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Rotor Stability Separates Sustained Ventricular Fibrillation From Self-Terminating Episodes in Humans

David E. Krummen, MD, FACC[1,2], **Justin Hayase, MD**[1,2], **David J. Morris, MD**[1,2], **Jeffrey Ho, MD**[1,2], **Miriam R. Smetak, BS**[2], **Paul Clopton, MS**[2], **Wouter-Jan Rappel, PhD**[1], and **Sanjiv M. Narayan, MD, PhD, FACC**[1,2]

[1]University of California San Diego, San Diego, CA

[2]Veterans Affairs San Diego Healthcare System, San Diego, CA

Abstract

Objective—We mapped human ventricular fibrillation (VF) to define mechanistic differences between episodes requiring defibrillation versus those that spontaneously terminate.

Background—VF is a leading cause of mortality, yet episodes may also self-terminate. We hypothesized that the initial maintenance of human VF is dependent upon the formation and stability of VF rotors.

Methods—We enrolled 26 consecutive patients (age 64±10 years, n=13 with LV dysfunction) during ablation procedures for ventricular arrhythmias, using 64-electrode basket catheters in both ventricles to map VF prior to prompt defibrillation per IRB-approved protocol. Fifty-two inductions were attempted and 36 VF episodes were observed. Phase analysis was applied to identify bi-ventricular rotors in the first 10 seconds or until VF terminated, whichever came first (11.4±2.9 seconds to defibrillator charging).

Results—Rotors were present in 16 of 19 patients with VF, and in all patients with sustained VF. Sustained, but not self-limiting VF, was characterized by greater rotor stability: (1) rotors were present in 68±17% of cycles in sustained versus 11±18% of cycles in self-limiting VF ($p<0.001$); with (2) maximum continuous rotations greater in sustained (17±11, range 7–48) versus self-limiting VF (1.1±1.4, range 0–4, $p<0.001$). Additionally, biventricular rotor locations in sustained VF were conserved across multiple inductions (7/7 patients, $p=0.025$).

Conclusions—In patients with and without structural heart disease, the formation of stable rotors identifies individuals whose VF requires defibrillation from those in whom VF spontaneously self-terminates. Future work should define the mechanisms that stabilize rotors and evaluate whether rotor modulation may reduce subsequent VF risk.

Correspondence to: David E. Krummen, MD, 3350 La Jolla Village Drive, Cardiology Section 111A, San Diego CA, 92161, Office: 858-642-3889, Fax: 858-552-7490, dkrummen@ucsd.edu.

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Keywords

arrhythmia mechanisms; electrical rotors; electrophysiology; ventricular fibrillation

Introduction

Ventricular fibrillation (VF) is a common, life-threatening arrhythmia and a major cause of the 700,000 cases of sudden cardiac death in the US and Europe annually (1). Although our understanding of VF mechanisms continues to improve (2), we still do not fully understand the mechanistic differences between VF episodes which perpetuate and those which spontaneously terminate (3).

Superficially, VF appears to be random and disorganized. However, significant progress has been made to identify deterministic features within VF (4,5). Detailed epicardial mapping suggests the co-existence of electrical rotors and disorganized activity in induced VF in patients with preserved ventricular function during open heart surgery (6). However, the importance of rotors and other propagation patterns to the maintenance of human VF remains uncertain. VF rotors have been studied in the context of ischemia (7) and scar (8) using animal models and explanted human hearts, yet these studies have not explained why some VF episodes require defibrillation while others self-terminate without consequence.

Prior work has shown the presence of rate gradients (9) in sustained VF, supporting the concept of spatial preferences for VF drivers. Subsequent work evaluating surface ECG patterns found evidence for repetitive spatial paths of VF sources (10). More recent studies have shown evidence that electrical rotors predominantly associate with areas of scar (11). Based upon these data, we hypothesized that greater electrical rotor stability would predict the perpetuation of early human VF and its progression to sustained VF.

Methods

Patient Enrollment

In this prospective study of the relationship between VF rotors and duration, we enrolled consecutive patients presenting for ventricular arrhythmia ablation at the University of California San Diego and VA San Diego Healthcare System. The protocol was approved by a joint UCSD/VA institutional review board (IRB), and all patients provided written, informed consent after a full discussion of risks and potential benefits. Exclusion criteria included the presence of ventricular thrombus, hemodynamic instability precluding the safe induction of VF, and unrevascularized coronary ischemia.

Antiarrhythmic drugs (mexilitine=2, amiodarone=1, dronedarone=1, and sotalol=6) were discontinued greater than 5 half-lives (6 weeks for amiodarone) prior to electrophysiology study. LV function was assessed by transthoracic echocardiography prior to the procedure.

Study Protocol

The study protocol is summarized in figure 1. Patients were intubated, ventilated and maintained under a consistent general anesthetic protocol. A decapolar catheter was placed

in the coronary sinus, and a quadripolar catheter was placed in the right ventricle (RV) for VF induction. Invasive arterial pressure and vital signs were monitored continuously throughout the case.

Basket catheters 64-electrode, Constellation, Boston Scientific, Natick, MA) were advanced for simultaneous recording into the RV and left ventricle (LV) either by retrograde aortic (figure 2A–B) or transseptal (figure 2C) approaches, as best suiting the clinical procedure. Basket catheter contact was evaluated by: (1) evaluating fluoroscopic basket catheter morphology to ensure uniform deformation by cineangiography (figure 2A–C), (2) imaging with intracardiac ultrasound, and (3) ensuring that electrogram amplitude both at baseline and during VF was acceptable. Electrodes with noisy or low amplitude signals (<0.5 mV) were excluded from analysis and the corresponding areas on phase mapping left blank; on average 10±7 electrodes (7.8%) were excluded in each case due to suboptimal contact or noise.

VF Induction

Following baseline programmed ventricular stimulation, rapid pacing was performed for 15 seconds, followed by a 1 minute recovery period, for each cycle length (CL) of 350, 300, 250, then decrementing by 10 msec until VF induction (figure 2D) or 2:1 capture (minimum CL 170 msec) per protocol, similar to prior work (12). As soon as VF was induced, defibrillator charging commenced, and VF was recorded during this charging period. VF was defibrillated as soon as charging was complete (11.4±2.9 seconds; range 8–15 seconds). After a 5 minute waiting interval, a second episode of VF was induced in each patient either with a second burst pacing induction or 3.2 seconds of rapid pacing followed by a 2 Joule T-wave shock (in patients with ICDs). VF was defined as varying ECG morphology with a rate >220 beats per minute as previously defined (8). Following the second attempted VF induction, the clinical procedure was commenced in routine fashion.

Electrogram Analysis

Unipolar electrograms (Bard Pro, Billerica, MA) were recorded at 1000 Hz and filtered from 0.05 to 500 Hz. Multipolar basket electrograms were analyzed offline using software that we have developed and described previously (13) and optimized for VF analysis, using phase analysis (14) of unipolar electrograms (6), within physiologic constraints (15,16). Data were analyzed for the first 10 seconds of VF or until termination, whichever came first.

Rotational activity was identified as a phase singularity formed at the intersection of depolarization and repolarization isolines (4) consisting of at least 1 rotation (figure 3). Rotors were defined as regions of rotational activity that controlled surrounding activation, and the criteria for numbers of rotations in human VF were derived in this study. Regions of centrifugal propagation without rotation were defined as focal activation (figure 4A–B). Continuous, disorganized ventricular activation without a clear rotational or focal activation (‘fibrillatory conduction’; figure 4C–D) was also documented.

Measuring Rotor Prevalence and Stability

We quantified the prevalence of rotational activity as the percent of VF cycles showing such activity, with stability quantified as the maximum number of consecutive revolutions of electrical activity within a region bounded by 2 electrodes in each axis. We performed receiver-operating characteristic (ROC) analysis to determine criteria for prevalence and stability which functionally separated sustained from self-limiting episodes of VF.

Modeling Endocardial Recording of Non-Endocardial VF Sources

To explore the endocardial projection of non-endocardial VF sources, we created a 3-dimensional computational model of a hairpin-shaped rotor filament with both ends terminating on the epicardium. The Barkley model (17) was implemented on 200×100×100 grid, and the filament was initiated as previously described (18). Additional details may be found in the online supplement.

Statistical Methods

Continuous variables are expressed as mean±standard deviation. The Student *t* test was used to compare continuous variables; the Fisher exact test was used to compare nominal variables. The ROC cutpoints were determined by optimization of the Youden index. The relationship between rotor stability and ejection fraction (EF) was calculated using Pearson correlation. Subject-wise and episode-wise statistics are indicated. For episode-wise comparisons, repeated measures analysis of variance was used to determine differences between self-limited and sustained VF episodes. The Bonferroni correction was applied for planned multiple comparisons. The paired t-test was used in the analysis of patients with both sustained and self-limited VF. Statistics were calculated using SPSS 19 (IBM, Somers, NY, USA).

Results

We enrolled 26 patients (13 with LV EF < 50%, age 64±10 y); demographics are shown in table 1. There were no thromboembolic complications or other adverse events during the study.

VF Induction

A total of 52 VF induction attempts were performed per IRB-approved protocol resulting in 36 episodes of VF (CL 210±26). Other VF induction attempts yielded monomorphic ventricular tachycardia (VT, n=8) and no ventricular arrhythmia (n=8) and were excluded from analysis. There were no significant differences in cycle length between pacing-induced and shock-induced VF (see online supplement for additional details and results). Of VF episodes, 21 lasted ≥8 seconds (“sustained VF”) and required defibrillation (duration 11.4±2.9 seconds), and 15 were self-limited (duration 3.9±1.4 seconds).

The demographics of patients with sustained and self-limited VF are shown in table 2. Cycle length was similar for sustained VF (203±25 msec) and self-limited VF (216±21 msec, p=0.08). Patients with self-limited VF had higher LV EF than those with sustained VF.

Ischemic cardiomyopathy was more common in patients with sustained VF (50%) than without (0%, p=0.03).

Rotors in VF

Localized sites of rotational activation were seen in 16 of 19 patients with VF (89%), and in all patients with sustained VF (10/10, 100%), in whom sustained rotors of longer prevalence and stability were found. Figure 3A and online movie 1 show a LV counterclockwise rotor during induced VF in a 73 year old patient with an EF of 23% presenting for first VT ablation. This rotor was mapped over 15 rotations; VF required defibrillation to terminate. Electrograms showing sequential activation around a core, spanning >80% of the VF cycle are shown in figure 3B. Vector analysis of the subsequent VF cycle (3C) shows stable activation around the core with wavefront spread to more distant tissue, controlling ventricular activation. In figure 3A, right ventricular activation is passive, consistent with transseptal conduction.

Spatial Conservation of Stable Rotors over Multiple VF Inductions

Stable VF rotors in sustained VF were conserved over multiple inductions: there were 7 patients in whom 2 episodes of sustained VF were induced. In each, rotor sites were conserved within 1 electrode radius (7/7 patients, p=0.023). Online movies 2 and 3 show sequential VF episodes in a 68 year old patient with recurrent VT in which the rotor recurs in the posteroseptal LV. In contrast, focal sources locations were infrequently conserved (2/10 patients with conserved focal source sites, p=NS).

Differences in Rotor Prevalence between Sustained and Self-Limited VF

Rotors were more prevalent in sustained VF; they were present for 68±17% of VF cycles in sustained VF versus 11±17% in self-limited VF (p<0.001). ROC analysis for rotor prevalence and VF outcome demonstrate that a cutoff of ≥45% of VF cycles showing rotors separated sustained from self-limited VF with 100% sensitivity and 93% specificity (figure 5A).

Focal and Disorganized Activation Patterns in VF

Figure 4A–B and movie 4 show an example of focal activation in a 68 year old patient with idiopathic cardiomyopathy (LV EF 32%), located in the anteroseptal LV during VF (CL 222 msec). Figure 4B shows basket electrograms with activation spanning only 45% of the VF cycle. VF terminated spontaneously after 4 seconds. Figure 4C–D and movie 5 show disorganized activation in a 52 year old patient with frequent, symptomatic PVCs and an EF of 69% during VF. Basket electrograms show disorganized activation spanning each VF cycle.

Figure 5B shows the prevalence of rotors and alternative activation patterns for all VF episodes. Notably, focal activity comprised a greater proportion of VF cycles in self-limited VF (78±29%) versus sustained VF (9±9%, p<0.001). Unlike rotors, focal sources were infrequently spatially conserved; 2 of 12 focal sources (17%, p=NS) were located within 1 inter-electrode separation between VF episodes. Disorganized activation (fibrillatory

conduction) was similarly prevalent between self-limited VF (23±16%) and sustained VF (10±15%, p=0.1).

Differences in Rotor Stability between Sustained and Self-Limited VF

Rotors in sustained VF persisted for more consecutive VF cycles (17±11 cycles, range 7–48) than rotors in self-limited VF (1.1±1.4 cycles, range 0–4, p<0.001). Figure 6A–B shows a sustained VF episode in which the rotor completes 17 rotations. This VF episode required defibrillation (fig 6B). Figure 6C shows self-limited VF with focal activation and no rotor. A cutoff of 7 consecutive rotations effectively separates sustained and self-limited VF episodes (fig 5C). Figure 5D shows temporal rotor stability for sustained and self-limited VF.

Patients with both Sustained and Self-Limited VF

There were 4 patients in whom sustained and self-limited VF were observed in separate episodes. Similar to the overall population, rotors were more prevalent (50±14% vs 8±15% of rotations, p=0.032) and more stable (15±10 vs 2±2 consecutive rotations, p=0.04) in sustained vs self-limited VF, respectively, for this subgroup.

Rotor Stability, Ventricular Substrate and Location

We found that VF rotor duration was negatively correlated with LV function (correlation coefficient 0.58, p=0.037). Although the majority of rotors and focal sources were found in the left (67%) versus right ventricle (33%), the difference was not statistically significant.

Endocardial Mapping of Non-Endocardial VF Sources

In our simulations, non-endocardial rotors presented endocardially as focal activation. Additional details may be found in the online supplement.

Discussion

There are three main findings of the present study. First, rotational activation is common in human VF, and stable rotors are common in sustained VF requiring defibrillation, which is most likely to result in clinical sequelae. Second, rotors lie in conserved areas for successive VF inductions, suggesting that rotors form in regions of favorable structural and functional substrate. Third, focal activation without stable rotors is more prevalent in self-limiting VF, and may thus reflect absence of this substrate. Importantly, these differences are seen in the subset of patients in whom both sustained and self-limited VF episodes were induced, and thus these findings are independent of substrate. These findings motivate studies to define the mechanisms underlying rotor formation, to explore the feasibility of localized therapies such as ablation, pre-emptive pacing, or cellular therapy to prevent VF.

The Importance of Rotors to VF Perpetuation

Multiple mechanisms have been observed in ongoing VF: sustained rotors (4,5) and focal sources (2) in canine ventricles; transient rotors and multiple wavelets in human ventricles (6). A central question is whether these mechanisms differ between VF that terminates spontaneously, and episodes which progress to sustained VF and result in symptoms, syncope, and sudden death.

In this work, we studied a spectrum of patients with ventricular arrhythmias. We found that rotor prevalence ≥45% of mapped cycles and stability ≥7 cycles identified VF that was sustained and required cardioversion from episodes that terminated spontaneously. While such cutoffs are somewhat arbitrary, since the mechanisms for and risk of VF likely form a spectrum, they re-emphasize that the formation of stable rotors is important to VF maintenance. Perhaps the most compelling support for the importance of stable rotor formation comes from the subgroup with both sustained and self-limited episodes. Despite identical substrate between episodes, significant differences in rotor stability were observed between the episode that required cardioversion and the episode that spontaneously terminated. Thus rotor stabilization is at least a hallmark of, and possibly a critical step in the transition of early VF to sustained VF.

Notably, the average rotor prevalence and stability in sustained VF was ~70% and 17 rotations, respectively. These values differ from prior work, but important differences must be considered. First, many prior studies used animal models (4,9,19), explanted human hearts supported by Langendorf-perfusion (8,11), or subjects undergoing open heart surgery (6,7). As shown by Qin and colleagues, such techniques may alter VF mechanisms (20). Our study employed multielectrode mapping via a percutaneous approach in patients, more closely approximating physiologic conditions. Additionally, we sampled a large proportion of the endocardium of both ventricles, including both sides of the interventricular septum, using biventricular basket catheters. Such catheters have previously been used to study VF in animal models (21–23), and patients (24). Second, we quantified ventricular activation patterns in early VF. Different mechanisms may predominate later in VF due to progression of ischemia and electrical remodeling.

Structural Determinants of VF Rotor Sites

Prior work has shown the dependence of rotors upon myocardial scar (8). A subsequent study supports the observation that rotors tend to more frequently localize at diseased substrate (11). Our work is in agreement with these findings, noting that the majority of sustained VF episodes, with more stable rotors, were found in patients with LV dysfunction. Ischemic cardiomyopathy, in particular, was more common in patients with sustained VF. However, the presence of structural heart disease or EF alone, were imperfect predictors of sustained VF; several patients without LV dysfunction had VF progress to sustained, clinically significant episodes.

Importantly, we found that sustained VF rotor locations were conserved between episodes. Such spatial conservation implies that structural factors or fixed spatial distributions of functional gradients determine rotor locations. In a separate case report (25), we have previously described a patient in whom VT ablation incidentally coincided with the primary VF rotor site. He then presented with recurrence of VT several months later, and again consented to the study protocol, which was then unable to induce VF, only monomorphic VT. Based on these findings, future studies should examine if targeted intervention may reduce the probability of sustained VF (19).

Future Directions: Rotor Sites As Therapeutic Targets

As with other arrhythmias, intervention in VF is possible at multiple points, including initiating triggers and sustaining sites. Previously, ablation of Purkinje-related premature ventricular contraction (PVC) triggers has been shown to decrease VF episodes in studies of patients without structural heart disease (26). However, the role of triggers in initiating VF in patients with structural heart disease is less clear (27), while sustaining mechanisms for VF are acknowledged as the predominant driver for sudden death in all patients. Inspired by animal models showing stable VF rotors (4), and by recent work in which stable atrial fibrillation rotors sites were successfully identified and ablated in real time (28), we hypothesize that VF rotors may be suitable targets for ablation to decrease ICD shocks for patients with recurrent VF.

Limitations

First, this study was limited by the spatial resolution of the electrode spacing (4–5 mm interelectrode, 10 mm interspline) of the basket catheters. However, from wavelength considerations, the minimum APD in human ventricle is ~140 ms (15) and minimum conduction velocity approximately 40 cm/second (16), providing a minimum circumference of approximately 7 cm (2 cm diameter). Thus, resolution should be sufficient to resolve such rotors. Second, only the endocardium was mapped. Based upon our simulation studies and others (29), endocardial mapping may misclassify non-endocardial rotors as focal sources, and thus underestimate the true prevalence of VF rotors. However, only a minority (17%) of mapped focal sources in this study displayed characteristics consistent with rotors. Furthermore, prior work in explanted human hearts has shown the prevalence of intramyocardial rotors is low (8), and animal models of VF have shown that endocardial intervention altered VF inducibility (19), consistent with the hypothesis that the endocardium is important for early VF maintenance. As a result, we believe that endocardial mapping in this study may underdetect <20% of rotors in early VF. Third, we enrolled a heterogeneous group of patients by design, using protocol-driven VF inductions to examine mechanistic differences in outcome. That differences between sustained and self-limited VF were consistent across patients regardless of LV function and induction type supports the generalizability of our findings. Fourth, VF was induced by rapid pacing and shock-on-T, and differences in ischemic time may have influenced VF mechanisms. However, the total difference in ischemic time (~11 seconds) was significantly below the 30 second threshold for ischemia to significantly alter VF (7). Furthermore, we were unable to identify differences in VF rate, regularity, number of rotors, or rotor duration (see online supplement) between these induction methods. Fifth, for practical reasons we could study only early VF in this procedural model. However, early VF is of critical importance because patients may become symptomatic and experience syncope or ICD shocks during early VF, and because early VF initiates the cascade leading to sustained VF. Sixth, we did not routinely ablate rotor sites and retest VF inducibility to prove that such sites are critical for the maintenance of VF as we have for atrial rotor sites (30), although we have reported a case (31) in which that did occur, and VF was subsequently not inducible with the study pacing protocol. Future studies of VF rotor ablation are planned. Seventh, artifact during VF may have created the appearance of rotors. However, such artifact was not seen during rapid

pacing prior to VF. Finally, the sample size of the study is small, which may limit the generalizability of our findings.

Conclusions

Rotor prevalence and stability separate sustained and self-limited VF. Rotor sites in sustained VF are conserved, and rotor stability is inversely correlated with LV function, implicating a dependence on a localized pro-arrhythmic substrate. Future studies should determine the conditions under which stable rotors form, and whether such sites may be safely modulated in humans to reduce subsequent VF risk.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

CL	Cycle Length
EF	Ejection Fraction
ICD	Implantable Cardioverter Defibrillator
IRB	Institutional Review Board
LV	Left Ventricle
ROC	Receiver Operating Characteristic
RV	Right Ventricle
VT	Ventricular Tachycardia
VF	Ventricular Fibrillation

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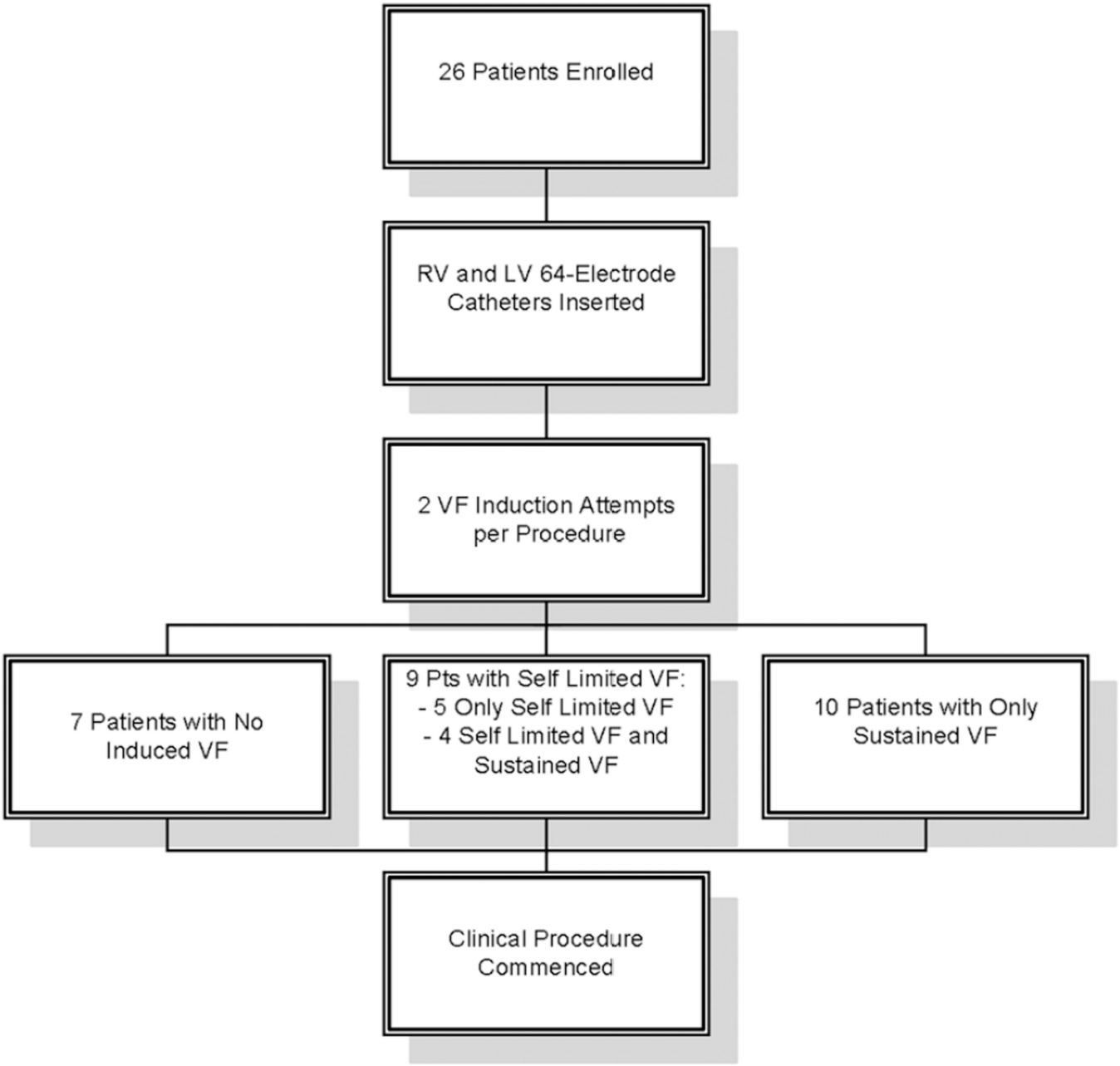

Figure 1. Study Protocol

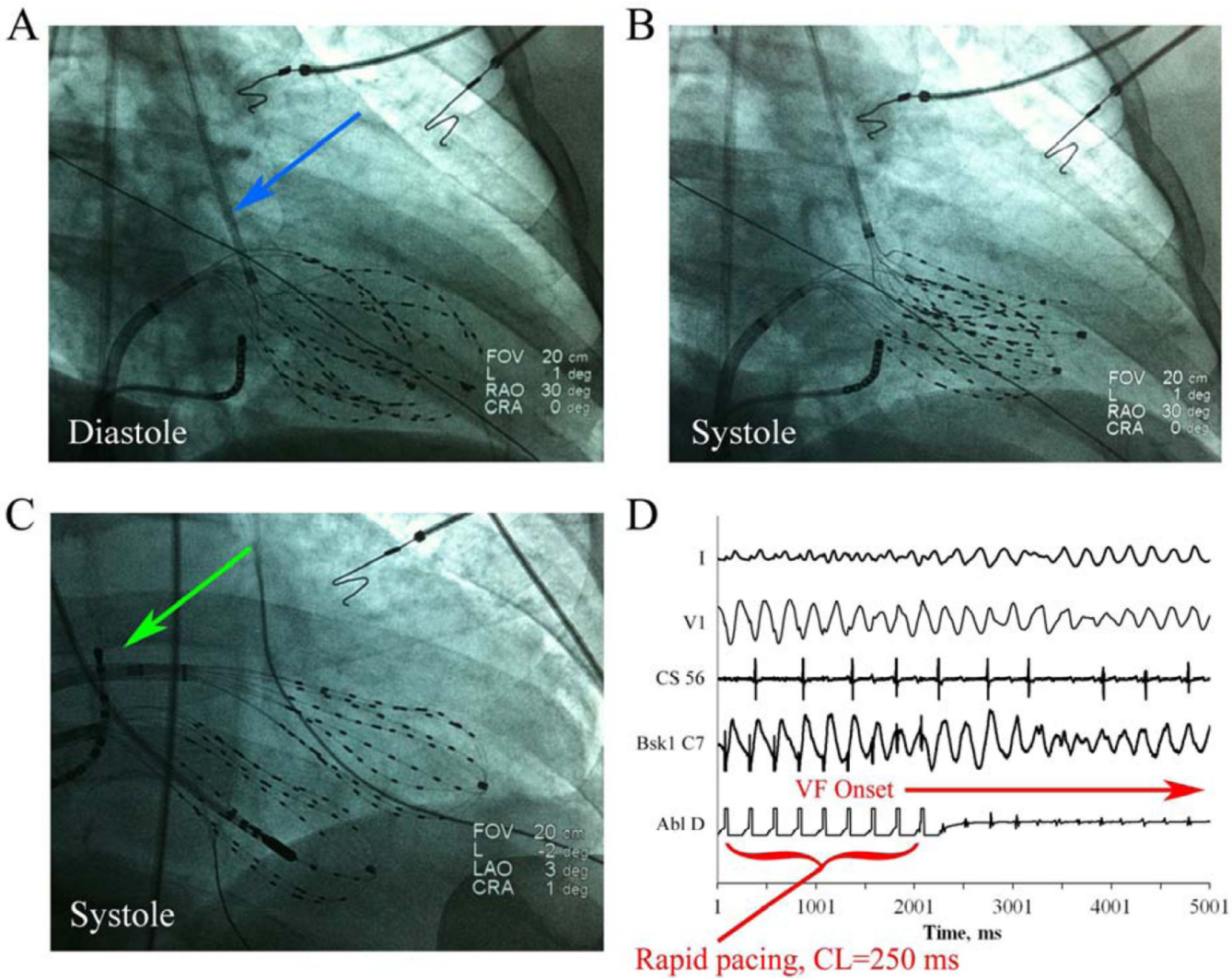

Figure 2. Biventricular mapping and VF induction

Right anterior oblique fluoroscopy of biventricular baskets during diastole (A) and systole (B) in a 51 year old patient with a normal LV EF, mild RV dysfunction, and symptomatic VT and PVCs. The LV basket was advanced via the retrograde aortic approach (blue arrow). (C) Antero-posterior fluoroscopy showing biventricular baskets in a 68 year old patient during systole in which the LV basket was advanced via transseptal catheterization (green arrow). (D) VF induction by protocol-driven rapid pacing (250 msec) showing surface ECG (I and V1) and intracardiac electrograms (CS56, LV basket C7, and ablation distal).

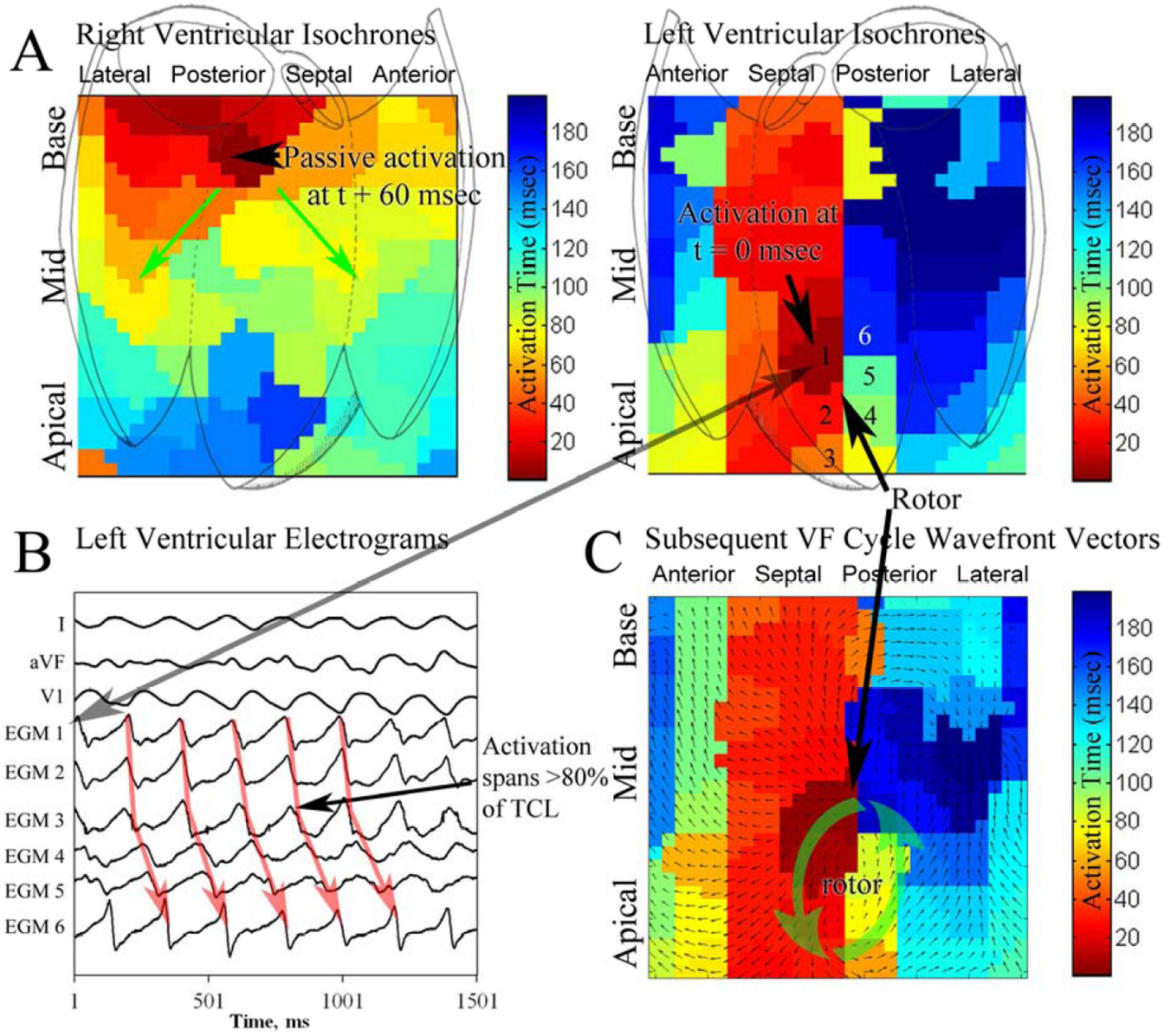

Figure 3. Biventricular VF analysis showing LV rotor

(A) Isochronal analysis of the RV and LV during ventricular fibrillation in a 73 year old patient, EF 25%, presenting for drug-refractory ventricular tachycardia. LV isochrones show a rotor (CL 220 msec) in the septal LV. This rotor persisted for 15 continuous rotations; rotor activity was seen in 72% of all VF cycles in this patient. (B) Basket electrograms during VF, numbered (1–6) near the rotor core. Note that activation spans <80% of the VF cycle length. (C) Wavefront vector analysis of the subsequent VF cycle, showing consistent rotation about the core with radial activation of distant tissue.

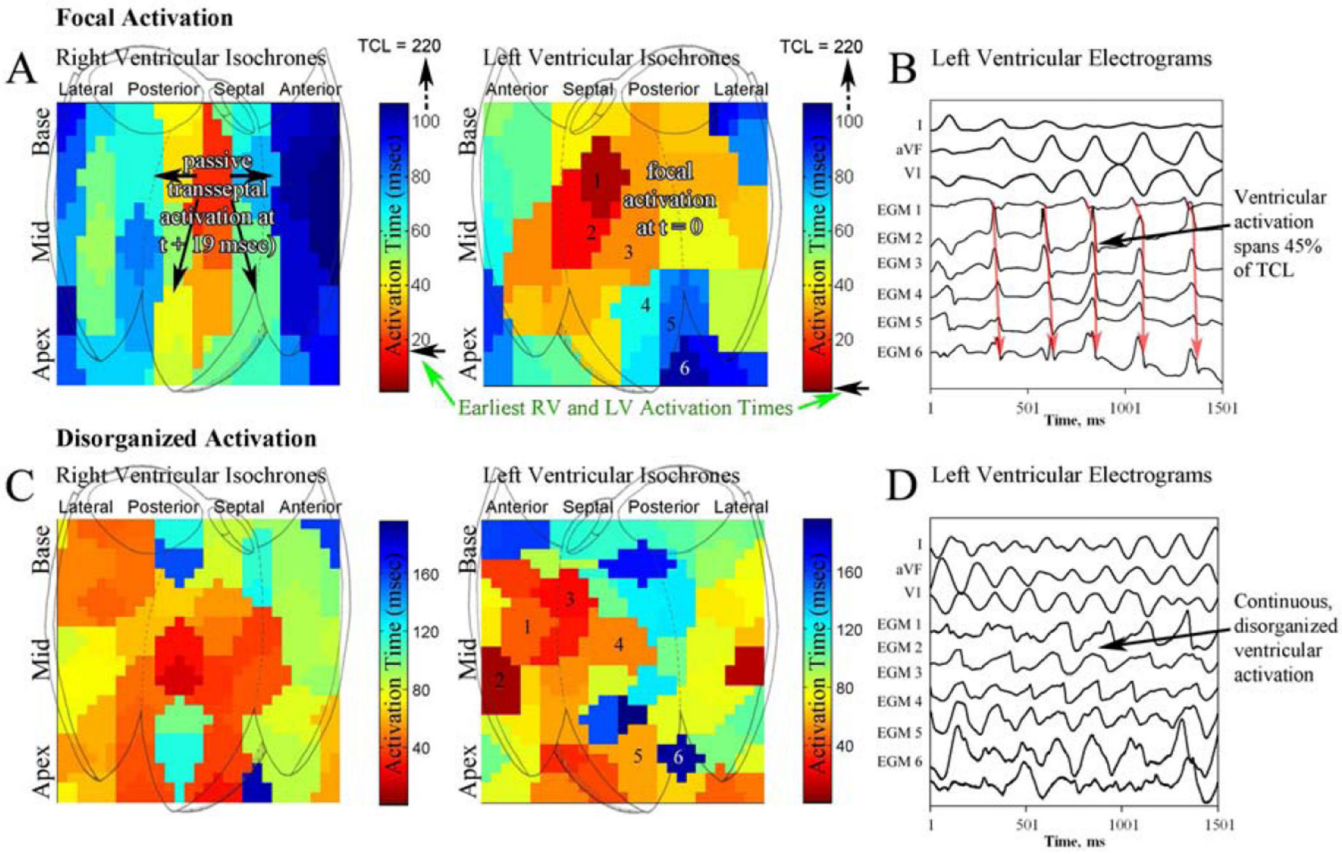

Figure 4. Focal and disorganized activation patterns during VF

(A) Biventricular isochronal analysis of ventricular fibrillation in a 68 year old patient with idiopathic cardiomyopathy, ejection fraction of 32%, showing a focal source in the anteroseptal LV with passive activation of the RV beginning 19 msec after LV activation. (B) Electrograms at increasing distance from the focal source. Note that LV endocardial activation spans approximately 45% of the VF cycle length. (C) Disorganized biventricular activation without a stable rotor during VF in a 52 year old patient with a normal EF. (D) Intracardiac electrograms are varying and chaotic, spanning the VF cycle.

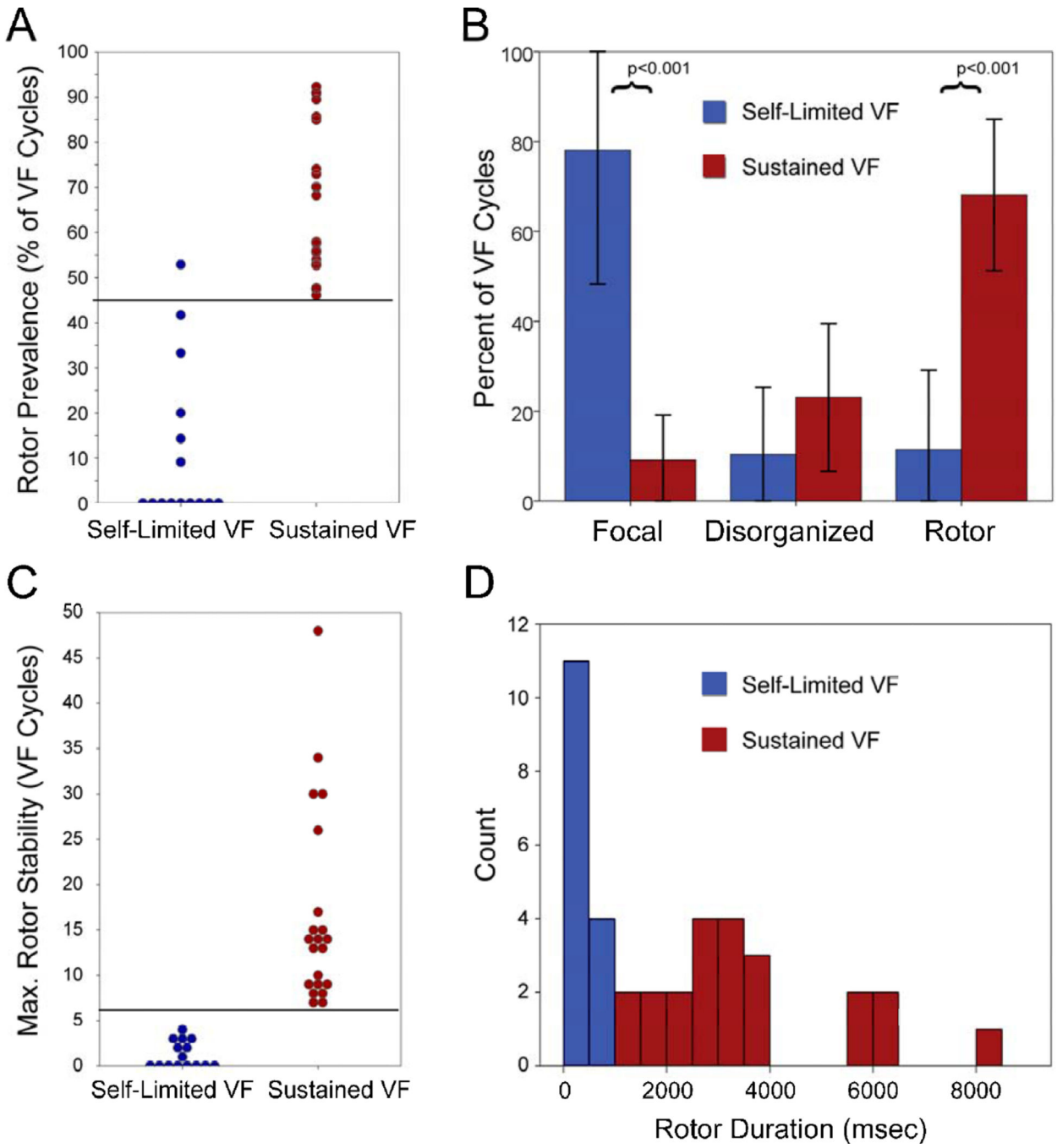

Figure 5. Activation patterns in sustained versus self-limited VF

(A) Rotor prevalence for each VF episode; prevalence ≥45% separates sustained from self-limited VF with 100% sensitivity and 93% specificity. (B) Rotor, disorganized and focal activation prevalence for the study. Rotor and focal activation prevalence were different between sustained and self-limited VF. (C) Maximum rotor stability (consecutive rotations) for each episode: sustained and selflimited VF populations do not overlap. (D) Histogram of temporal rotor stability (milliseconds) for sustained and self-limited VF. When rotors persisted <1 second, VF perpetuated and required defibrillation.

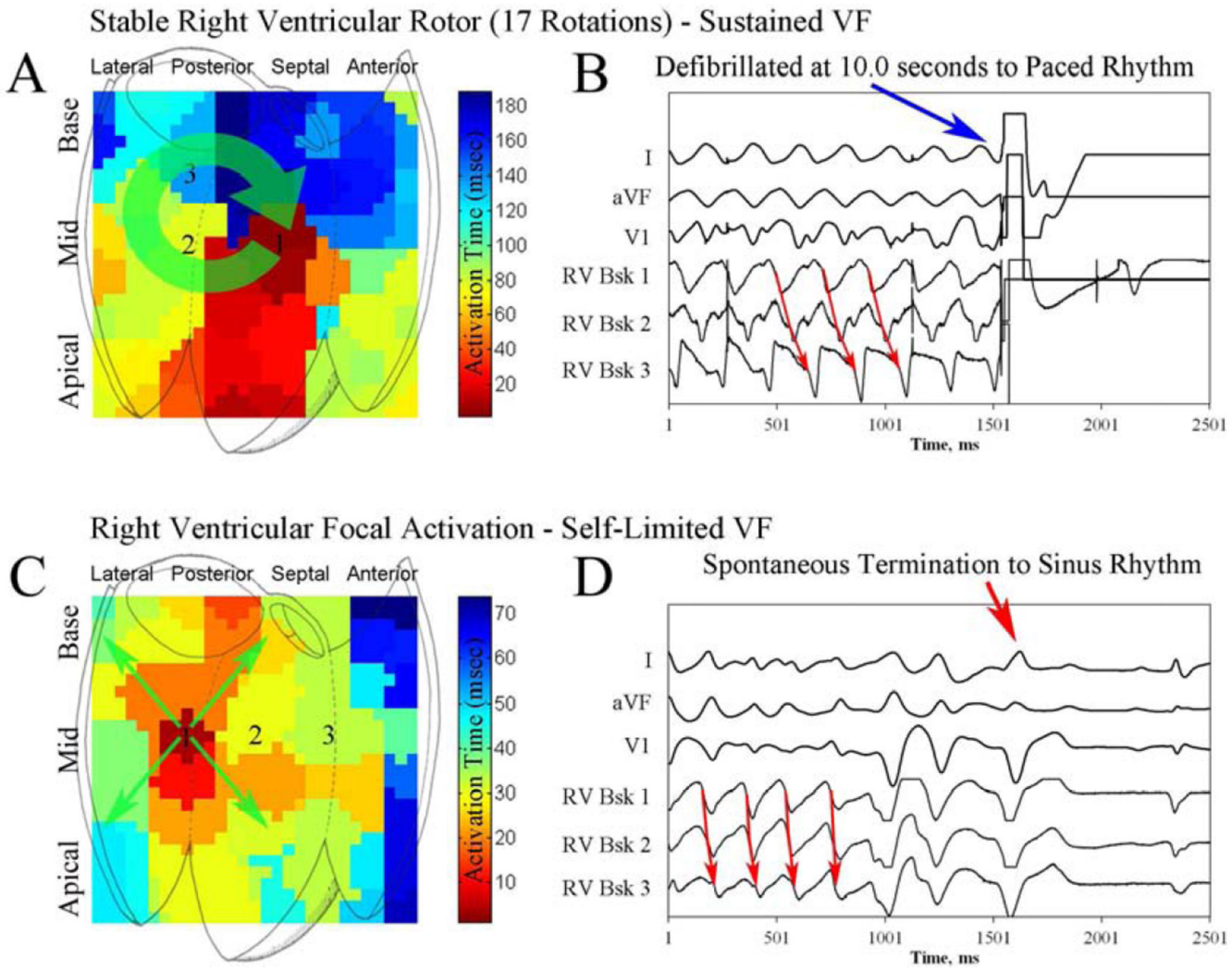

Figure 6. Right ventricular rotor and focal activation during VF

(A) Isochronal analysis of VF showing a right ventricular rotor in a 79 year old patient with ischemic cardiomyopathy. Maximum rotor stability was 17 rotations, and rotor prevalence was 91%. (B) This VF episode was defibrillated (right side) 10 seconds after induction. (C) VF isochronal analysis demonstrating a right ventricular focal source in a 68 year old patient with preserved left ventricular function. (D) VF terminated spontaneously.

Table 1

Study Demographics

Characteristics	Preserved EF	LV Dysfunction	p Value
n	10	12	-
Age (years) ±SD	62±13	67±7	0.27
Left atrial diameter, mm±SD	36±5	45±12	0.08
Left ventricular EF (%)	65±8	33±8	**0.001**
Hypertension (%)	6(60)	10(83)	0.35
Diabetes mellitus (%)	3(30)	3(25)	1
Hyperlipidemia (%)	8(80)	10(83)	1
Coronary disease (%)	6(60)	6(50)	0.69
Prior Myocardial Infarction (%)	3(30)	4(33)	1
Prior PCI (%)	3(30)	4(33)	1
CABG (%)	1(10)	3(25)	0.59
COPD (%)	1(10)	1(8.3)	1
Medications			
Beta-Blocker (%)	7(70)	11(92)	0.29
ACEI/ARB (%)	4(40)	10(83)	0.07
Digoxin (%)	1(10)	4(33)	0.32
Calcium Channel Blockers (%)	1(10)	3(25)	0.59
Mexilitine (%)	0	1(8)	1
Amiodarone (%)	0	0	1
Sotalol (%)	1(10)	5(42)	0.16
Dofetilide (%)	0	0	1
Coumadin (%)	2(20)	5(42)	0.38
Aspirin (%)	5(50)	4(33)	0.67
Statin (%)	7(70)	7(58)	0.68

Table 2

Demographics of Patients with Sustained and Self-Limited VF

Characteristics of Pts with VF	Self-Limited VF	Sustained VF	p Value
n	9	10	-
Age (years) ±SD	64±8	67±7	0.37
Left atrial diameter, mm±SD	39±9	44±13	0.45
Left ventricular EF (%)	52±14	32±9	**0.002**
Hypertension (%)	8(89)	8(80)	1
Diabetes mellitus (%)	3(33)	2(20)	0.63
Hyperlipidemia (%)	7(78)	9(90)	0.58
Coronary disease (%)	5(56)	6(60)	1
Prior Myocardial Infarction (%)	2(22)	5(50)	0.35
Prior PCI (%)	5(56)	2(20)	0.17
CABG (%)	2(22)	4(40)	0.63
COPD (%)	0	2(20)	0.47
Cardiomyopathy, EF<50%	3(33)	10(100)	**0.003**
Etiology: Ischemic CMP	0	5(50)	**0.03**
Etiology: Nonischemic CMP	3(33)	5(50)	0.65
Medications			
Beta-Blocker (%)	7(78)	9(90)	0.58
ACEI/ARB (%)	6(67)	8(80)	0.63
Digoxin (%)	0	4(40)	0.09
Calcium Channel Blockers (%)	1(11)	2(20)	1
Mexilitine (%)	0	2(20)	0.47
Amiodarone (%)	0	1(10)	1
Dronedarone (%)	0	0	1
Sotalol (%)	0	5(50)	**0.03**
Dofetilide (%)	0	0	1
Warfarin (%)	1(11)	5(50)	0.14
Aspirin (%)	5(56)	4(40)	0.66
Statin (%)	7(78)	6(60)	0.63