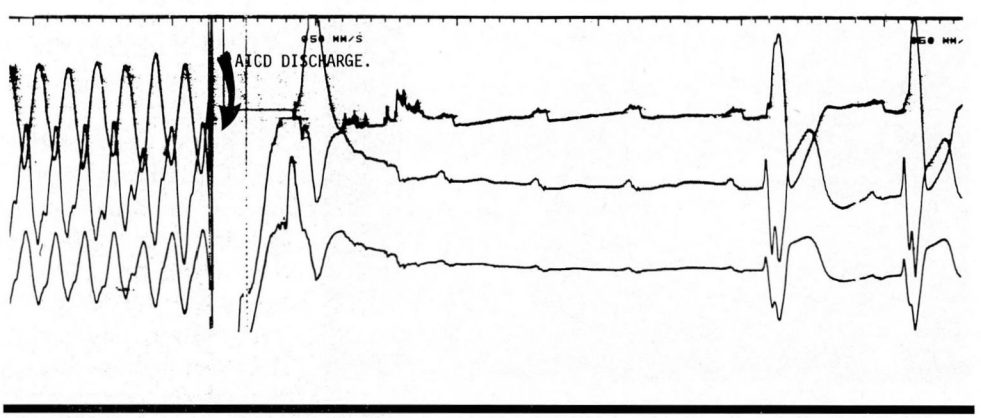

FIGURE 5. Four seconds of complete heart block following an AICD discharge (arrow), prior to activation of the ATP. Both ATP devices provide backup VVI pacing.

Evaluating device compatibility

Single- and dual-chamber pacing may interfere with normal AICD function, although with careful planning most interactions can be prevented.[20,27–29] The important considerations are that the pacing electrodes be bipolar; that the rate-counting AICD sensing electrodes be bipolar and distanced as far as possible from the pacing electrode (ideally in the other ventricular chamber); and that pacing and sensing parameters be optimized. Specific testing is essential at the time of pacemaker implantation to ensure recognition of tachycardia by the AICD during possible loss of pacemaker inhibition; for example, a pacemaker pulse during ventricular fibrillation that is sensed falsely by the AICD as QRS would preclude a lifesaving AICD shock.

The pacemaker should be programmed to its asynchronous mode at maximum energy output; then, correct AICD function must be confirmed during induced ventricular fibrillation.

In this series, ATP function, VVI pacing mode, memory, and telemetry function were not adversely affected by AICD discharges in any patient.

Current tachycardia detection algorithms are suboptimal; ideally, the tachycardia rate threshold should be

less than the slowest VT_S rate and greater than the maximum exercise-induced heart rate with a moderate safety margin. If this is impossible to achieve, then pharmacologic adjustment of the sinus rate may be necessary. For example, in one patient, sinus tachycardia triggered his ATP, which activated his AICD. He required beta blocker therapy in addition to his usual antiarrhythmic therapy to control his sinus rate.

ATP termination of rapid VT_S

As retrieved from the memory of the ATP, all patients in this series had VT_S at rates ranging from 140 bpm to 190 bpm. In the absence of complete ECG documentation, we are unable to determine how many of these episodes were spontaneous or resulted from ATP-induced VT_S acceleration, but in two episodes VT_S acceleration was ECG-documented. The clinical impression is that the ATPs do not significantly reduce the number of AICD discharges for rapid VT_S.

Pace-termination of rapid VT_S in this patient group was largely unsuccessful at implantation of the devices, suggesting that termination of spontaneous, rapid VT_S requires an AICD discharge.

The memory of the Orthocor II documented 10 AICD discharges for episodes of VT unlikely to have persisted to the time of the discharge. This observation demonstrates a limitation of the AICD: It fails to recheck if a tachycardia is still present just prior to discharge. Although ATP termination of slow VT_S is well documented, the two documented episodes of AICD discharges induced by VT_S acceleration is probably an underestimation. Further clarification of this issue will require careful scrutiny over a longer follow-up period with ECG monitoring.

Since both devices function independently, they may simultaneously attempt to terminate an episode of VT. This could cause the ATP pulse burst to induce further VT—a phenomenon that was observed in the laboratory during device testing.

Limitations

Both ATPs are single-chamber devices; if antibradycardia pacing is required, it will be in the ventricular demand mode, which will lead to atrioventricular asynchrony. This could significantly reduce cardiac output (as demonstrated by the development of the pacemaker syndrome in the three patients in this series). Although both ATPs are among the most sophisticated devices currently available, the tachycardia detection algorithm based on heart rate is a major limitation.

An improved algorithm might incorporate intracardiac electrograms and physiologic sensors.[30] Both ATPs have extensive memory capability, but they lack the ECG documentation that is needed to assess rhythms.

As discussed, the two major limitations of the 1986–1990 AICD models are the lack of programmability and, especially, the inability to check if a tachycardia has terminated once capacitor charging commences.

Feasibility of combined devices

Drug-refractory VT_S can be terminated by antitachycardia pacing without causing patient discomfort. However, even in patients with stable VT_S, automatic ATPs should be implanted only in conjunction with the AICD. VT_S stability over time is unpredictable. The AICD provides backup defibrillation for rapid or accelerated VT_S and ventricular fibrillation. Furthermore, in the few patients who develop transient bradyarrhythmias after an AICD discharge, the ATP provides pacing backup.

The combined use of an ATP and the AICD is a feasible therapeutic approach for carefully selected patients who have both slow and rapid recurrent VT_S. All patients in this series required concomitant pharmacologic therapy to reduce the frequency and rapidity of episodes. Nevertheless, the devices terminated potentially lethal arrhythmias a mean of 154 times per patient.

Antitachycardia pacing-defibrillator combination therapy may serve as a bridge to the "ideal tachyarrhythmia device," designed to provide tiered levels of therapy. These include antitachycardia discrete pulse therapy, low energy cardioversion therapy, and defibrillation backup, along with sophisticated tachycardia recognition algorithms, telemetered event recording to confirm rhythm before and after electronic therapy, and bradycardia backup.[31]

Although these ideal goals are attainable, it is unlikely that they will be available in a practical and affordable package before the 21st century. Even then, careful medical selection based on need relative to cost of the most sophisticated devices will be required. These risk/benefit cost-effectiveness issues will have even more importance in less affluent areas of the world; therefore, use of combination antitachycardia pacing devices with implantable defibrillators will likely persist for the next two decades.

Careful patient selection and detailed system analysis to avoid adverse interactions between the two devices will continue to be the key to successful use of combined device therapy.

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