SSN 0725 1007/06/\$22.00

ISSN 0735-1097/06/\$32.00 doi:10.1016/j.jacc.2005.12.062

# Isolated Potentials During Sinus Rhythm and Pace-Mapping Within Scars as Guides for Ablation of Post-Infarction Ventricular Tachycardia

Frank Bogun, MD, FACC, Eric Good, DO, Stephen Reich, MD, Darryl Elmouchi, MD, Petar Igic, MD, Kristina Lemola, MD, David Tschopp, MD, Krit Jongnarangsin, MD, Hakan Oral, MD, FACC, Aman Chugh, MD, Frank Pelosi, MD, FACC, Fred Morady, MD, FACC

Ann Arbor, Michigan

OBJECTIVES	The purpose of this study was to identify ventricular tachycardia (VT) isthmus sites by pace-mapping within scar tissue and to identify electrogram characteristics that are helpful in identifying VT isthmus sites during sinus rhythm (SR).
BACKGROUND	Pace-mapping has been used in the scar border zone to identify the exit site of post- infarction VT.
METHODS	In 19 consecutive patients (18 men, mean age $66 \pm 9$ years, mean ejection fraction $0.24 \pm 0.12$ ) with post-infarction VT, a left ventricular voltage map was generated during SR. Pace-mapping was performed at sites with abnormal electrograms or isolated potentials. Radiofrequency ablation was performed at isthmus sites as defined by pace-mapping (perfect pace-map = $12/12$ matching electrocardiogram leads; good pace-map = $10/12$ to $11/12$ matching electrocardiogram leads) and/or entrainment mapping
RESULTS	A total of 81 VTs (mean cycle length 396 $\pm$ 124 ms) were inducible. In 16 of the 19 patients, a total of 81 VTs (mean cycle length 396 $\pm$ 124 ms) were inducible. In 16 of the 19 patients, a total of 41 distinct isthmus areas of 41 distinct VTs were identified and successfully ablated. All but one displayed isolated potentials during SR. Furthermore, 22 of the 81 VTs (27%) for which no isthmus was identified became noninducible after ablation of a targeted VT. The 16 patients in whom $\geq$ 1 isthmus was identified and ablated were free of arrhythmic events during a mean follow-up of 10 months.
CONCLUSIONS	During SR, excellent or good pace-maps at sites of isolated potentials within areas of scar identify areas of fixed block that are protected and part of the critical isthmus of post-infarction VT. Shared common pathways might explain why non-targeted VTs might become noninducible after ablation of other VTs. (J Am Coll Cardiol 2006;47:2013–9) © 2006 by the American College of Cardiology Foundation

In patients with post-infarction ventricular tachycardia (VT), isolated potentials identify areas bounded by anatomical barriers, are frequently present at critical sites within a re-entry circuit, and can be detected during sinus rhythm (SR) (1,2). Pace-mapping has been used to identify the exit site of re-entry circuits (3). The purpose of this study was to determine whether isolated potentials detected during SR in conjunction with pace-mapping in areas of scar are helpful in identifying areas critical to the re-entry circuit of postinfarction VT.

## **METHODS**

**Patient characteristics.** The subjects of this study were 19 consecutive patients (18 men, mean age  $66 \pm 9$  years) referred for radiofrequency ablation of recurrent VT. Their mean left ventricular ejection fraction was  $0.24 \pm 0.12$ . All patients had a history of  $\geq 1$  myocardial infarction (anterior in five, inferior in six, and both anterior and inferior in eight). Catheter ablation was performed because of frequent implantable cardioverter-defibrillator (ICD) discharges in 13 patients and because of recurrent VT in 6 patients.

patients had failed therapy with  $\geq 1$  antiarrhythmic drug, including amiodarone in 14 of 19 patients. All patients had implanted ICDs.

A total of 81 VTs were induced in the 19 patients. The mean cycle length of the induced VTs was 396  $\pm$  124 ms. Thirty-three VTs had a left bundle branch block morphology, and 48 had a right bundle branch block morphology. Mapping data were collected in the following sequence: 1) right ventricular programmed stimulation was performed to assess inducibility of any VT with up to four extrastimuli from two right ventricular sites; 2) a voltage map was constructed from the endocardial surface of the left ventricle, and pacing was performed from sites with abnormal electrograms and isolated potentials; 3) if VTs were hemodynamically tolerated, an activation map was performed during VT and entrainment mapping was done; and 4) radiofrequency energy was delivered if an isthmus was identified according to the outlined criteria (see the following text). After ablation of a particular isthmus region, programmed stimulation was repeated to assess inducibility of the targeted VT.

**Electrophysiologic study and mapping.** After informed consent was obtained, a 6-F electrode catheter was introduced into the right femoral vein and positioned in the right ventricular apex. Programmed right ventricular stimulation

From the University of Michigan Medical Center, Ann Arbor, Michigan. Drs. Morady and Oral have served as consultants to Biosense-Webster.

Manuscript received September 19, 2005; revised manuscript received November 4, 2005, accepted December 5, 2005.

bbreviations	and	Acronyms	
LUDIC Viations	anu	1 tony ms	

- ICD = implantable cardioverter-defibrillator
- SR = sinus rhythm

A

VT = ventricular tachycardia

was performed with one to four extrastimuli. An attempt was made to induce the clinical VT. Identification of the clinically relevant VT was on the basis of a 12-lead electrocardiogram whenever available or on the VT cycle length documented in the stored electrograms of the ICDs. Left ventricular mapping was performed with femoral artery access and a retrograde aortic approach. An electroanatomic mapping system (CARTO; Biosense Webster, Diamond Bar, California) was employed in all patients, with a 7-F mapping/ablation catheter that had a 4-mm tip electrode and a 2-mm ring electrode separated by 1 mm. Electrograms were filtered at 50 to 500 Hz. The intracardiac electrograms and leads V1, I, II, and III were displayed on an oscilloscope and recorded at a speed of 100 mm/s. The recordings were stored on optical disc (EP Medsystems Inc., West Berlin, New Jersey). Systemic heparinization was maintained throughout the procedure.

Mapping protocol. A voltage map was generated during the baseline rhythm, which was either sinus or paced. Right ventricular pacing was not performed while the substrate map was established unless patients were pacemaker dependent (n = 2). An electrogram amplitude <1.0 mV was defined as infarct scar (4). Pace-mapping then was performed at all sites that displayed an abnormal electrogram (5) or an isolated potential. Isolated potentials were defined as potentials that were separated from the ventricular electrogram by an isoelectric segment of >20 ms (Fig. 1). The pacing morphology was compared with that of the induced VTs. A pace-map was considered "perfect" if the QRS complexes in all 12 leads during pacing were identical to those of the targeted VT. A pace-map was considered "good" if the QRS complexes during pacing and during VT were identical in 10 or 11 of the 12 leads.

Sites that displayed an isolated potential during SR were marked on the electroanatomic mapping system. Sites where there was failure of ventricular capture with pacing at an output of 10 mV and with a 2-ms pulse width were considered to be inexcitable scar and also were marked on the electroanatomic mapping system (Fig. 2) (6).

Upon completion of the voltage map, VT was again induced by programmed ventricular stimulation, and activation and entrainment mapping were performed in all hemodynamically-tolerated VTs that could be readily induced.

**Radiofrequency ablation.** Radiofrequency ablation was performed in the critical isthmus of the VT re-entry circuits. The following criteria were used to identify an isthmus area: 1) concealed entrainment (7); 2) a perfect or good pace-map at a site with an isolated potential or abnormal



**Figure 1.** Example of an isolated potential recorded within a posterior left ventricular scar. The isolated potential (**arrow**) is separated from the ventricular electrogram by an isoelectric segment of 120 ms. The amplitude of the isolated potential is 0.035 mV. The electrogram width is 260 ms. In this patient there were 11 different inducible ventricular tachycardias (VTs); after the ablation procedure and the displayed lesions (Fig. 2; 20 radiofrequency lesions of  $\geq 60$  s were delivered), these VTs were no longer inducible. Map dist = distal electrode pair of the mapping catheter.

electrogram (Figs. 3, 4 and 5); and 3) termination of VT by mechanical contact with the mapping catheter and noninducibility of the VT when the mapping catheter was in contact with the site. If radiofrequency ablation resulted in noninducibility of the targeted VT, this was considered to be confirmation that the site was actually within a critical isthmus.

The clinical VTs that triggered the majority of ICD discharges were primarily targeted. Other VTs were targeted if a critical isthmus was identified during mapping. Applications of radiofrequency energy were titrated to maintain a target temperature at the electrode-tissue interface of 60°C and were delivered during VT whenever possible. When ablation was performed during VT, energy application was continued for at least 30 s if adequate heating at the electrode-tissue interface was achieved. If VT terminated within 30 s, the energy application was continued for 60 s, then repeated for another 60 s. If the VT did not terminate in the first 30 s, the energy application was discontinued and other target sites were sought. When radiofrequency ablation was performed during SR, the applications of energy were 60 to 120 s in duration and multiple additional energy applications were delivered in the surrounding area as long as pace-mapping criteria indicated a perfect or good match with the targeted VT.



Figure 2. Voltage map of the posterior aspect of the left ventricle in the same patient as in Figure 1. Radiofrequency energy was delivered at points marked in red and at the pace-map sites illustrated in Figures 3 and 4. Sites with an isolated potential during sinus rhythm are marked in **blue** and sites where there was non-capture are marked in **grey**.

In 10 of 19 patients, because the delivered power was <20 W, an 8-mm-tip ablation catheter was substituted for the 4-mm-tip catheter. With this catheter, the target power was set at 70 W, and the target temperature was  $60^{\circ}$ C.

If a critical isthmus could not be identified with the aforementioned criteria, radiofrequency ablation energy was performed in linear fashion from the scar to areas in the border zone where the best pace-map was obtained, as previously described (3).

After ablation, programmed ventricular stimulation was repeated at two right ventricular sites. Successful catheter ablation was defined as termination of VT by an application of radiofrequency energy and/or the subsequent noninducibility of the targeted VT.

During follow-up, patients were treated with the same antiarrhythmic drugs that they had been taking before the ablation procedure.

**Data analysis.** Electrogram width was measured with electronic calipers from the onset to the offset of the ventricular electrogram or, if an isolated potential was present, to the end of the isolated potential. Bipolar electrogram amplitude also was measured with electronic calipers. Pace-maps were evaluated by two independent investigators, and differences were resolved by consensus.

Mapping/ablation sites were considered distinct if they were separated by  $\geq 5$  mm on the electroanatomic depiction of the left ventricle.

Statistical analysis. Continuous variables are expressed as the mean  $\pm$  1 standard deviation and were compared with Student *t* test. Discrete variables were compared with the Fisher exact test or by chi-square analysis, as appropriate. If a cell size was <5, the Fisher exact test was used. A p value <0.05 was considered statistically significant.

A two-group t test was used to compare between pairs of groups with different pace-maps. Bonferroni adjustments to p values were performed for multiple between-pair comparisons. A paired t test was used to compare the number of ICD therapies before and after radiofrequency ablation (Fig. 6).

The analytic method we chose assumed that the data obtained from multiple sites for each patient were statistically independent. Analyzed data points were at a distance from each other, and the association to different VT circuits was assessed for each point.



Figure 3. Example of a perfect pace-map. The targeted ventricular tachycardia (VT) is shown in the left panel and the pace-map obtained at the site displayed in Figure 1 is shown in the right panel. The QRS complexes during VT and pacing are identical in all 12 leads.



Figure 4. Example of a good pace-map, in which the QRS complexes during ventricular tachycardia (VT) (left) and pacing (right) are identical in 10 of 12 leads. Leads II, III, and aVF display some minor differences in the morphology of the paced QRS complexes as compared with the targeted VT.



Figure 5. The voltage map shown in Figure 2, indicating where the pace-maps in Figures 3 and 4 were obtained.

#### RESULTS

**Catheter ablation.** In 16 of the 19 patients, at least one critical isthmus of a VT re-entry circuit was identified. In three patients in whom 16 VTs were inducible, no critical isthmus could be identified.

A critical isthmus was identified in the re-entry circuit of 41 of the 81 inducible VTs, and each of these 41 VTs was successfully ablated. These VTs included the clinical VTs in 16 of 19 patients. An additional 22 of 40 VTs in which a critical isthmus was not identified became noninducible after ablation of the targeted VTs. Mean fluoroscopy time was  $62 \pm 25$  min, and mean procedure time was  $346 \pm 92$  min. A mean of  $25 \pm 16$  min of radiofrequency energy were delivered per patient.

Pace-mapping and electrogram characteristics (Tables 1, 2, and 3). Pace-mapping was performed at 681 distinct sites where the mean electrogram amplitude was  $0.48 \pm 0.67$  mV and the mean electrogram width was  $91 \pm 52$  ms. Isolated potentials were identified in all patients. The maximal width of the isoelectric segment separating ventricular electrogram from an isolated potential in patients with left bundle branch block or pacing was not different compared with patients without pacing or without left bundle branch block ( $145 \pm 103$  ms vs.  $138 \pm 72$  ms; p = 0.9). Isolated potentials were found more frequently at sites with good or perfect pace-maps than at sites with abnormal electrograms (p < 0.0001). Pace-maps at 65% of sites

displaying an isolated potential were either perfect or good, compared with 5% of sites with abnormal/fragmented electrograms (p < 0.0001). At sites with isolated potentials, the mean stimulus–QRS interval was longer than at sites with abnormal electrograms ( $108 \pm 43 \text{ ms vs. } 81 \pm 54 \text{ ms}$ , p < 0.0001). With a cutoff isoelectric interval of  $\geq 20 \text{ ms}$  for the definition of isolated potentials, the sensitivity and specificity for identifying an isthmus area were 80% and 84%, respectively. With an isoelectric interval of  $\geq 50 \text{ ms}$ , the sensitivity and specificity for identifying an isthmus area were 54% and 90%, respectively. Sites with a perfect pace-map had a longer isoelectric segment separating the ventricular electrogram from the isolated potential as compared with sites with closely matching pace-maps ( $105 \pm 79 \text{ ms vs. } 70 \pm 51 \text{ ms}$ ; p < 0.001).

Isthmus characteristics (Table 1). Of 41 critical isthmus areas, 17 (41%) were identified by a perfect pace-map, 14 (34%) by a good pace-map, 4 (10%) by mechanical interruption of VT, and 6 (15%) by concealed entrainment. An isolated potential was present during SR in the critical isthmus in 40 of 41 patients (98%). In the isthmus areas, a mean of  $5 \pm 4$  sites (range 1 to 15 sites) per VT with either a perfect or good pace-map were identified.

**VT mapping (Table 1).** Entrainment and activation mapping was performed in 6/81 VTs (7%) in 6 of 19 patients (32%). The remaining VTs were either not hemodynamically tolerated or not readily inducible. A critical isthmus was



**Figure 6.** Number of appropriate implantable cardioverter-defibrillator (ICD) therapies delivered during the three months before and at three months and six months after ventricular tachycardia ablation. The mean  $\pm$  1 standard deviation is shown at each time point. RF = radiofrequency catheter ablation.

identified by entrainment mapping in these six VTs. When pacing was performed during SR at these sites, there always was either a good or perfect pace-map. Furthermore, all six sites displayed an isolated potential during both VT and SR.

Four VTs terminated and/or became noninducible because of mechanical contact by the mapping catheter. The pace-map was perfect or good at all four sites.

**Follow-up.** The mean duration of follow-up was  $9.8 \pm 6$  months (range 2 to 21 months). The mean number of appropriate ICD therapies decreased from  $60 \pm 95$  in the 3 months before ablation to  $0.5 \pm 1.3$  at 3 months of follow-up and to  $1.4 \pm 4$  at 6 months of follow-up (Fig. 6). Among the three patients in whom a critical isthmus could not be identified, none had a decrease in the number of ICD discharges. One of these three patients died of heart failure three months after the ablation procedure, one underwent heart transplantation four months after the

Table 1. Criteria Used to Identify 41 Critical Isthmus Sites

Criterion	Identified Isthmus	IPs Present During SR
Concealed entrainment	6 (15%)	6
Perfect pace-map	17 (41%)	17
Good pace-map	14 (34%)	14
Mechanical VT termination	4 (10%)	3

IP = isolated potential; SR = sinus rhythm; VT = ventricular tachycardia.

Table 2.	Comparison	of Sites	With	and	Without Isolated	
Potential	s					

	Isolated Potential	Isolated Potential	. 17.1
Unaracteristics	rresent	Absent	p value
No. of evaluated sites	269	412	
Amplitude (mV)	$0.14\pm0.25$	$0.74 \pm 0.77$	< 0.0001
EGM width (ms)	$199 \pm 70$	$130 \pm 27$	< 0.0001
S-QRS (ms)	$108 \pm 43$	$81 \pm 54$	< 0.0001
Perfect pace-map	69/269	1/412	$< 0.0001^{*}$
Good pace-map	103/269	21/412	< 0.0001

\*p value assessed by Fisher exact test.

EGM = electrogram; S-QRS = stimulus-QRS interval.

ablation procedure, and the third patient underwent a repeat ablation procedure.

#### DISCUSSION

**Main findings.** In this study, pace-mapping within postinfarction scar at sites where there was an isolated potential during SR was a reliable indicator of a critical isthmus in the VT re-entry circuit. The strategy used in this study to identify isthmus sites resulted in freedom from recurrent VT in 16 of 19 patients (84%) during a mean follow-up period of 10 months.

**Isolated potentials.** Isolated potentials have been described in experimental studies of healed canine infarcts (8) and in infarcted human papillary muscles (9). The anatomic substrate of isolated potentials are single myocardial muscle strands that are separated by fibrous tissue (8,9). A zigzag course of activation accounts for delayed activation (10). Sites with isolated potentials during SR can identify critical areas within a VT re-entry circuit (2,11).

Different criteria have been used for defining isolated potentials (11). Compared with a criterion of  $\geq 20$  ms between the ventricular electrogram and the isolated potential, a criterion of  $\geq 50$  ms results in improved specificity at the expense of a decline in sensitivity. Using a longer isoelectric segment criterion therefore might result in the failure to detect some VT isthmus areas. Furthermore, prior studies demonstrated a higher prevalence of isolated potentials when right ventricular pacing was performed as compared with SR (11). Different criteria for defining the length of the isoelectric segment between ventricular electrogram

**Table 3.** Comparison of Sites With Perfect, Good, and PoorPace-Maps

Perfect	Good	Poor
70	124	487
$0.07 \pm 0.04^{*}$	$0.19 \pm 0.3^{*}$	$0.63 \pm 0.7^{*}$
$241 \pm 83^{*}$	$177 \pm 65^{*}$	$140 \pm 38^{*}$
$120 \pm 52^{*}$	99 ± 41*	86 ± 53*
69/70 (99%)†	102/124 (82%)†	96/487 (20%)†
	Perfect   70   0.07 ± 0.04*   241 ± 83*   120 ± 52*   69/70 (99%)†	PerfectGood70124 $0.07 \pm 0.04^*$ $0.19 \pm 0.3^*$ $241 \pm 83^*$ $177 \pm 65^*$ $120 \pm 52^*$ $99 \pm 41^*$ $69/70$ $(99\%)^{\dagger}$ $102/124$ $(82\%)^{\dagger}$

Comparison between pairs of groups with different pace-maps. \*p value significant with Bonferroni adjustments. †p value < 0.001 comparing the prevalence of isolated potentials across the groups with chi-square test.

Abbreviations as in Table 2.

and isolated potential might explain the differences between this study and prior investigations (11).

In a prior study in patients with post-infarction VT, approximately 33% of VTs shared a critical isthmus (12). Furthermore, isolated potentials were present at the majority of sites with shared common pathways. These data are consistent with the results of the present study, in which approximately 25% of VTs became noninducible after ablation of a targeted VT.

**Pace-mapping.** Pace-mapping has been used to identify appropriate target sites for ablation of post-infarction VT along the scar border zone (3). In the present study, pace-mapping was performed throughout the area of scar in which abnormal electrograms were recorded, not only along the scar border zone. Brunckhorst et al. (13) also reported that pace-mapping helped to identify the VT isthmus; however, the electrogram characteristics at a critical isthmus were not described, and pacing was performed at all left ventricular sites. The presence of isolated potentials greatly enhances the ability to identify a critical isthmus of a VT re-entry circuit during SR.

When good and perfect pace-maps were compared, there were significant differences in electrogram amplitude as well as electrogram width and stimulus-QRS intervals. Furthermore, at sites with perfect pace-maps the prevalence of isolated potentials was higher than at sites with good pace-maps. This suggests that sites with good pace-maps are not as protected as sites with perfect pace-maps and that the re-entry circuit might be composed of functional components at these sites, as has been suggested by others (14-16); however, there was no difference in outcomes at target sites that displayed a perfect versus a good pace-map. Therefore, even a pacemap that has QRS complexes identical to those of the VT in only 10 of 12 leads might be sufficient to identify a VT isthmus site, when pace-mapping is performed where an isolated potential is present during SR within the scar tissue. Of note is that a mean of five sites with perfect or good pace-maps was identified per targeted VT when an isthmus could be identified. Therefore, multiple radiofrequency energy applications were delivered when an isthmus area was identified by pace-mapping.

**Study limitations.** Pace-mapping was not performed at all left ventricular sites; therefore the true prevalence of sites with perfect pace-maps might be underestimated. Sites with normal electrograms, however, are unlikely to play a critical role in post-infarction VTs. Focusing only on sites with isolated potentials when pace-mapping might not identify re-entry circuits that are mainly functional in nature and might not display an isolated potential during SR might have accounted for the failure to identify re-entry circuits in three patients, although epicardial or intramural circuits cannot be excluded either. Furthermore, whereas the study demonstrates the high sensitivity of an isolated potential recorded during SR for a critical isthmus, the study provides no data on the specificity of isolated potentials for successful ablation sites.

**Conclusions.** This study demonstrates that an isolated potential recorded during SR within a post-infarction scar, in conjunction with pace-mapping, is a sensitive indicator of a critical isthmus in the VT re-entry circuit. Shared critical isthmuses might explain why non-targeted VTs might become noninducible after ablation of targeted VTs.

**Reprint requests and correspondence:** Dr. Frank Bogun, Division of Cardiology, University of Michigan Health System, TC B1 140, 1500 East Medical Center Drive, Ann Arbor, Michigan 48109-0366. E-mail: fbogun@umich.edu.

### REFERENCES

- Bogun F, Bahu M, Knight B, et al. Response to pacing at sites of isolated diastolic potentials during ventricular tachycardia in patients with previous myocardial infarction. J Am Coll Cardiol 1997;30:505–13.
- Bogun F, Bender B, Li YG, et al. Analysis during sinus rhythm of critical sites in re-entry circuits of postinfarction ventricular tachycardia. J Interv Card Electrophysiol 2002;7:95–103.
- Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesions for control of unmappable ventricular tachycardia in patients with ischemic and nonischemic cardiomyopathy. Circulation 2000; 101:1288–96.
- Callans DJ, Ren JF, Michele J, Marchlinski FE, Dillon SM. Electroanatomic left ventricular mapping in the porcine model of healed anterior myocardial infarction. Correlation with intracardiac echocardiography and pathological analysis. Circulation 1999;100:1744–50.
- 5. Josephson M. Clinical Cardiac Electrophysiology Techniques and Interpretations. Philadelphia, PA: Lea & Febiger, 1993.
- Soejima K, Stevenson WG, Maisel WH, Sapp JL, Epstein LM. Electrically unexcitable scar mapping based on pacing threshold for identification of the reentry circuit isthmus: feasibility for guiding ventricular tachycardia ablation. Circulation 2002;106:1678-83.
- Bogun F, Bahu M, Knight B, et al. Comparison of effective and ineffective target sites that demonstrate concealed entrainment in patients with coronary artery disease undergoing radiofrequency ablation of ventricular tachycardia. Circulation 1997;95:183–90.
- Gardner PI, Ursell PC, Fenoglio JJ Jr., Wit AL. Electrophysiologic and anatomic basis for fractionated electrograms recorded from healed myocardial infarcts. Circulation 1985;72:596–611.
- de Bakker JM, Coronel R, Tasseron S, et al. Ventricular tachycardia in the infarcted, Langendorff-perfused human heart: role of the arrangement of surviving cardiac fibers. J Am Coll Cardiol 1990;15:1594–607.
- de Bakker JM, van Capelle FJ, Janse MJ, et al. Slow conduction in the infarcted human heart. 'Zigzag' course of activation. Circulation 1993;88:915–26.
- 11. Arenal A, Glez-Torrecilla E, Ortiz M, et al. Ablation of electrograms with an isolated, delayed component as treatment of unmappable monomorphic ventricular tachycardias in patients with structural heart disease. J Am Coll Cardiol 2003;41:81–92.
- Bogun F, Li YG, Groenefeld G, et al. Prevalence of a shared isthmus in postinfarction patients with pleiomorphic, hemodynamically tolerated ventricular tachycardias. J Cardiovasc Electrophysiol 2002;13:237–41.
- Brunckhorst CB, Delacretaz E, Soejima K, Maisel WH, Friedman PL, Stevenson WG. Identification of the ventricular tachycardia isthmus after infarction by pace mapping. Circulation 2004;110:652–9.
- Downar E, Harris L, Mickleborough LL, Shaikh N, Parson ID. Endocardial mapping of ventricular tachycardia in the intact human ventricle: evidence for reentrant mechanisms. J Am Coll Cardiol 1988;11:783–91.
- Pogwizd SM, Hoyt RH, Saffitz JE, Corr PB, Cox JL, Cain ME. Reentrant and focal mechanisms underlying ventricular tachycardia in the human heart. Circulation 1992;86:1872–87.
- Callans DJ, Zardini M, Gottlieb CD, Josephson ME. The variable contribution of functional and anatomic barriers in human ventricular tachycardia: an analysis with resetting from two sites. J Am Coll Cardiol 1996;27:1106–11.