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Ventricular Tachyarrhythmias in Implantable Cardioverter Defibrillator Recipients: Differences Between Ischemic and Dilated Cardiomyopathies

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1. Introduction

Progress in implantable cardioverter-defibrillator (ICD) technology has led to significant improvements in the management of malignant ventricular tachyarrhythmias (VT) from patients with different etiologies [Henkel & Witt 2006, Javaray & Monahan 2005]. Figure 1 depicts the case of a patient who experienced a malignant ventricular fibrillation rapidly resolved by the ICD.

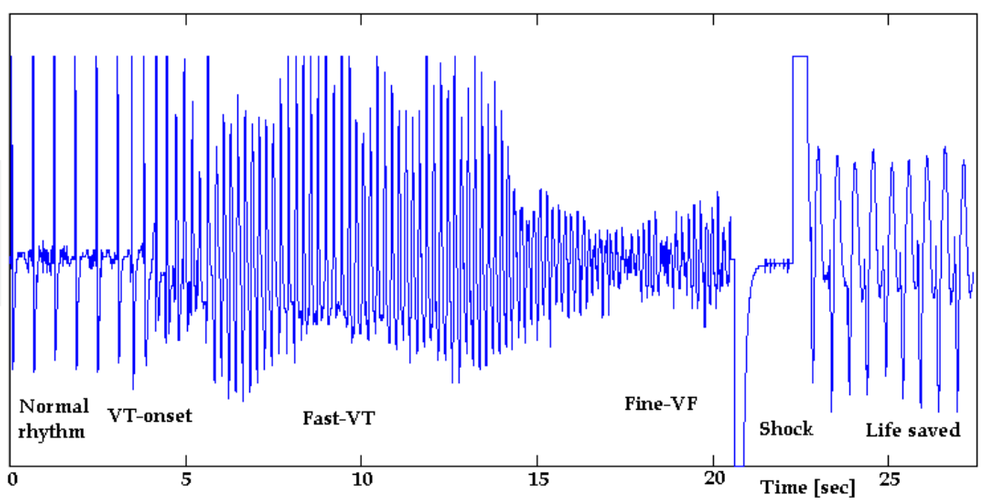

Fig. 1. Example of malignant ventricular tachycardia degenerating in ventricular fibrillation, automatically recognized and treated by the ICD.

In addition, the ability of these devices to record and store information on electrical cardiac activity immediately preceding the arrhythmia onset and during its course has enabled the

assessment of the initiation pattern and characteristics of these arrhythmias. Eventually, at the end of the nineties, it has been shown that coronary artery disease (CAD) and dilated cardiomyopathy (DCM) underlie different mechanisms of spontaneous and induced VT [Pogwizd et al. 1998, Chung et al. 1997]; moreover ,differences in spontaneous VT from ICD recipients began to be investigated. Indeed, since the early 2000, various studies have analyzed intracardiac electrograms (EGMs) and initiation VT pattern [Gorenek et al. 2006, Taylor et al. 2000, Saeed et al. 2000], while others have investigated circadian distribution [Carson et al. 2000, Englund et al. 1999, Eksik et al. 2007, Taneda et al. 2001] of spontaneous VT in ICD patients. Some of these studies [Taylor et al. 2000, Saeed et al. 2000, Englund et al. 1999] have failed to detect differences in both the patterns of initiation and in EGMs between patients with different etiologies of heart disease. In contrast, differences in the circadian distribution were detected in other studies [Carson et al. 2000, Taneda et al. 2001], showing that patients with CAD had a peak of the VT episodes in the morning, whereas patients with non-ischemic cardiomyopathy and heart failure had a more uniform VT distribution during the light hours and a few episodes during the night. The main limitation of these studies lies in their definition of non-ischemic cardiomyopathy, which included different cardiac diseases such as dilated cardiomyopathy, hypertrophic cardiomyopathy, valvular heart disease, right ventricular dysplasia and others.

Unlike previous studies, this study focuses exclusively on patients with CAD and those with DCM. Therefore, by analyzing spontaneous VT from ICDs, we sought to determine whether there were differences in EGM characteristics obtained from far-field recordings, VT prevalence, initiation patterns and circadian distribution between patients with CAD and DCM. A subset of this study was preliminarily presented [Casaleggio et al. 2008].

2. Methods

2.1 Electa registry

Sixty-seven patients were enrolled in the ElectA Registry (Electrogram Analysis) which included subjects undergoing implantation of St. Jude Medical single chamber ICD devices (models: Contour, Profile and Angstrom) for secondary prevention of aborted sudden cardiac death (SCD) due to ventricular fibrillation or sustained VT episodes (with duration greater than 30 sec). Among them, 46 had ischemic cardiomyopathy due to CAD, 17 had primary DCM and 4 had other etiology: one Brugada Syndrome, one RV Dysplasia and two hypertrophic cardiomyopathies.

ICDs allowed acquisitions of the EGMs in bipolar or far-field modes. In bipolar mode, EGMs were recorded between distal coil and electrocatheter tip at the right ventricular lead tip, and filtered with an high pass at 12 Hz. In far-field mode, EGMs were recorded between the distal coil and the active can of the device without low frequency filtration. In our previous study [Casaleggio et al. 2006], we examined the differences between bipolar and far-field recordings, and observed that the two modes of acquisition should not be combined to avoid characterization errors. Furthermore, far-field recordings easily recognized pacing beats, allowed a better definition of the T-wave in sinus rhythm and a higher discrimination of the QRS morphology for the off-line diagnosis of VT (see Figure 2).

In order to study VT-onset, ICDs were programmed to store EGMs of 2 minutes at a sampling frequency of 250 Hz, that allowed up to three EGMs recordings on a single channel at each follow-up. Moreover, since storage was triggered on VT initiation (when VT is shorter than 2 minutes), the protocol required a pre-trigger of 20 sec. at minimum to

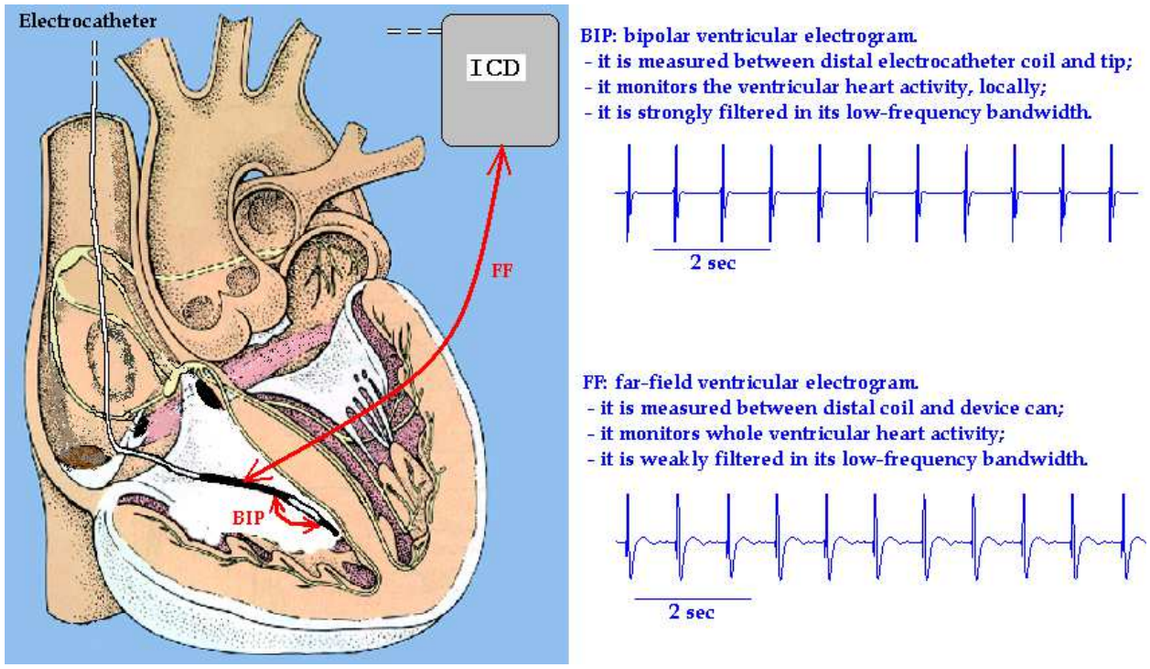

Fig. 2. Schematic of bipolar and far-field EGMs from ICDs. In the right side of the panel the main characteristics of the two types of recordings are described and examples are shown.

monitor basal rhythm immediately before VT initiation. Patients gave written consent for the implantation , follow-up and planned evaluations; they were identified by their ICD serial numbers. Patients were excluded from the ElectA study if they had an indication to dual-chamber ICD implantation, or refused to consent. ICD settings were at the discretion of the physician, except for a pre-trigger at 20 seconds. No requirements were proposed either for VT- therapy (rate cutoff and monitoring zone) or bradycardia pacing, although all clinicians programmed a lower rate in VVI mode at 50-60 beats per minute.
Clinical information for each patient including age, gender, heart disease, left ventricular ejection fraction, and antiarrhythmic drug therapy at the time of ICD implant, were documented by review of clinical records. Information on VT events and circadian distribution were retrieved from the log file of the ICD device. ICD follow-up was routinely performed every 3-4 months and whenever patients felt palpitations, dizziness or shock.

2.2 Patient population

The present study focused only on patients with CAD or DCM. The CAD group included 46 patients (26 of which had VT episodes) with a history of previous myocardial infarction, angina, coronary angioplasty or coronary by-pass surgery. The DCM group included 17 patients (11 of which had VT episodes) with progressive cardiac dilation and left ventricular systolic dysfunction (ejection fraction less than 0.40), without significant CAD defined as greater than 50% stenosis in at least one vessel by coronary angiography. In all patients secondary etiology was excluded by clinical evaluation of potential causes such as alcoholism, thyroid disease, viral infection of the heart, valvular heart abnormalities, heredofamilial disease, or toxic drugs. No endomyocardial biopsy was obtained.
The baseline characteristics of CAD and DCM patients that experienced spontaneous malignant VT episodes are reported in Table 1.

	CAD	DCM	p
N. of patients with VT	26	11	
Clinical Characteristics			
Age (years)	70 ± 10	64 ± 12	n.s. (α)
Gender (male/female)	23 / 3	10 / 1	n.s. (β)
Follow-up (months)	33 ± 10	24 ± 13	<0.03 (α)
Ejection Fraction (%)	35 ± 8	31 ± 9	n.s. (α)
NYHA Class: I / II+III (n)	2 / 24	2 / 9	n.s. (β)
Basal rhythm: Sinus / Atrial fibrillation (n)	23 / 3	8 / 3	n.s. (β)
Treatment at ICD implant: Amiodarone , n (%) Beta-blockers , n (%) Amiodarone + Beta-blockers , n (%) None or other drugs , n (%)	 14 (54%) 1 (4%) 3 (11%) 8 (31%)	 3 (27%) 1 (9%) 1 (9%) 6 (55%)	

Table 1. Clinical Characteristics of CAD and DCM patients with VT episodes; (α) Statistical analysis performed with Student T-test; (β) Statistical analysis performed by chi-square test.

2.3 Modes of VT initiation and evaluated EGM characteristics

All VT episodes (monomorphic and polymorphic VT and ventricular fibrillation) were analyzed by cardiologists. VT were identified by a sudden increase in heart rate (defined as a rhythm greater than 140 beats per minute) along with a change in EGM morphology from the baseline rhythm. Arrhythmias due to atrial fibrillation or atrial flutter were excluded from the analysis. Using EGMs, VT initiation was characterized as follows: VT initiated by a premature ventricular contraction (PVC), defined as PVC pattern (Fig. 3A); VT initiated by a short-long-short cycle (SLS pattern, Fig. 3B); VT initiated with a PVC immediately after a paced ventricular beat (PM pattern, Fig. 3C). The pause preceding paced beats was considered appropriate if it was consistent with the programmed pacing escape interval.

For each VT episode the following features were analyzed: (i) *VT-cycle* defined as the median value of the VT cycles, in milliseconds (msec); (ii) *Coupling interval* (CI) taken as the interval between the first beat of VT and the previous baseline beat, in msec; (iii) *Prematurity index* (PI) calculated by normalizing the coupling interval to the preceding RR interval; (iv) *Median heart cycle* of the 20 seconds immediately preceding VT onset (HC-PreVT), in msec; (v) number of PVC and (vi) number of paced beats in the 20 sec preceding VT. The morphological analysis of PVC and VT was not considered.

Circadian distribution analysis was made over a 3 hour time interval, performing a sufficiently detailed analysis and an adequate number of VT episodes in each time interval.

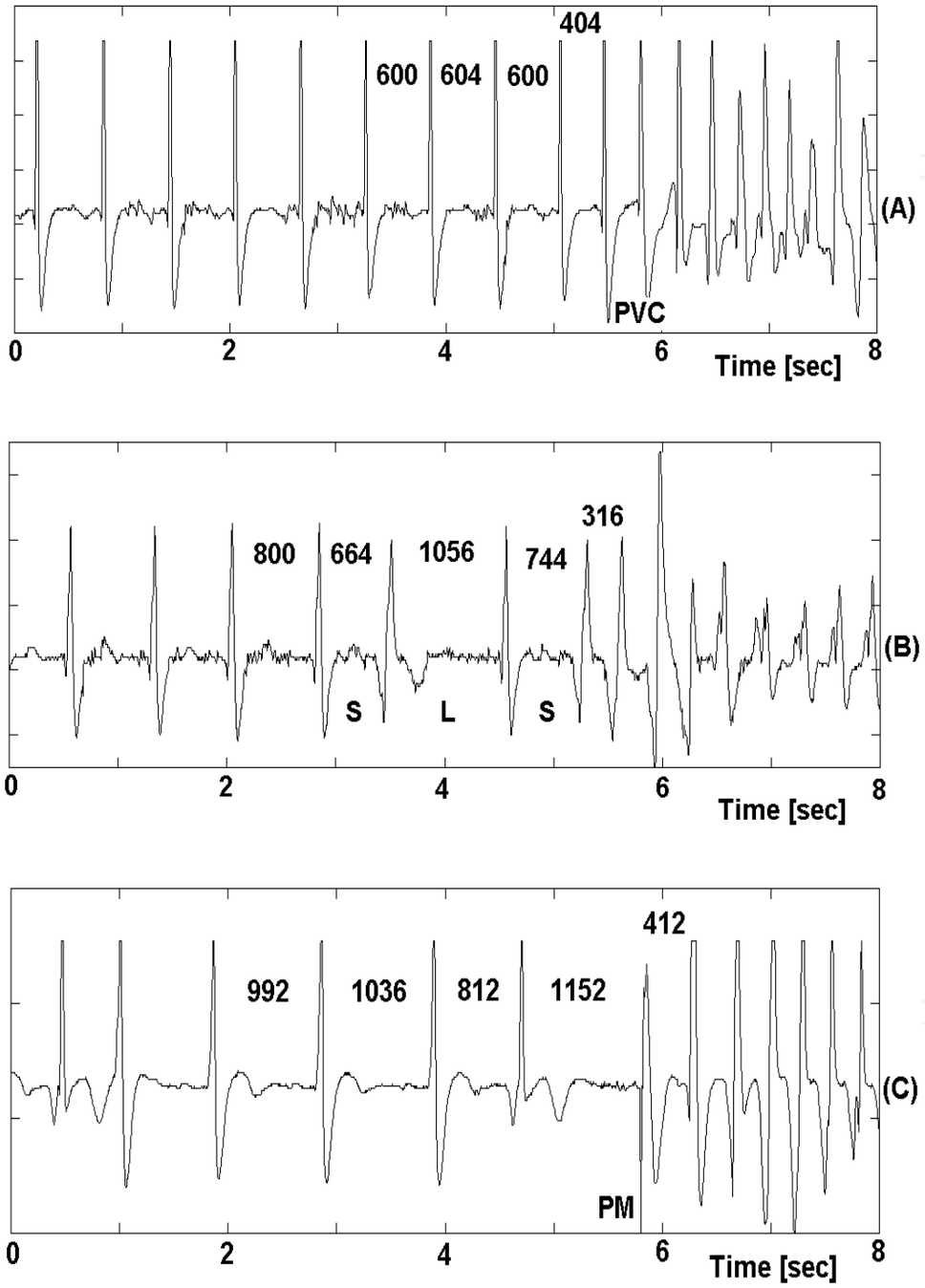

Fig. 3. Examples of tachyarrhythmia initiation patterns from ICD recordings: Panel (A) shows a PVC onset, Panel (B) is a short-long-short (SLS) onset, Panel (C) illustrates a tachyarrhythmia initiated immediately after a paced beat (PM onset); Numbers between R-peaks indicate RR intervals in msec.

2.4 Statistical analysis

Intervals and continuous variables are expressed as mean ± standard deviation. Differences between groups are analyzed using the Student T-test for continuous variables or the Chi-square test for categorical variables. Statistical significance is assumed when $p<0.05$. The number of VT per patient, and the number and type of VT-onset experienced by the patients, are considered together with their average values. Differences between average values are analyzed using the Student T-test.

Circadian distributions are modeled by polynomial (fifth order) and harmonic regression (two harmonics). It is expected that distributions with peaks and dumb-peaks can better be fitted by harmonic models, while smooth distributions would be better modeled as polynomials. Goodness of fit of the regression models has been tested using the coefficient of determination R^2, defined as in equation (1):

$$R^2(y) = 1 - \frac{\sum_i (y_i - \hat{y}_i)^2}{\sum_i (y_i - \overline{y})^2} \tag{1}$$

In equation (1) the value of y_i represents the measured variable for each 3-hour interval, $\overline{y}_i$ is its mean value, and $\hat{y}_i$ is the approximation obtained by the regression model. No formal sample size calculation has been performed.

3. Results

A total of 218 VT episodes were recorded in far-field mode during a follow up period of 30 ± 12 months. The analysis included all VT episodes requiring ICD therapy by antitachycardia pacing or shock cardioversion, as well as non-treated VT that recovered spontaneously. In particular 165 VT episodes which occurred in 37 patients, including 26 CAD patients (79 episodes) and 11 DCM patients (86 VT episodes) were analyzed. The remaining 53 VT episodes, (29 from DCM and 24 from CAD patients) could not be analyzed because, lasting more than 2 minutes, VT initiation was not available for ICD programmable setting. No under-sensing was observed.

As shown in Table 2, there were significant differences between CAD and DCM patients with VT episodes. In particular, DCM patients experienced a significantly higher number of VT episodes and more variable pattern of VT initiation.

Table 3 presents the statistical analysis of EGM features associated with specific VT initiation patterns in patients with CAD or DCM. There were no significant differences between CAD and DCM groups. However, within each group, the SLS and PM patterns showed a significantly lower prematurity index than the PVC pattern. In addition, the PM pattern demonstrated a significantly longer heart cycle preVT versus PVC and SLS ($p < 0.01$).

Figures 4 and 5 depict the circadian distributions of VT episodes in CAD and DCM patients, with an analysis in the overall groups (Fig. 4) and in subgroups with different patterns of VT initiation (Fig. 5). Figure 4 shows the presence of the two peaks in CAD patients, and a more uniformly distribution during daylight hours with a few VT episodes during the night in DCM patients.

	CAD	DCM	P
Number of patients with VT	26	11	
Analysis of the 20 sec basal rhythm preceding VT-onset			
Paced beats / All beats	1 / 28	1 / 27	n.s. (β)
Premature ventricular contraction / All beats	2 / 27	3 /27	n.s. (β)
Analysis of VT episodes			
Number of VT episodes	79	86	
Number of VT episodes per patient, mean ±SD	3 ± 2	8 ± 5	<0.02 (α)
Analysis of the VT-onset patterns			
Total number of VT per pattern:			
PVC, n (%)	59 (75%)	51 (59%)	n.s. (γ)
SLS, n (%)	13 (16%)	22 (26%)	
PM, n (%)	7 (9%)	13 (15%)	
Number of patients with 1, 2 or 3 patterns:			
Patients with a single initiation pattern, n (%)	22 (85%)	5 (45%)	< 0.01 (δ)
Patients with two initiation patterns, n (%)	4 (15%)	3 (27.5%)	
Patients with three initiation patterns, n (%)	0 (0%)	3 (27.5%)	
Average number of patterns per patient, mean ±SD	1.1 ± 0.4	1.8± 0.9	< 0.01 (α)

Table 2. Analysis of VT episodes in patients with CAD or DCM; Different statistical tests were applied: (α) Statistical analysis is performed with Student T-test; (β) Statistical analysis is performed by chi-square test; (γ) chi-square test is done on 3x2 table to test different N*umber of VT per pattern* in CAD vs. DCM; (δ) chi-square test is computed on 3x2 table to test different *Number of patients with 1, 2 or 3 patterns* in CAD vs. DCM patients.

	CAD Patients			DCM Patients		
VT onset	PVC	SLS	PM	PVC	SLS	PM
# of Episodes	59	13	7	51	22	13
VT cycle (msec)	356±62	325±45	298±68	323±48	343±44	332±85
CI (msec)	497±147	515±70	600±136	499±162	542±112	533±125
PI	0.75±0.18	0.56±0.11*	0.5±0.12*	0.76±0.22	0.62±0.12*	0.48±0.14*+
HC-PreVT (msec)	697±170	663±112	1018±121*+	665±142	716±146	923±151*+

Table 3. EGM features associated with specific VT onset patterns in patients with CAD or DCM. Statistical significance is indicated as follows: * p <0.01 versus PVC within the same etiology group; + p<0.01 versus SLS within the same etiology group. Legend: VT patterns are defined as follows: PVC: VT initiated by a premature ventricular contraction; SLS: VT initiated by a short-long-short cycle; PM: VT initiated with a PVC immediately after a paced beat. CAD: coronary artery disease; DCM: dilated cardiomyopathy; CI: coupling interval, PI: prematurity index, HC-PreVT: heart cycle in the 20 seconds preceding VT-onset.

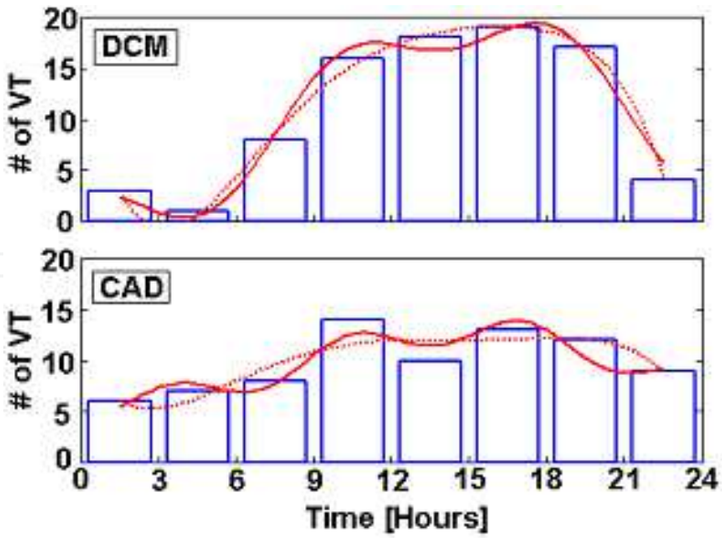

Fig. 4. Circadian distribution over a 3 hour time interval of CAD and DCM patients along with polynomial (dashed line) and harmonic (solid line) regression models.

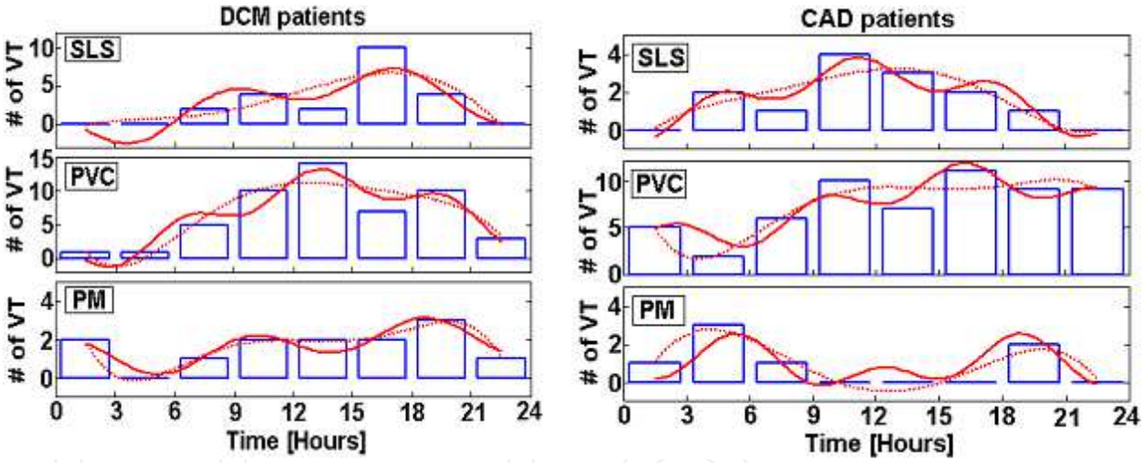

Fig. 5. Circadian distribution over a 3 hour time interval of CAD and DCM patients separated for the three different modes of onset. Corresponding polynomial (dashed line) and harmonic (solid line) regression models are shown.

To substantiate the observation that CAD and DCM population have different circadian distribution of VT episodes, the coefficients of determination (R^2), corresponding to the harmonic and polynomial regression models depicted in Figure 4, are computed. VT circadian distribution of CAD patients is better approximated by a harmonic model (R^2=0.963 vs. R^2=0.767 of polynomial regression). In DCM patients, harmonic and polynomial regression lead to similar R^2 values (R^2=0.983 vs. R^2=0.997, respectively), although the higher R^2 value is obtained approximating circadian distribution using a polynomial model.

Finally, determination coefficients of circadian distributions computed for homogeneous VT initiation pattern (PVC, SLS and PM) are resumed in Table 4.

	CAD Patients			DCM Patients		
	PVC	SLS	PM	PVC	SLS	PM
Harmonic model	**0.84**	**0.92**	0.71	0.78	**0.91**	0.85
Polynomial model	0.83	0.77	**0.9**	**0.86**	0.68	**0.98**

Table 4. Determination coefficient for harmonic and polynomial regressions: differences between CAD and DCM patients with similar VT-initiation patterns. Better performances are indicated in bold.

4. Discussion

The aim of this study was to determine whether there were differences in spontaneous VT initiation patterns and circadian distribution between ICD patients with CAD and DCM. Analysis of VT onset identified episodes triggered by a premature beat and two patterns of VT preceded by short-long-short cycles. The latter includes the SLS onset, which can be viewed as a spontaneous form, and the PM onset in which the ICD device facilitates VT initiation during anti-bradycardia pacing.

Significant differences between CAD and DCM were observed in the number of VT per patient and in the variability of the VT onset pattern (see Table 2). Patients with DCM had a significantly higher number of VT episodes despite clinical characteristics, ejection fraction and NYHA class were similar to CAD patients. It should be noted, as reported in Table 1, that the duration of follow-up was significantly longer in the CAD group, making the observation of a lower number of VT episodes in this group even more significant.

Other EGM features such as VT-rate, coupling interval and prematurity index, as well as those obtained from the EGM in the 20 sec preceding VT-onset (heart cycle, average number of PVC and of paced beats) were also studied (see Table 2 and 3) and, consistent with previous observations, no significant differences between CAD and DCM groups were observed. This might indicate that the studied EGM parameters do not express the intrinsic differences in the cardiac substrate of the two etiologies, as they are shown in the literature [Pogwizd et al. 1998, Sarter et al. 1998].

4.1 VT initiation patterns

In the literature the PVC onset is the most frequent mode of VT initiation [Taylor et al. 2000, Saeed et al. 2000]; SLS onset has been described as an arrhythmogenic mechanism [El-Sherif et al. 1999] distinct from PVC onset also in ICD recipients [Sarter et al. 1998]. Finally PM onset, observed only in case studies at the end of the nineties [Vlay & Vlay 1997], has been observed recently to occur in 8% – 15% of VT episodes in ICD patients [Sweeney et al. 2007].

Premature ventricular contraction (PVC) was the most frequent VT initiation pattern, especially in CAD patients.

Our results are consistent with findings from other authors [Gorenek et al. 2006, Taylor et al. 2000, Saeed et al. 2000], who underscored the importance of the initiation pattern of sustained VT in patients with ICDs. Gorenek et al. [Gorenek et al. 2006] and Saeed et al. [Saeed et al. 2000] analyzed the differences between VT onset preceded by several

immediate PVCs (denominated extrasystolic onset) and VT with sudden onset, showing that extrasystolic onset was the most common pattern of VT initiation and was associated with lower ejection fraction. Sudden-onset initiation was more common with better preserved systolic function. Non sudden onset episodes required higher levels of shock energy and more frequent multiple shock achievements than sudden onset episodes [Gorenek et al. 2006].

In the present study the analysis of the modes of VT-onset indicated that 12% of VT initiate immediately after a paced beat, which may suggest a proarrhythmic effect of pacing in some patients [Sweeney et al. 2007, Himmrich et al. 2003]. These results are consistent with recent findings: Sweeney et al [Sweeney et al. 2007] found a 9.4% rate of VT initiated immediately after anti-bradycardia pacing in patients with a single chamber ICD, such as those used in our study. In our study the prevalence of VT with PM-onset was higher in DCM (15%) than CAD (9%), but statistical analysis with chi-square test shows that this difference is not significant ($p<0.28$). On the contrary, a statistically significant difference between CAD and DCM populations is obtained from the analysis of the number of initiation pattern indicating a more heterogeneous VT-onset in DCM patients (see Table 2). In particular, in 3 DCM patients all the three initiation patterns are present, while most of CAD initiated their VT always with the same mode.

Results in Table 3 indicate EGM features do not allow discriminating differences between CAD and DCM, suggesting that EGM features do not correlate with the cardiac disease substrate. However, in each group, significantly higher prematurity index and median heart cycle PreVT were observed in VT with PM-onset with respect to other VT initiation modes. The HC_PreVT can be explained by observing that anti-bradycardia pacing is a consequence of a low spontaneous heart rate. The significantly higher PI of PM-onset episodes, observed both in CAD and DCM patients, deserves further investigation.

4.2 Circadian distribution

In the literature the circadian distributions of VT episodes in the CAD population has been studied and a non-uniform distribution with clear peaks in the morning and afternoon has been observed [Englund et al. 1999, Eksik et al. 2007]. Results are controversial in the case of non-ischemic patients. While in some papers [Englund et al. 1999, Anand et al. 2007] non-ischemic disease presents circadian distribution similar to ischemic patients, in other studies [Carson et al. 2000, Taneda et al. 2001] non-ischemic patients do not have the morning peak and circadian distribution is almost uniformly distributed during daylight hours. The present study shows the presence of the two peaks in CAD patients, and a more uniformly distribution during daylight hours in DCM patients (see Fig. 4). This conclusion is further supported by our observation that the timing of VT-onset in CAD and DCM populations are described by structurally different statistical models, with harmonic and polynomial regression providing the best fitting function in CAD and DCM, respectively. The finding that the circadian distribution of VT in CAD patients is better described by a harmonic function (such as a bimodal activity) may suggest that the underlying arrhythmogenic mechanism could be oscillating during the 24-hour period, as is the case of the autonomic sympathetic-vagal balance. This hypothesis is supported by Taneda et al. who described a much higher peak of VT in the morning in patients not taking beta-blockers vs. patients who did [Figure 2 in Taneda et al. 2001]. Similarly, Dorian suggested that sympathetic activation

can lead to a distributed shortening or, in some cases, prolonged action potential in the substrate, increasing the risk for reentrant arrhythmia [Dorian 2005].
By contrast, the result that the DCM circadian distribution is polynomial rather than harmonic, may suggest a different underlying mechanism at the basis of VT initiation. The observation that almost all VT occur during the day-light and almost none during the night, might lead to the hypothesis that sympathetic tone could be involved. In fact, Pogwizd described that a cardiac preserved beta-receptor responsiveness might likely facilitate early and delayed after-depolarizations, and, consequently, a triggered activity [Pogwizd et al. 2001, Pogwizd et al. 2004].
Such considerations might lead to speculate that sympatho-vagal tone changings could facilitate a reentry in the CAD group, while a higher sympathetic tone could determine a dangerous triggered activity in the DCM one.
It is noteworthy that the three different initiation patterns of VT episodes also show differences in the circadian distribution. (Figure 5 and Table 4).
VT initiated with PM pattern are better described, in both DCM and CAD patients, by a polynomial model. Distribution is almost uniform during the observed 24 hours in the DCM group , while it is mostly focused during the night in the CAD group. Although the number of VT is small (7 PM-onset in CAD and 13 in DCM), the result in the CAD population may be consistent with a lower heart rate occurring during the night, promoting a greater anti-bradycardia pacing intervention . It is less obvious to explain the finding in DCM, because the PM-onset occurs almost uniformly during the whole day. An increased prevalence of post-extrasystolic pauses distributed during the whole day, determining a frequent pacing escape, is more likely.
Circadian distribution of SLS patterns are well described by a harmonic models in both CAD (R^2 = 0.92 vs 0.77 for polynomial model) and DCM (R^2 = 0.91 vs 0.68 for polynomial model) with VT initiation especially between 9 a.m. to 6 p.m. in CAD, while it is evident a very strong peak between 3 p.m. to 6 p.m. in DCM patients.
Finally, the distribution of the PVC patterns is better described by a polynomial regression in the DCM group (R^2=0.86 vs. 0.78 for harmonic regression) with a peak in the middle of the day and no VT during the night (hour range: 0-6 a.m.); a different behavior is observed in the CAD group where a harmonic model fits slightly better (R^2=0.84 vs. 0.83 for polynomial model), with distribution showing small peaks between 9 a.m. and 12 a.m. and 3 p.m. to 6 p.m.

4.3 Limitations

The main limitations of this registry lie in the small number of patients and in the lack of endomyocardial biopsy in the DCM group. In addition, only single chamber ICDs were considered: they may present increased risk of PM-onset vs dual and three-chamber ICDs [Sweeney et al. 2007]. About the pharmacological treatment, the only detailed information was referred to implant date: eventual changes, during the follow-up period, were not collected.
Moreover, to examine the basal heart rhythm before the VT initiation, a good quality EGM at 250 Hz was obtained. As a consequence, for technical reasons, the ICD diagnostic parameters were programmed to store at the most three VT episodes. This setting, however, could have caused a lack of some episodes. For instance, in a DCM patient with some VT storms, successfully treated with the anti-tachycardia pacing, it was not possible to analyze

all the episodes, but only the latest ones. In order to limit these drawbacks, the same settings were used for CAD and DCM groups and the follow-up was performed periodically, (minimum of 3 to maximum 6 months) unless the ICD delivered a shock. In that case, the patient was invited to contact the hospital for a visit as soon as possible and data were retrieved by the ICD. It is authors' opinion that the frequent follow-up could be sufficient to minimize the number of undetected VT episodes.

5. Conclusion

This study examines the differences between CAD and non-ischemic DCM patients with ICD. Our results show that patients with DCM exhibit a significantly higher prevalence of VT episodes ($p<0.02$) and a significantly greater variability in the VT initiation pattern ($p<0.01$). The circadian distribution (in a 3 –hour period analysis) of the VT-onset is also different in the two groups: CAD patients exhibit a morning peak: between 9 and 12 a.m. and an afternoon peak (around 6 p.m.), whereas DCM patients show a more uniform distribution during waking hours and very few episodes during the night.
Eventually, no significant differences between CAD and DCM are observed from the analysis of VT-rate, coupling interval, prematurity index. Likewise, the analysis of the EGM signal during the 20 seconds immediately preceding VT onset show no significant differences in the heart-cycle, prevalence of PVC, nor prevalence of paced beats.
This study (based on characterization of EGM, VT-onset and VT circadian distribution) does not define a relationship between observed CAD-DCM differences and underlying CAD-DCM electrogenetic-mechanisms. Nevertheless, our findings suggest different foundations of VT initiation in patients with ischemic versus idiopathic dilated cardiomyopathy.

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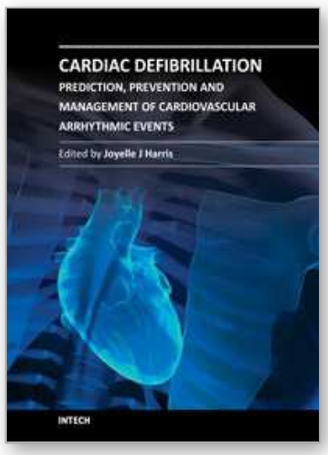

Cardiac Defibrillation - Prediction, Prevention and Management of Cardiovascular Arrhythmic Events

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Millions of people throughout the world currently depend on appropriate, timely shocks from implantable cardioverter defibrillators (ICDs) to avoid sudden death due to cardiovascular malfunctions. Therefore, information regarding the use, applications, and clinical relevance of ICDs is imperative for expanding the body of knowledge used to prevent and manage fatal cardiovascular behavior. As such, the apt and timely research contained in this book will prove both relevant to current ICD usage and valuable in helping advance ICD technology. This book is divided into three comprehensive sections in order to cover several areas of ICD research. The first section introduces defibrillator technology, discusses determinants for successful defibrillation, and explores assessments of patients who receive defibrillation. The next section talks about predicting, preventing, and managing near catastrophic cardiovascular events, and research presented in the final section examine special cases in ICD patients and explore information that can be learned through clinical trial examinations of patients with defibrillators. Each chapter of this book will help answer critical questions about ICDs.

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