A Hemodynamically Responsive Antitachycardia System
Development and Basis for Design in Humans

Todd J. Cohen, MD, and L. Bing Liem, DO

Current automatic implantable cardioverter-defibrillators detect tachyarrhythmias primarily by rate-only algorithms and cannot adequately distinguish hemodynamically stable from unstable tachyarrhythmias. The responses of right atrial (mean) and right ventricular pressures (mean, systolic, diastolic, and pulse) to 64 induced and paced supraventricular and ventricular tachyarrhythmias were studied in 10 patients (left ventricular ejection fraction of 32±6%) to develop an algorithm capable of differentiating stable from unstable rhythms. Tachyarrhythmias were defined as hemodynamically unstable when mean arterial pressure decreased by 25 mm Hg or more during 15 seconds. Mean right atrial, right ventricular systolic, and right ventricular pulse pressures were found to be useful in distinguishing the hemodynamic significance of a tachyarrhythmia. A combined detection algorithm was developed that identified a hemodynamically unstable rhythm when the heart rate was 150 beats/min or more and mean right atrial pressure increased by 4 mm Hg or more and right ventricular systolic pressure decreased by 5 mm Hg or more during 15 seconds. This algorithm was then applied to the next 20 consecutive patients (left ventricular ejection fraction of 34±4%) and compared with the current rate-only algorithm (heart rate of 150 beats/min or more) in 143 tachyarrhythmias, and the sensitivity and specificity for detection of hemodynamically unstable tachyarrhythmias were determined. The rate-only detection algorithm had 100% sensitivity but only 68% specificity for detection of unstable tachyarrhythmias, whereas the combined rate-mean right atrial pressure–right ventricular systolic pressure detection algorithm had sensitivity and specificity of 100%. Therefore, the performance of an antitachycardia system may be significantly improved by detection algorithms that integrate hemodynamic and rate criteria. (Circulation 1990;82:394–406)

Despite the emergence of newer and more potent antiarrhythmic drugs, sudden cardiac arrest from ventricular tachyarrhythmias remains a leading cause of mortality in the United States. Consequently, therapy with devices such as the automatic implantable cardioverter-defibrillator (AICD, Cardiac Pacemaker Inc., St. Paul, Minn.), which has been shown to be safe and effective, has gained significant popularity.1–9

Tachyarrhythmia detection by such devices has thus far relied entirely on electrogram analysis, which unfortunately is of limited value in determining the hemodynamic consequence or patient’s tolerance of a tachyarrhythmia.10–22 Thus, although the AICD ensures effective and rapid shock delivery to terminate a potentially fatal ventricular tachycardia or ventricular fibrillation, it also frequently delivers discomforting shocks for a variety of minimally symptomatic supraventricular tachycardias and nonsustained ventricular tachyarrhythmias. Such discharges may also contribute to premature defibrillator battery depletion.

Accurate assessment of the hemodynamic stability of a tachyarrhythmia is necessary in determining the urgency and most appropriate mode of therapy. Direct long-term measurements of cardiac output or arterial pressure would be most desirable but are not currently available. Some investigators have, therefore, studied other indirect parameters for determining the presence of hemodynamic compromise. Using implanted right ventricular (RV) pressure sensors, Olson and colleagues23 were able to detect ventricular fibrillation in dogs based on changes in RV pulse pressure (calculated from the difference between RV systolic and
diastolic pressures). Cohen and coworkers,\textsuperscript{24} using a canine pacing model, investigated the usefulness of mean right atrial (RA) and RV pressures in assessing hemodynamic stability during simulated ventricular and atrial tachycardias, and they demonstrated that these parameters were useful for assessing tachycardia stability. Based on those findings, a manually operated hemodynamically responsive antitachycardia system was successfully tested in one canine model.\textsuperscript{25} With advances in pressure-sensor technology for long-term monitoring, incorporation of these and similar parameters into an implantable antitachycardia system for humans would appear feasible.\textsuperscript{26–28}

To be suitable for incorporation into an antitachycardia system, hemodynamic parameters should change appreciably during the onset of a significant tachyarrhythmia and return toward baseline after termination. To identify such parameters, changes in RA and RV pressures were measured with arterial pressure during induced and paced tachyarrhythmias in 30 patients. Data from the first 10 patients were used to develop detection algorithms that would distinguish hemodynamically stable from unstable tachyarrhythmias. These algorithms were then applied to the next 20 patients to determine their sensitivity and specificity for detection of unstable tachyarrhythmias.

**Methods**

**Patients**

All patients undergoing clinical electrophysiological study for diagnosis or treatment of suspected ventricular tachyarrhythmias at Stanford were included in this study. All patients gave informed consent to the protocol approved by the committee for the protection of human subjects at the Stanford University Medical Center.

Thirty patients were prospectively studied. There were 24 male and six female patients, ages ranging from 15 to 78 years (55±3 years, mean±SEM), who had a mean left ventricular ejection fraction of 33±3%. Seventeen had underlying coronary artery disease and remote myocardial infarction, six had nonischemic cardiomyopathy (five dilated and one hypertrophic), one had Ebstein's anomaly with atrial septal defect, and six had no known structural heart disease. All patients were in sinus rhythm at baseline.

**Data Acquisition**

Intracardiac electrograms were recorded with standard multipolar electrode catheters positioned at the high RA, the His bundle position, and the RV apex. An additional fluid-filled catheter was used to measure simultaneous pulsatile and mean RV pressure, and the side port of an introducer sheath with the distal portion located in the RA was simultaneously used to measure pulsatile and mean RA pressures. Arterial pressure was monitored from a femoral arterial line. Pressures were recorded with Gould-Statham pressure transducers (P-23, Gould Inc., Glen Burnie, Md.) connected to an amplifier system (Honeywell Medars 200, Honeywell Medical Electronics, Pleasantville, N.Y.) for the RA and RV pressures and a pressure amplifier system (Bloom Associates Ltd., Reading, Pa.) for arterial pressure. RV pulse pressure was calculated from the systolic and diastolic measurements. All pressure signals and intracardiac and surface electrograms were recorded simultaneously on magnetic tape and on a paper recorder (Gould ES 1000) with the patient in the supine position.

**Pacing and Programmed Electrical Stimulation Protocol**

A programmed electrical stimulator (Bloom Associates) was used for pacing and for inducing ventricular tachyarrhythmias. Stimuli were 2-msec rectangular pulses delivered at twice the diastolic capture threshold. Pressure measurements were taken at baseline physiological heart rates and after 30 seconds of pacing. RA pacing, followed by RV pacing, was performed at cycle lengths 600, 500, and 400 msec with a 1-minute interval between to allow return of hemodynamic parameters and heart rate to baseline. In nine patients, simultaneous RA and RV pacing was also performed with the same protocol.

Ventricular tachyarrhythmia induction was performed in the standard manner with up to three extrastimuli from two RV sites.\textsuperscript{29,30} During a sustained ventricular tachyarrhythmia (defined as lasting at least 30 seconds or requiring termination for hemodynamic compromise), a 12-lead electrocardiogram was obtained for analysis of tachycardia morphology, and the intracardiac activation sequence was analyzed for presence of ventriculoatrial (VA) conduction. Hemodynamic responses at onset, during, and after termination of the first sustained ventricular tachycardia and ventricular fibrillation were continuously recorded. Termination was manually initiated, with overdrive ventricular pacing or cardioversion-defibrillation, and was not attempted during the first 30 seconds of tachyarrhythmia. No attempt was made to terminate the tachyarrhythmia with cough cardioversion. In five patients, two-dimensional and color Doppler echocardiograms were obtained during ventricular tachycardia.

**Statistical Analysis**

Changes in mean RA, RV (systolic, diastolic, pulse, and mean), and mean arterial pressures were analyzed with the paired and unpaired Student's \( t \) test, and statistical significance was arbitrarily defined as \( p \) less than 0.05 (two tailed). Pressures at baseline were compared with those during 30 seconds of rapid RA and RV pacing at each cycle length in all patients. To assess the effects of atrioventricular (AV) timing, we compared pressure changes in patients that had 1:1 AV conduction with changes in those that did not have 1:1 AV conduction during rapid RA pacing, and pressure changes in patients with VA conduction were compared with changes in those who had VA dissociation during rapid RV pacing. In nine patients, simultaneous RA and RV pacing was performed, and
hemodynamic responses to simultaneous RA and RV pacing were compared with those during RA and RV pacing at each cycle length.

In patients with induced ventricular tachyarrhythmias, pressures at baseline were compared with pressures at 5, 15, and 30 seconds of tachyarrhythmia, and pressures at 30 seconds of tachyarrhythmia were compared with those at 15 and 30 seconds after termination. All data are presented as mean ± SEM.

**Algorithm Design**

The hemodynamic responses of the first 10 patients were quantitatively examined and used to design combined hemodynamic and rate detection algorithms. Four algorithms were tested in the next 20 patients and compared with the rate-only algorithm (most frequently used by AICDs) for their sensitivity and specificity in detecting a hemodynamically unstable tachyarrhythmia (defined as a decrease in mean arterial pressure of 25 mm Hg or more during 15 seconds). In addition, the responses to a cough and 15 seconds of Valsalva maneuver were studied at baseline in five of the 20 patients to determine whether these events could be mistaken for an unstable tachyarrhythmia.

**Results**

**Hemodynamic Responses to Rapid Pacing**

The baseline mean cycle length before pacing was 760±30 msec. Hemodynamic responses to 30 seconds of rapid RA and RV pacing are shown in Table 1. During 30 seconds of rapid RA pacing, mean RA pressure was the only right heart parameter that increased significantly from baseline and did so only at the 400-msec cycle length by 2±0 mm Hg (p<0.0001), whereas mean arterial pressure decreased by 4±2 mm Hg (p<0.05). RV pressures (systolic, diastolic, pulse, and mean) did not change significantly.

During 30 seconds of RV pacing, increases in mean RA pressure and decreases in mean arterial pressure from baseline were significant at all cycle lengths (p<0.0001). At 30 seconds of RV pacing, mean RA pressure increased by 2±0 mm Hg from baseline at the 600-msec cycle length, by 3±0 mm Hg at the 500-msec cycle length, and by 4±0 mm Hg at the 400-msec cycle length. Mean arterial pressure decreased from baseline by 7±1 mm Hg at the 600-msec cycle length, by 7±1 mm Hg at the 500-msec cycle length, and by 16±2 mm Hg at the 400-msec cycle length. For any given cycle length, significantly greater increases in mean RA and decreases in mean arterial pressures were observed during RV compared with RA pacing (p<0.001). In addition, there was also a small, but significant, increase in mean RV pressure from baseline at the 400-msec cycle length (by 2±1 mm Hg; p<0.05). Changes in RV systolic, diastolic, and pulse pressures were not significant at any cycle lengths.

Figure 1 shows tracings of RA, mean RA, RV, mean RV, and mean arterial pressures during RA and RV pacing at the 400-msec cycle length. Note the erratic appearance of cannon “a” waves during RV pacing with VA dissociation.

**Influence of Inappropriate Atrial Timing Sequences**

During RA pacing at 400-msec cycle length, nine of 30 patients had less than 1:1 AV conduction (ventricular response rate, 730±40 msec) but no significant difference in hemodynamic responses
Cohen and Liem  Hemodynamically Responsive Antitachycardia System 397

FIGURE 1. Tracings of right atrial, mean right atrial, right ventricular, mean right ventricular, and mean arterial pressures during atrial pacing (panel A) and ventricular pacing (panel B) at the 400-msec cycle length in one patient. Right atrial pacing at the 400-msec cycle length resulted in Wenckebach atrioventricular nodal conduction, and right ventricular pacing at the same cycle length resulted in atrioventricular dissociation. Asterisks mark cannon “a” waves during atrioventricular dissociation. ECG, electrocardiogram; HRA, high right atrium (electrogram); RVA, right ventricular apex (electrogram); MAP, mean arterial pressure; RAP, right atrial pressure; MRAP, mean right atrial pressure; RVP, right ventricular pressure; MRVP, mean right ventricular pressure.

compared with those who had 1:1 AV conduction. During RV pacing, significantly greater pressure changes from baseline were observed in those with VA conduction (seven patients) than in those with VA dissociation (23 patients) but only for mean RA pressure, which increased by 4±1 mm Hg at the 600-msec cycle length and 4±0 mm Hg at the 500-msec cycle length in the VA conduction group compared with 2±0 mm Hg at the 600- and 500-msec cycle lengths in the VA dissociation group (p<0.05). Only two patients had VA conduction during RV pacing at the 400-msec cycle length.

In nine patients, simultaneous RA and RV pacing was also performed (Table 1). These patients had a mean age of 61±4 years, left ventricular ejection fraction of 35±5%, and baseline cycle length of 730±60 msec (p=NS compared with the other 21 patients). Responses were most similar to rapid RV pacing with VA conduction, with significant increases in mean RA pressure at each cycle length and significant decreases in mean arterial pressure at all except the 600-msec cycle length. Changes in RV diastolic pressure were only significant during pacing at the 400-msec cycle length, decreasing by 2±1 mm Hg (p<0.05). Changes in RV systolic, RV pulse, and mean RV pressure were not significant at any cycle length. Figure 2 demonstrates actual electrogram and pressure tracings during simultaneous RA and RV pacing at the 400-msec cycle length, showing continuous appearance of cannon “a” waves.
FIGURE 2. Tracings of right atrial, mean right atrial, right ventricular, mean right ventricular, and mean arterial pressures to simultaneous right atrial and right ventricular pacing at the 400-msec cycle length. Right atrial pressure tracing reveals the continuous appearance of cannon “a” waves. ECG, electrocardiogram; HRA, high right atrium (electrogram); RVA, right ventricular apex (electrogram); MAP, mean arterial pressure; RAP, right atrial pressure; MRAP, mean right atrial pressure; RVP, right ventricular pressure; MRVP, mean right ventricular pressure.
Hemodynamic Responses to Induced Ventricular Tachyarrhythmias

Twenty-four of the 30 patients in this study had induced ventricular tachyarrhythmias. Nineteen patients had induced ventricular tachycardia, four had induced ventricular tachycardia and ventricular fibrillation, and one had induced ventricular fibrillation only. Hemodynamic responses were determined for these 28 tachyarrhythmias (23 ventricular tachycardias and five ventricular fibrillations; cycle length, 250±10 msec) and after arrhythmia termination (cycle length, 700±30 msec) in 24 patients. Tachyarrhythmias were terminated by defibrillation in 17 episodes, by ventricular overdrive pacing in six, and spontaneously in five. Table 2 shows the mean hemodynamic responses to induced ventricular tachyarrhythmias and their termination. Figure 3 shows hemodynamic responses during and after termination of a ventricular tachycardia in one patient. During 30 seconds of induced ventricular tachycardia, mean arterial, RV systolic, and RV pulse pressures increased significantly, whereas mean RA pressure rapidly increased. Significant changes from baseline were observed for these parameters at 5, 15, and 30 seconds of tachyarrhythmia (p<0.0001). Within 30 seconds after termination, mean arterial, RV systolic, and RV pulse pressures returned and overshot their baseline values, whereas mean RA pressure returned toward but did not reach its baseline value. After termination, changes in these parameters were all significant (p<0.0001) compared with values before termination (during 30 seconds of tachyarrhythmia).

Mean arterial pressure consistently decreased from its baseline of 82±4 to 47±3 mm Hg at 5 seconds after tachyarrhythmia onset, continued to decrease to 38±3 mm Hg at 15 seconds, then rebounded slightly to 41±4 mm Hg at 30 seconds. By 30 seconds after tachyarrhythmia termination, mean arterial pressure returned and overshot its baseline value by 7±3 mm Hg.

RV systolic and RV pulse pressures consistently decreased during ventricular tachyarrhythmias, in parallel with changes in mean arterial pressure. RV systolic pressure decreased from 39±2 to 23±2 mm Hg at 5 seconds after tachyarrhythmia onset, to 24±1 mm Hg at 15 seconds, and to 27±2 mm Hg at 30 seconds. RV pulse pressure decreased from 31±3 to 14±2 mm Hg at 5, 15, and 30 seconds after tachyarrhythmia onset. By 30 seconds after tachyarrhythmia termination, RV systolic and pulse pressures returned and overshot their baseline values by 12±2 and 9±2 mm Hg, respectively.

Mean RA pressure consistently and rapidly increased at the onset of a tachyarrhythmia from a baseline value of 7±1 to 11±1 mm Hg at 5 seconds and continued to increase to 14±1 mm Hg at 15 seconds and 17±1 mm Hg at 30 seconds. After termination, mean RA pressure returned toward but did not reach baseline at 30 seconds (10±1 mm Hg).

RV diastolic and mean pressures only increased significantly at 30 seconds of tachyarrhythmia, but both failed to return to baseline by 30 seconds after termination (p=NS compared with the value at 30 seconds of induced tachyarrhythmia).

In the four patients in whom both ventricular tachycardia and ventricular fibrillation were induced, hemodynamic responses to both types of tachyarrhythmias were similar. VA conduction occurred during ventricular tachycardia in only one patient, who had qualitatively similar hemodynamic responses to the patients with VA dissociation. In five patients, two-dimensional and color Doppler echocardiograms demonstrated a mild increase in the degree of tricuspid regurgitation from baseline during induced ventricular tachycardias.

Detection Algorithms

The responses of mean RA and RV pressures (systolic, diastolic, pulse, and mean) to 64 induced and paced supraventricular and ventricular tachyarrhythmias were studied in the first 10 patients (left
Figure 3. Tracings of hemodynamic responses of mean arterial, right atrial, mean right atrial, right ventricular, and mean right ventricular pressures to ventricular tachycardia at the 220-msec cycle length. During ventricular tachycardia, mean arterial, right ventricular systolic, and right ventricular pulse pressures decreased; and mean right atrial pressure increased significantly. Mean right ventricular pressure increased only slightly. At 39 seconds after tachycardia onset, the arrhythmia was asynchronously defibrillated (200 J) back to sinus rhythm. Asterisks denote unsuccessful attempts to terminate the tachyarrhythmia by overdrive pacing. ECG, electrocardiogram; HRA, high right atrium (intracardiac electrogram); RVA, right ventricular apex (intracardiac electrogram); RVOT, right ventricular outflow tract (intracardiac electrogram); LVA, left ventricular apex (intracardiac electrogram); MAP, mean arterial pressure; RAP, right atrial pressure; MRAP, mean right atrial pressure; RVP, right ventricular pressure; MRVP, mean right ventricular pressure.
ventricular ejection fraction of 32±6%) to develop an algorithm to differentiate hemodynamically stable from unstable rhythms. In this study, the right heart parameters that exhibited the most significant changes during tachyarrhythmias and consistently returned toward baseline included mean RA, RV systolic, and RV pulse pressures. To identify a tachyarrhythmia and develop the optimal algorithm, pressure changes of each of these parameters, with a heart rate criterion, were examined alone and in combination. Parameter changes after 15 seconds of both paced (RA and RV) and induced ventricular tachyarrhythmias were examined in concert with their rate. Algorithms examined for their ability to detect hemodynamically unstable tachyarrhythmias included 1) heart rate of 150 beats/min or more and a mean RA pressure increase of 5 mm Hg or more during 15 seconds (rate-mean RA pressure algorithm), 2) heart rate of 150 beats/min or more and a decrease in RV systolic pressure of 6 mm Hg or more during 15 seconds (rate-RV systolic pressure algorithm), 3) heart rate of 150 beats/min or more and a decrease in RV pulse pressure of 7 mm Hg or more during 15 seconds (rate-RV pulse pressure algorithm), and 4) a combined detection algorithm: heart rate of 150 beats/min or more and mean RA pressure increase of 4 mm Hg or more and RV systolic pressure decrease of 5 mm Hg or more during 15 seconds (rate-mean RA pressure–RV systolic pressure algorithm). These algorithms were then applied to the next 20 consecutive patients (left ventricular ejection fraction of 34±4%; p=NS compared with the first 10 patients) and compared with the current rate-only algorithm (heart rate of 150 beats/min or more) in 143 tachyarrhythmias, and the sensitivity and specificity of detection hemodynamically unstable tachyarrhythmias were determined. Table 3 shows the characteristics of the 143 tachyarrhythmias in which the above detection algorithms were tested. Of the 143 tachyarrhythmias studied, 60 were supraventricular (57 RA paced tachycardias with 1:1 AV conduction, two spontaneous atrial fibrillations, and one spontaneous atrial flutter with 2:1 AV conduction), and 83 were ventricular (60 RV paced tachycardias, 18 induced ventricular tachycardias, and five induced ventricular fibrillations). Twenty-seven of the 143 tachyarrhythmias satisfied our definition of “hemodynamically unstable.” Induced ventricular tachycardia cycle lengths ranged from 205 to 390 msec with four 300-msec or more (two of which were hemodynamically stable). The rate-only detection algorithm had 100% sensitivity, but only 68% specificity, for detection of unstable tachyarrhythmias, whereas the combined rate-mean RA pressure–RV systolic pressure detection algorithm had 100% detection sensitivity and specificity (Table 4).

In five patients, hemodynamic and heart rate responses to cough and 15 seconds of Valsalva maneuver were studied with the rate-mean RA pressure–RV systolic pressure algorithm to determine whether these responses can be mistaken for a hemodynamically unstable tachyarrhythmia. Consistent increases in mean RA and RV systolic pressures were observed during Valsalva maneuver and coughing, and heart rates did not increase beyond 120 beats/min; therefore, no events were incorrectly identified.

### Discussion

In this study, we systematically compared the effects of supraventricular and ventricular paced tachycardias and showed that for a given cycle length, ventricular pacing causes greater hemodynamic sequelae. In addition, we demonstrated that changes in mean arterial pressure can be accurately reflected by changes in right heart pressures. Increases in mean RA pressure reflected not only rate-related impairment in diastolic filling but atrial contractions against closed AV valves. The continuous occurrence of these contractions, evidenced by cannon “a” waves during RV pacing with VA conduction and during simultaneous RA and RV pacing, resulted in more pronounced increases in mean RA pressure than appropriately timed atrial contractions during sinus rhythm or atrial pacing. RV pacing with VA dissociation resulted in intermittent cannon “a” waves and intermediate increases in mean RA pressure.
Mild increases in tricuspid regurgitation resulting from asynchronous myocardial contractions during tachycardias may further contribute to this increase in mean right atrial pressure. Similar changes were reflected by RV (systolic and pulse) pressures but only with more rapid tachyarrhythmias (induced ventricular tachycardia and ventricular fibrillation). Slower tachycardias, used in our pacing protocol, failed to demonstrate any significant change, illustrating that diastolic dysfunction may predominate at lower tachyarrhythmia rates and systolic dysfunction at higher rates.\(^{31}\) In addition, the hemodynamic responses to both paced and induced tachyarrhythmias were unaffected by the patient’s left ventricular ejection fraction.

Because currently available antitachycardia devices, such as the AICD, detect tachyarrhythmias primarily by rate-only algorithms, they frequently cannot differentiate hemodynamically significant from insignificant tachycardias. Clinically inappropriate discharges of these devices during asymptomatic, hemodynamically stable tachycardias such as atrial fibrillation or other supraventricular arrhythmias have been reported in approximately 25% of patients with implanted AICDs.\(^{32-34}\) Such unnecessary discharges may prematurely deplete the battery supply, inflict pain on the conscious patient, and, most importantly, induce ventricular tachycardia or ventricular fibrillation.\(^{35}\) We documented such an event in a patient who participated in this study (Figure 4). During the study, he experienced a stable spontaneous supraventricular tachycardia and an unstable induced ventricular tachycardia. Later, he died suddenly, out of hospital, as a result of AICD-induced ventricular fibrillation during a supraventricular tachycardia with unsuccessful device rescue. Perhaps a hemodynamically responsive detection algorithm might have prevented this unfortunate event.

Rate-only detection algorithms perform suboptimally, perhaps because hemodynamic compromise results not only from rate-related shortening of diastole impairing ventricular filling, but also from other factors including asynchronous myocardial contractions caused by abnormal site and sequence of ventricular activation, loss of “atrial kick” from inappropriately timed atrial systole, ischemia, and neuroendocrine effects.\(^{24,36-45}\) Other detection algorithms that rely solely on electrogram analysis (sudden-onset rate detection, AV timing sequence, morphology, or electrogram frequency spectrum analysis) cannot adequately determine the hemodynamic state of a tachyarrhythmia.

A hemodynamic parameter incorporated into an implantable antitachycardia system should ideally have 1) stability at baseline, 2) abrupt and significant change during a hemodynamically significant tachyarrhythmia and rapid return to baseline after termination, 3) distinguishability of its changes during tachyarrhythmia from those due to baseline variation and other physiological events such as changes in body position or Valsalva maneuvers, 4) ease of measurement, and 5) sensor durability. By coupling the suitable hemodynamic parameters found in this study (mean RA, RV pulse, or RV systolic pressures) to the electrocardiographic signal, we should be able to develop an antitachycardia system capable of distinguishing hemodynamically stable from unstable tachyarrhythmias. This study provides important data regarding right heart pressure parameters and their potential for incorporation into a hemodynamically responsive antitachycardia system.

Although mean RA, RV pulse, or RV systolic pressures are useful parameters for such systems, individually they may be subject to perturbations from changes in position, cough, and Valsalva maneuver. Therefore, an algorithm that combines rate, mean RA, and RV systolic pressures was designed, not only to distinguish significant tachyarrhythmias, but also to discriminate between potentially confounding events. In 20 patients, this algorithm demonstrated 100% sensitivity and specificity of detection of hemodynamically unstable tachyarrhythmias, and it was capable of distinguishing unstable tachyarrhythmias from coughs and Valsalva maneuvers. Figure 5 depicts actual hemodynamic and electrical signals, which were integrated with the combined rate-mean RA pressure–RV systolic pressure algorithm to manually trigger both an antitachycardia pacemaker and a defibrillator. This algorithm detected a stable tachycardia, and ventricular overdrive pacing was initiated. When the tachyarrhythmia accelerated and became unstable from the overdrive attempt, the back-up defibrillator was triggered and successfully terminated the tachyarrhythmia.

The implementation of a hemodynamically responsive antitachycardia system would require a long-term stable implantable pressure sensor system, possibly of strain-gauge or piezoelectric design. Transducer drift could be minimized by comparing short-term (e.g., 15 seconds) to long-term (e.g., 15 minutes) mean pressures.\(^{25}\) This configuration would permit the discrimination of gradual hemodynamic changes during chronic stable atrial fibrillation from abrupt changes during unstable atrial fibrillation. If both RA and RV pressures are used in the algorithm, the RA pressure sensor, rather than atmospheric pressure, may serve as reference and may be compared directly to RV pressure. Two sensors with predictable equivalent drift patterns could be aligned so that the pressure difference would be relatively independent of drift. Programmability of detection algorithm criteria (rate, pressures, and time required to determine the hemodynamic state) would permit the antitachycardia system’s performance to be patient specific and sensitive to relatively slow unstable ventricular tachycardias. Similar algorithms may be designed with shorter times to determine hemodynamic instability, thus minimizing time-dependent tachyarrhythmia effects on defibrillation thresholds and, if desired, permitting the patient to remain conscious during arrhythmia termination. Future detection algorithms may integrate more sophisticated electrogram signal analysis (capable of distinguishing supraventricular from
ventricular tachyarrhythmias) with hemodynamic criteria (capable of distinguishing stable from unstable rhythms) to select specific treatment algorithms (i.e., tiered antiarrhythmic therapy).

This study has several limitations. Pressure changes were recorded while patients were supine and, therefore, may not represent changes while standing. We suspect, however, that such changes

Cohen and Liem  Hemodynamically Responsive Antitachycardia System  403

FIGURE 4. Tracings that show firing of an automatic implantable cardioverter-defibrillator (AICD) during a supraventricular tachycardia may induce ventricular fibrillation (and sudden cardiac arrest). This study patient had been complaining of asymptomatic AICD discharges and was therefore given a memory-loop recorder and was instructed to activate this system immediately upon experiencing such a discharge. Three days later, out of hospital, he experienced an apparent asymptomatic discharge (having activated his recorder), then collapsed and could not be resuscitated by either his AICD or cardiopulmonary resuscitation. Memory-loop recorder tracings demonstrated AICD-induced ventricular fibrillation during a supraventricular tachycardia (above his device's rate cutoff) followed by an unsuccessful AICD rescue. AICD 30 J, automatic implantable cardioverter-defibrillator discharge of 30 J.
Figure 5. Tracings that depict hemodynamic and electrical signals that were integrated, using the rate-mean right atrial pressure–right ventricular systolic pressure algorithm, to manually control an antitachycardia pacemaker and defibrillator. A hemodynamically stable tachyarrhythmia was detected that permitted manual initiation of overdrive ventricular pacing, accelerating the tachyarrhythmia with resultant hemodynamic instability, followed by defibrillation rescue. VT, ventricular tachycardia; VF, ventricular fibrillation; ECG, electrocardiogram; HRA, high right atrium (intracardiac electrogram); HBP, His bundle proximal (intracardiac electrogram); HBD, His bundle distal (intracardiac electrogram); RVA, right ventricular apex (intracardiac electrogram); MAP, mean arterial pressure; RAP, right atrial pressure; MRAP, mean right atrial pressure; RVP, right ventricular pressure; MRVP, mean right ventricular pressure.
may be even greater during clinical arrhythmic events, in which the patient would fall from an upright to a supine position. In addition, although right and left heart pressure changes were sufficiently large to permit use of fluid-filled catheters, manometer catheters might have permitted finer pressure delineations. Finally, algorithms were tested in only 20 patients, during primarily paced and induced tachyarrrhythmias, with very few spontaneously occurring arrhythmias. Rapid ventricular pacing at rates between 100 and 150 beats/min permitted algorithm testing during hemodynamically stable slow-paced ventricular tachycardias. Most induced ventricular tachyarrrhythmias in which algorithms were tested were both rapid and hemodynamically unstable. Further testing, with greater numbers and varieties of spontaneous tachyarrrhythmias (including slower ventricular tachycardias and more rapid atrial fibrillations), is necessary to prove the usefulness of these algorithms in an implantable hemodynamically responsive antitachycardia system.

In conclusion, we found that mean RA, RV systolic, and RV pulse pressures are useful for incorporation into a hemodynamically responsive antitachycardia system. Four algorithms, which combined at least one of these parameters with the heart rate criterion, were tested in 20 patients and were found to have a higher specificity for detecting unstable tachyarrrhythmias than the rate-only algorithm. Therefore, the application of these algorithms to antitachycardia systems may decrease the frequency of device firings and improve device longevity and performance.

Acknowledgments
We thank the Stanford University Medical Center’s Electrophysiology Laboratory nursing and engineering staff for their assistance, Drs. Edwin Alderman, William Clusin, and Enrico Veltori for their critique of the manuscript, Dr. Randall Lee for his technical assistance, and Dr. Irene Hill for her assistance in statistical analysis.

References

**KEY WORDS** • hemodynamics • automatic implantable cardioverter-defibrillator • tachyarrhythmia detection • antiarrhythmia system
A hemodynamically responsive antitachycardia system. Development and basis for design in humans.
T J Cohen and L B Liem

Circulation. 1990;82:394-406
doi: 10.1161/01.CIR.82.2.394

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
Copyright © 1990 American Heart Association, Inc. All rights reserved.
Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
http://circ.ahajournals.org/content/82/2/394