Influence of cycle length variations on antitachycardia pacing effectiveness among ICD patients

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BACKGROUND Antitachycardia pacing (ATP) fails to terminate 5% to 25% of ventricular tachycardias (VTs) occurring in implantable cardioverter-defibrillator patients. We speculated that small fluctuations in VT cycle length (CL) may be related to the efficacy of subsequent ATP.

OBJECTIVE The purpose of this study was to determine the relationship between the R-R variations of the last 12 R-R intervals before ATP and the efficacy of the first ATP attempt.

METHODS We studied 551 VTs (CL 329 ± 35 ms) occurring in 67 patients. We also analyzed the percentage of variation (P-RR), which was calculated by dividing the mean difference between each R-R interval and the next one by the CL (×100), and the acceleration index (AI), which was calculated by dividing the CL of the first 6 R-R intervals by the CL of the next 6.

RESULTS The effectiveness of the first ATP therapy was 81%, being higher in VTs with AI <1 (85% vs 64%; P <.001). After classifying the events according to the tertiles of P-RR, ATP efficiency was better in higher values of P-RR (VTs with AI <1): 99% (third tertile) vs 85% (second tertile) vs 76% (first tertile), P <.001; and for VTs with AI ≥1: 94% vs 68% vs 42% (P <.001). By logistic regression, P-RR (%; odds ratio 2.37; P <.001), and AI <1 (odds ratio 4.17; P <.001) were found to be independent predictors of successful first ATP attempts.

CONCLUSION Small changes in CL increase the effectiveness of ATP significantly. VTs with lower degrees of R-R fluctuations, especially when the pattern is a progressive CL shortening, are infrequently terminated by ATP.

KEYWORDS Antitachycardia pacing; Implantable cardioverter-defibrillator; Ventricular tachycardia

ABBREVIATIONS **AI** = acceleration index; **ATP** = antitachycardia pacing; **CI** = confidence interval; **CL** = cycle length; **CL-dif** = difference between the shortest and the longest cycle length; **ICD** = implantable cardioverter-defibrillator; **LVEF** = left ventricular ejection fraction; **Mn-RR** = mean difference between each R-R interval with the next one; **P-RR** = percentage of variation of R-R intervals; **VT** = monomorphic ventricular tachycardia

Introduction

The vast majority of monomorphic ventricular tachycardias (VT) occurring in patients with structural heart disease are due to a reentrant mechanism.[1] Reentrant tachycardias are suitable for termination by overdrive pacing on the basis of the presence of an excitable gap; this is the difference between the cycle length (CL) and the refractory period of the circuit.

Implantable cardioverter-defibrillators (ICDs) are able to perform a variety of antitachycardia pacing (ATP) modalities to terminate spontaneous VT. Despite the high efficacy of ATP in terminating VT in ICD patients, 5% to 35% of such episodes, depending on the CL, are not finished by ATP.[2–6]

Several factors affect the ability of ATP to terminate a reentrant VT. The 2 most important factors are the tachycardia CL and the duration of the excitable gap. Both are interrelated, because the faster the VT, the shorter the excitable gap. In fact, different studies have shown that the CL is the most important predictor of ATP success in spontaneous VT occurring in ICD patients.[3,7] In addition, the probability of VT termination is influenced by the conduction time from the stimulation site to the site of impulse formation, which depends on the proximity of the pacing site to the circuit (closer is better).[8] In this sense, it has been shown that ATP from the right ventricular apex is less effective for VTs with basal exit[9] and that biventricular pacing (compared with right ventricular pacing) is more effective in ischemic patients.[10]

However, to the best to our knowledge, no other stored electrogram-based variables have been studied in order to establish their relationship with the effectiveness of ATP. Because CL variability is common in VT occurring spontaneously in ICD patients,[11] we conducted a prospective study aimed at determining the relationship between both the degree and the pattern of variation of VT CL and the efficiency of the subsequent ATP.

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Methods

Patient selection, devices, and follow-up

Two hundred sixteen patients with structural heart disease (left ventricular ejection fraction [LVEF] 31% ± 10%; prior myocardial infarction 67%; New York Heart Association functional class II–III 62%) and standard indications for single-chamber ICD therapy (primary prevention 56%) were consecutively included in this study.[5] Inclusion criteria were broad, excluding only ICD patients believed to be unlikely to have a substrate for monomorphic VT susceptible to ATP (hypertrophic cardiomyopathy, long QT syndrome, or Brugada syndrome). Patient recruitment was performed at ICD implantation.

All patients underwent implantation of pectoral devices with a transvenous endocardial lead positioned at the right ventricular apex. Upon discharge from the hospital, patients were instructed to list the time and date of symptoms possibly related to spontaneous ventricular arrhythmia, such as syncope or near-syncope. In addition, patients were asked to record their pharmacologic treatment. We correlated the dates of each VT detected and stored by the devices with the medical treatment of the patients and the dates of the symptoms reported by them. In each case, we determined the equivalent dose of β-blockers at VT presentation. Dose equivalents were defined using metoprolol (mg/day) as a reference.[12]

Patients were seen every 3 months, including clinical visit, symptom monitoring, and ICD interrogation. Symptoms were correlated with ICD-stored episode data of spontaneous ventricular arrhythmias. Syncope required complete loss of consciousness with loss of postural tone. Near syncope was defined as severe dizziness or light-headedness without complete loss of consciousness.

The study complied with the Declaration of Helsinki. Enrollment followed acceptance of the protocol by the institutional review board, and informed consent was obtained from all patients.

Device programming

Detection and therapy programming were standardized and included 3 zones: ventricular fibrillation: CL <250 ms; ventricular tachycardia zone 1 (fast VT): CL from 250–320 ms; and ventricular tachycardia zone 2 (slow VT): CL from 321–400 ms.

For Medtronic devices, ventricular fibrillation detection required that 18 of the last 24 R-R intervals have CL <250 ms (>240 bpm). The fast VT detection zone was defined within the ventricular fibrillation zone (fast VT zone via ventricular fibrillation): when any of the final 8 R-R intervals preceding the moment of detection was <250 ms (>240 bpm), the episode was classified as ventricular fibrillation and received a high-energy shock. When all of the last 8 R-R intervals were >250 ms (<240 bpm), the episode was detected as fast VT.

For Boston Scientific devices, the episodes were detected when 8 of 10 R-R intervals had a CL within the detection interval and, during the subsequent 1.5 seconds, at least 6 of every 10 R-R intervals were within the predefined limits for VT detection.

First ATP therapy in the fast VT zone was a single ATP sequence (5-pulse-burst pacing train at 84% of the VT CL). Failed ATP was followed by shock and then other shocks as necessary. Therapies for slow VT included 3 consecutive ATP bursts of 15 pulses at 91% of the VT CL, with no decrement. Failed ATP was followed by a sequence of shocks.

All devices were programmed to store the far-field electrogram before the onset of the episodes detected to aid in rhythm classification. In the Medtronic devices the "Smart mode" was programmed to "off."

Rhythm classification and definitions

All tip-to-ring and far-field-stored electrograms from spontaneous episodes were classified using predetermined criteria based on visual inspection and comparison with sinus rhythm electrograms.[13–15] Exclusion of supraventricular tachycardias and classification of ventricular tachyarrhythmias (ventricular fibrillation, fast VT, and slow VT) were performed by 2 independent investigators. Episodes exceeding the electrogram storage capability of the device were not included in this analysis.

The CL of VT and the variability of the R-R intervals were determined manually from the marker channel in the last 12 R-R intervals preceding the first ATP attempt. The variability in the R-R intervals was determined as the difference between the shortest and the longest CL (CL- dif) and as the percentage of variation (P-RR), which was calculated by dividing the mean difference between each R-R interval and the next one (Mn-RR) by the VT CL: (Mn-RR/VT CL) × 100 (Figure 1). The pattern of variation was defined by the acceleration index (AI), which was calculated by dividing the CL of the 6 R-R intervals preceding the first ATP attempt by the CL of the previous 6 R-R intervals.

ATP therapy was deemed successful when the post-therapy rhythm was not a ventricular tachyarrhythmia. The maximal temporal delay between the last ATP stimulus and the first non–VT-VF beat accepted for classifying the therapy as successful was 5 seconds. Acceleration was defined as a >10% decrease in CL after the first ATP attempt. Episode duration was defined according to the device and included the time after therapy until the episode termination criterion was met.

Statistical analysis

Statistical analysis was performed using the SPSS package version 11.5 for Windows (SPSS Inc, Chicago, IL). Normal and continuous variables are given as mean and standard deviation. Categorical variables are given as number of patients and percentage. Comparison of categorical variables was performed using the χ^2 test (or Fisher exact test if n <5). Comparison of 2 normal variables (determined by the