Ventricular tachycardia ablation

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The role of catheter ablation in the treatment of ventricular arrhythmias has been changing in the last decade, and this form of therapy now aims at curing multiple ventricular tachycardia morphologies and complex substrates in patients with structural heart disease (post-infarction and idiopathic dilated cardiomyopathy). Under these circumstances, conventional mapping is not feasible and accurate, and the development of new diagnostic methods has become necessary. The non-contact mapping system has been introduced to study the activation pattern of any ventricular arrhythmia by a "single-cycle" analysis, and has brought to the characterization of unstable and of non-sustained forms of arrhythmia. The evaluation of the arrhythmogenic substrate has similarly become more precise by the more common use of the electroanatomic mapping (CARTO), which is being applied to identify areas of scarred tissue responsible for ventricular arrhythmias, to map stable tachycardias and to validate the creation of a line of block. By means of this technological advancement, the identification of critical isthmi and deep intramural circuits has also led to new ablation strategies, frequently simplifying the procedure and minimizing complications.

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Among the emerging indications of catheter ablation, the treatment of ventricular tachycardia (VT), particularly in the setting of certain forms of structural heart disease, represents an increasingly complex challenge. Originally aimed at the treatment of "easy" cases, such as relatively slow and well tolerated paroxysmal VTs late after infarction, catheter ablation is now faced most frequently with difficult patterns, characterized by hemodynamic instability, the occurrence of multiple morphologies of monomorphic VT (pleomorphism), or even by polymorphic arrhythmias. The improved life expectancy in survivors of myocardial infarction and in patients with various forms of left ventricular dysfunction, following the widespread use of the implantable cardioverter-defibrillator (ICD), is bringing about an increased number of patients, in whom repeated shocks cause a significant discomfort and worsen quality of life, or pose a significant threat on the patients' cardiac status and even on survival[1-3].

For all the above-mentioned reasons the need for a curative approach, such as that potentially offered by catheter ablation, is felt as an important goal by those electrophysiologists who are frequently involved in the management of patients with VT, both with post-infarction cardiomyopathy as well as with idiopathic dilated cardiomyopathy (IDCM).

In this review the most recent applications of VT catheter ablation and the use of advanced mapping and ablation techniques will be discussed.

Ventricular tachycardia in the setting of post-infarction cardiomyopathy

Recent evidence has proven that catheter ablation performed using a single catheter approach based on conventional activation mapping and pacing techniques, frequently associated with concomitant administration of antiarrhythmic drugs, can offer a reasonable rate of success over the long term in patients presenting with recurrent hemodynamically tolerated post-infarction VT or incessant monomorphic arrhythmia following a myocardial infarction[4]; it has been shown that the overall sudden death and cardiac death rates reported are comparable to those achieved in patients treated exclusively by an ICD. However, this type of clinical presentation is offered by a minority of patients, whereas a combination of hemodynamically untolerated VTs and multiple morphologies is more frequently documented; furthermore, the non-reproducible induction of different tachycardias during the electrophysiologic study is an additional issue that complicates the ablation procedure. In these instances mapping and abla-

tion performed by the single catheter technique offers a low success rate. In the experience by O'Donnell et al.[5], the success rate was higher among those patients presenting with one clinical and inducible morphology, on the other hand, the outcome was much poorer in the presence of pleomorphism, particularly if untolerated arrhythmias occurred. To this regard, there are several technical issues that should be addressed when a catheter ablation of VT is considered, including pleomorphism and presentation of untolerated VTs.

Pleomorphism and untolerated forms of ventricular tachycardias. Pleomorphism is a peculiar feature of the arrhythmogenic substrate in patients with a prior myocardial infarction and a frequent cause of failure of catheter ablation. In a recent study by our group, including a cohort of 137 consecutive patients undergoing catheter ablation of post-infarction VT, the outcome of patients presenting with a single VT morphology was compared to that of patients with multiple spontaneous VTs[6]. Pleomorphism caused a lower acute success rate (69 vs 23%) and was associated with a worse long-term arrhythmia-free survival. In this experience, in fact, the difficulty to effectively treat patients with spontaneous multiple morphologies of VTs was related to the presence of multiple critical isthmi; of note, patients with a single morphology of clinical VT in whom other forms could only be induced in the electrophysiologic lab, had in the majority of cases a single isthmus responsible for the arrhythmia: this was the reason why they could be more easily ablated causing the suppression of all – clinical and non-clinical – VTs. Given the importance to ablate all inducible VTs, which was the major predicting factor for long-term success, the most frequent cause responsible for failure was related to hemodynamic intolerance, that was more frequent in patients with clinically documented pleomorphism, resulting from the more advanced degree of left ventricular dysfunction and to the short cycle length of the arrhythmia. Of note, in this study, only a conventional point-by-point mapping was used in the majority of cases, this limiting the acute success rate compared to the minority of cases supported by a non-conventional mapping system.

It results therefore that in this situation a precise definition of the arrhythmogenic substrate and of the many possible pathways potentially able to sustain a VT is pivotal to guide a successful procedure: different advanced mapping techniques have been for this reason introduced into clinical practice to improve the analysis of endocardial activation during unstable and untolerated forms of VTs, and to allow the accurate characterization of the arrhythmic substrate responsible for one or more reentry circuits, leading to strategies of linear catheter ablation.

Non-contact mapping (Endocardial Solutions). Through the placement of a balloon-mounted multielctrode array catheter, the non-contact mapping system allows a fairly accurate reconstruction of the virtual three-dimensional geometry of the left ventricular endocardium. A computer-based processing of the non-contact unipolar electrograms recorded by the 64 electrodes composing the array, allows the visualization of the activation sequence of the endocardium, with a practically unlimited possibility of projecting virtual electrograms from any ventricular site. This system offers the unique possibility of an off-line analysis of each desired beat, during sinus rhythm or during any moment of a VT. It is therefore possible to have a fast, untolerated VT induced, and to record a short segment of arrhythmia. Following arrhythmia interruption, the subsequent analysis of the activation pattern can be performed while the patient is in stable sinus rhythm: this allows the detection of the diastolic pathway and of the exit site during each VT. Significant information can be obtained on the critical isthmus of different monomorphic VTs, polymorphic VTs, non-sustained VTs. Similarly, the analysis of the activation pattern during sinus rhythm helps in defining areas with lower amplitude, fractionated, or with late potentials normally related to the scar tissue (substrate mapping). This information is essential to define the mechanism of a given arrhythmia and to place properly defined ablation lines, whose precise location can also be checked. Limitations of this mapping system include the need for two catheters in the left ventricle; sometimes the attainment of a stable position of the bulky and stiff 9F balloon catheter is difficult, particularly in patients with tortuous peripheral arteries or atherosclerotic aorta, or with aortic valve stenosis. Furthermore, the accuracy of the system decreases at increasing distances of the endocardium from the core of the multielectrode array so that in dilated ventricles the analysis of the activation pattern of certain areas is not always reliable. In spite of this, the non-contact mapping system represents a formidable tool for the treatment of untolerated VTs.

In our Center, mapping and ablation of untolerated VTs was guided by the non-contact technique in 76 patients. The clinical characteristics of the study population are shown in table I. One hundred sixty one episodes of VT were induced (mean cycle 302 ms, range 220-450 ms); a satisfactory mapping could be obtained in 153 episodes (95%). Identification of the VT exit point or of the diastolic pathway led to VT ablation. As shown in table II, the success rate of ablation, for all VTs, but particularly in the post-infarction subset, was significantly greater targeting the diastolic pathway as compared to the exit point. Over the long-term (Table III), the recurrence rate of the clinical VT and of any VT was significantly lower when prevention of all VTs was achieved at the control electrophysiologic study performed after ablation[7]. The high recurrence rate of any arrhythmic event, even following a successful procedure, is however a strong indication for ICD implant in these patients.

Table I. Non-contact mapping-guided ventricular tachycardia (VT) radiofrequency catheter ablation: population characteristics and mapping results.

No. patients	76
Etiology	
Post-infarction	35
ARVD	18
IDCM	18
CHD surgery	5
Mean ejection fraction (%)	
Post-infarction	33.7
IDCM	31
Clinical setting: excessive ICD therapy	45
VT induced	161
Post-infarction	108
ARVD	28
IDCM	23
CHD surgery	6
Mean cycle (ms)	302 (220-450)
VT mapped	153 (95%)
Hemodynamic decompensation	All induced VTs

ARVD = arrhythmogenic right ventricular dysplasia; CHD = congenital heart disease; ICD = implantable cardioverter-defibrillator; IDCM = idiopathic dilated cardiomyopathy.

An example of linear ablation in polymorphic post-infarction VT guided by non-contact mapping is represented in figure 1.

Electroanatomic mapping system (CARTO). Another approach that has become very popular for the treatment of VTs, is the evaluation of the arrhythmic substrate by the electroanatomic system. Based on the precise spatial guidance achieved by low-intensity magnetic fields, the system offers a rather accurate reconstruction of the left ventricular geometry, allowing both a voltage map – defining the scar area – and the activation map – localizing areas of the latest activation during sinus rhythm and displaying the pattern of endocardial activation during stable VTs –; this helps in characterizing the reentry circuit of a given arrhythmia and in guiding properly placed linear lesions. An example is shown in figure 2. Many investigators have developed strategies for mapping and ablation of unstable VTs, based on a hybrid approach involving both precise scar-mapping and pace-mapping, trying to reproduce from selected sites the morphology of previously induced and terminated fast VTs. Leading experiences conducted in European and North American Centers in a relatively high number of patients, showed that in the majority of patients the electroanatomic mapping system can effectively guide a linear-lesion strategy performed by transecting reentry critical isthmi, thus limiting the extension of lesions[8-13].

Ventricular tachycardia in idiopathic dilated cardiomyopathy

The histopathologic and electrophysiologic characteristics of VTs in IDCM are even more complex and less well defined when compared to post-infarction VT, as documented by different authors[14,15]. Also acute and long-term results of catheter ablation are less favorable, when conventional mapping and ablation techniques are used, with an overall clinical success rate of < 60%. In fact, three different mechanisms of

Table II. Differences in the procedural outcome related to the ablation approach.

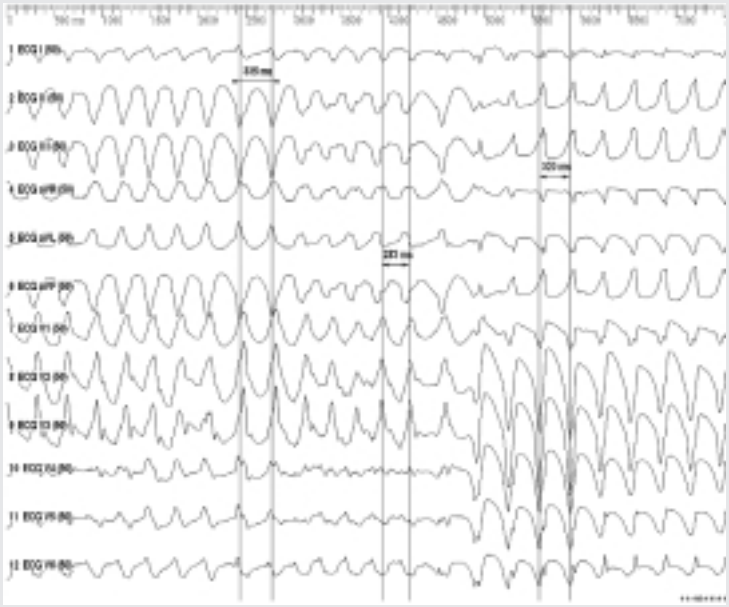
	Ablated VTs (%)		Overall (%)
	Linear lesion on the diastolic pathway	Encircling exit point	
Post-infarction VT	78	40*	71
All VTs	73	41*	63

VT = ventricular tachycardia. * $p < 0.05$ vs linear lesion on the diastolic pathway.

Table III. Differences in the long-term recurrence rates related to the ablation outcome.

	Recurrences		Failure
	Effective ablation of all induced VTs	Effective ablation of clinical VTs with persistent inducibility of non-clinical morphologies	
No. patients	37	20	14
Clinical arrhythmia recurrence	17%	37%	87%
Arrhythmic events	29%	75%	100%

VT = ventricular tachycardia.



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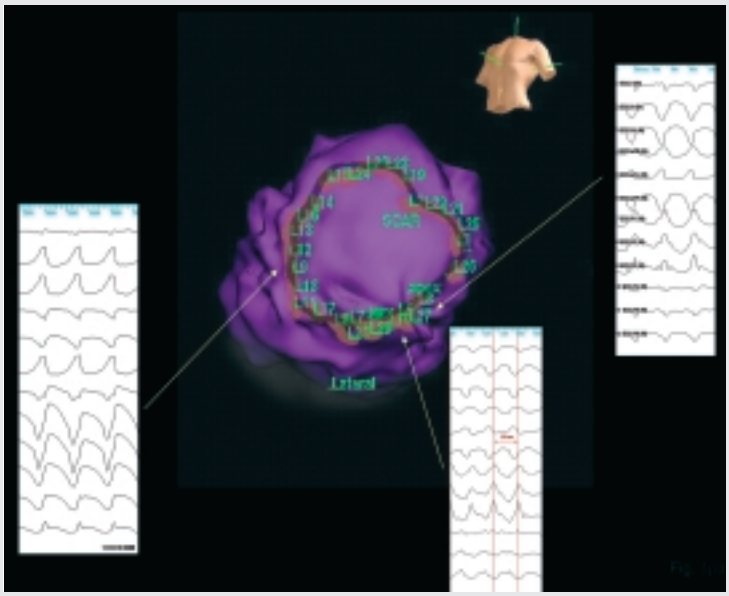

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Figure 1. *Apical infarction. Polymorphic ventricular tachycardia (transition from ventricular tachycardia 1 to 2 and 3)* (panel A). *Virtual reconstruction of the apical aneurysm by non-contact mapping* (panel B). *The exit point at the scar border zone of three ventricular tachycardias is identified and a linear lesion is performed with successful prevention of ventricular tachycardia inducibility.*

VT (complex scar-related reentry, focal automaticity, bundle branch reentry) are responsible for VT in patients with IDCM and the optimal approach for catheter mapping and ablation of all IDCM-VTs cannot be defined; in this setting, VT ablation requires the preliminary assessment of the proper arrhythmia mechanism in a given patient. The most common mechanism for monomorphic IDCM-VTs is, however, myocardial reentry involving areas of scarred tissue. A better characterization of the arrhythmic substrate responsible for IDCM-VTs has been provided by Hsia et al.[16] by the use of electroanatomic mapping; these authors showed that in the majority of patients only a restricted area of endocardium is affected by abnormal low-voltage electrograms (< 1.8 mV), accounting for < 25% of the endocardial surface, which is typically located in the basal area of the left ventricle surrounding the mitral valve annulus. A correlation between

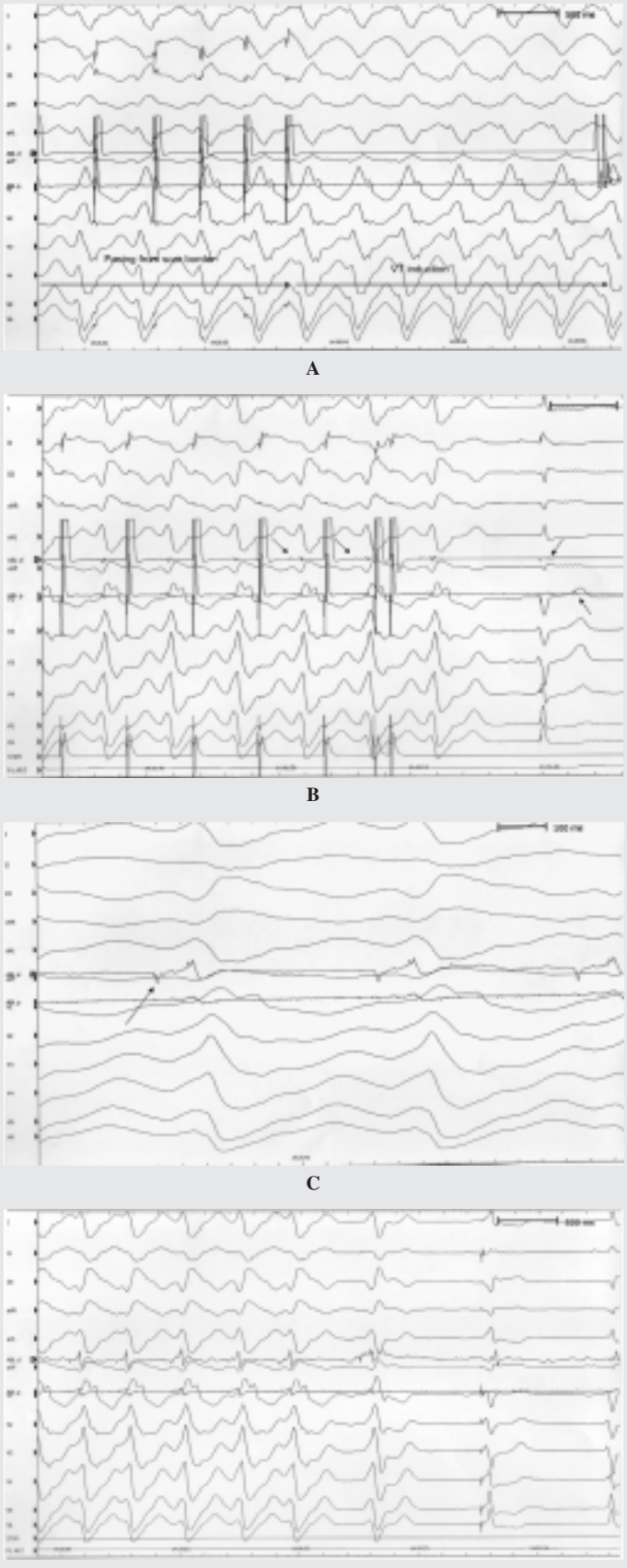
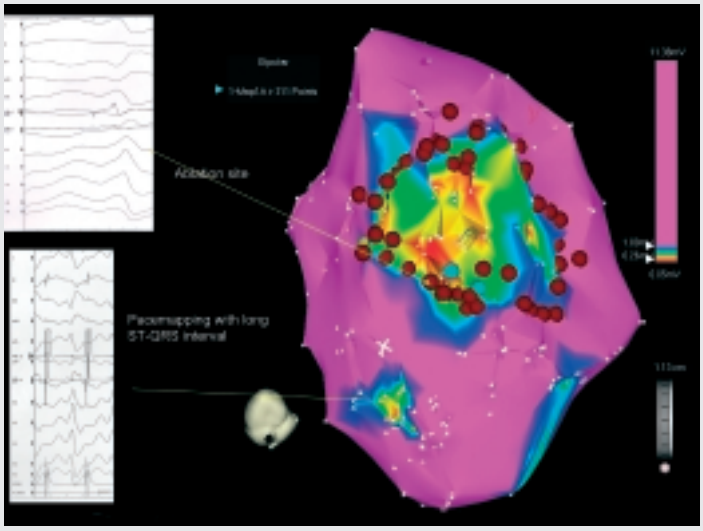


Figure 2. *Linear ablation of post-infarction ventricular tachycardia (VT) guided by CARTO mapping.* Panel A: *induction of VT by pacing from the entrance site of the critical isthmus, with long St-QRS interval and late and fragmented electrograms during sinus rhythm* (panel B). *Ablation site* (panel C) *identified by activation mapping and VT termination during radiofrequency delivery* (panel D) *are shown.*



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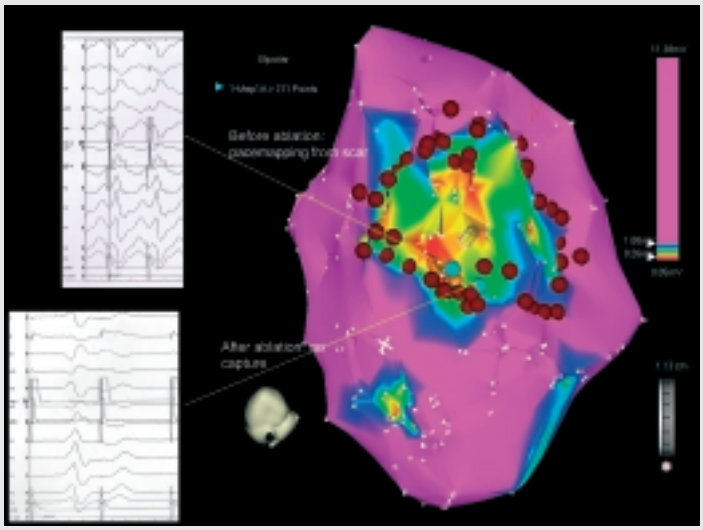

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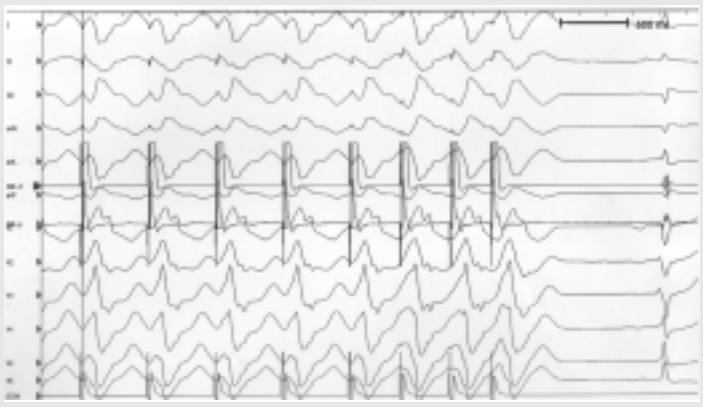

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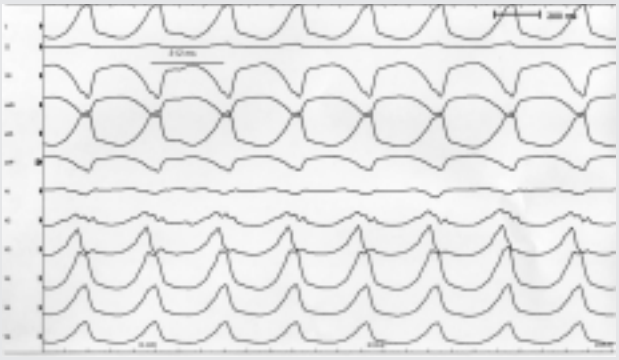
Figure 2. *Linear ablation of post-infarction ventricular tachycardia (VT) guided by CARTO mapping. In* panel E *the entrance and ablation sites are shown in bipolar voltage map of the inferior scar area. Lesions to encircle the scar are performed.* Panel F: *no capture can be obtained by pacing at the VT exit site after effective ablation and no VT can be further induced* (panel G).



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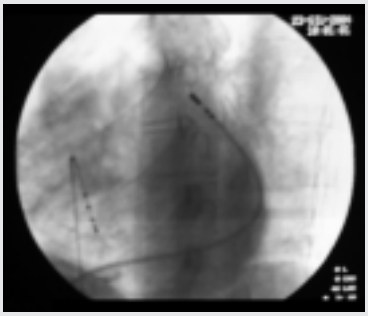

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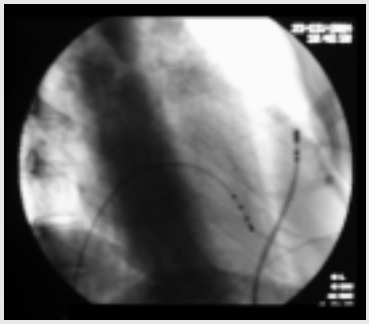

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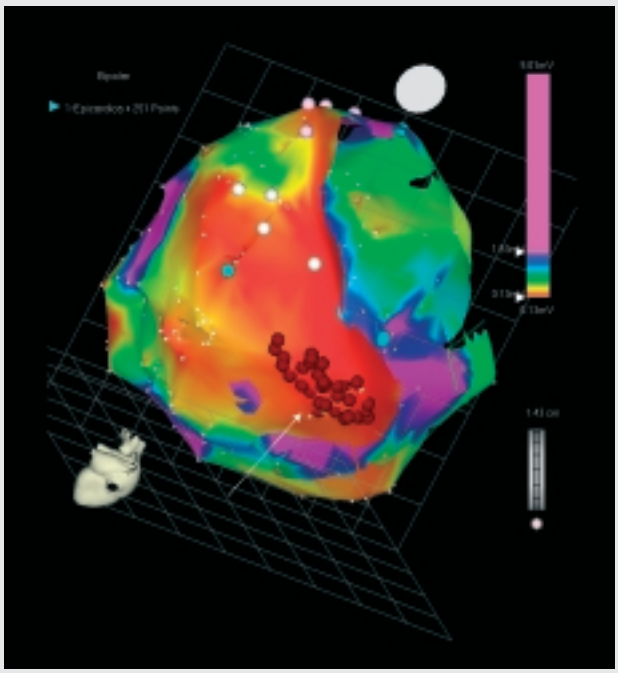

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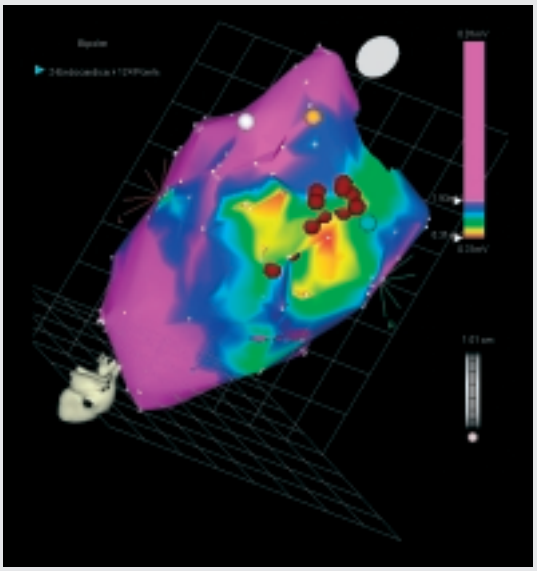

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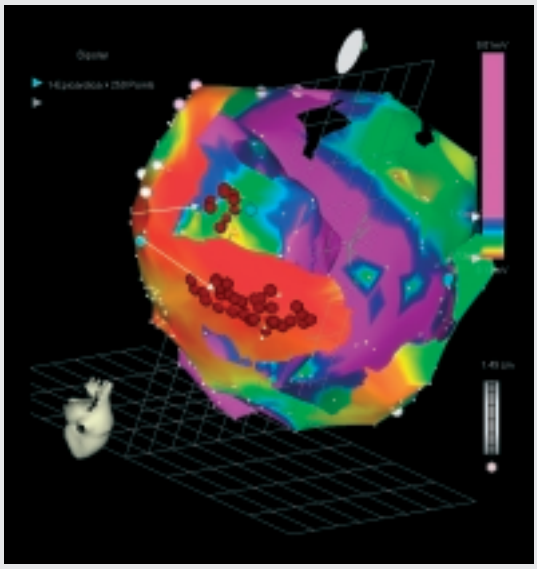

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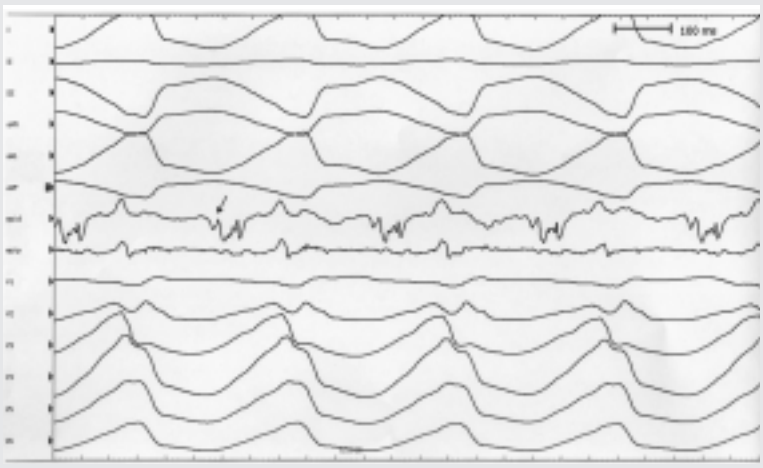
Figure 3. *Ablation of epicardial ventricular tachycardia in a patient with idiopathic dilated cardiomyopathy guided by endocardial and epicardial electroanatomic mapping.* Panel A: *clinical ventricular tachycardia.* Panels B *and* C: *left and right anterior oblique views of the ablation site at the lateral epicardium.* Panels D-F: *CARTO mapping of the epicardium* (panel D)*, the endocardium* (panel E)*, and both showing the epicardial ablation site* (arrows) *at the basal lateral left ventricle.*



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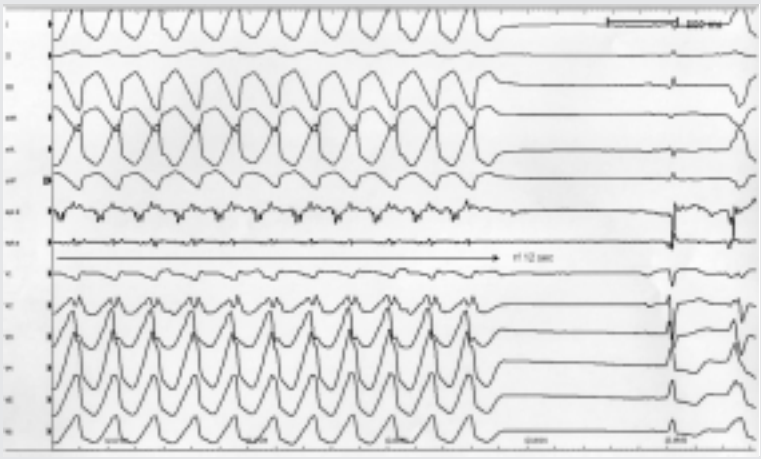

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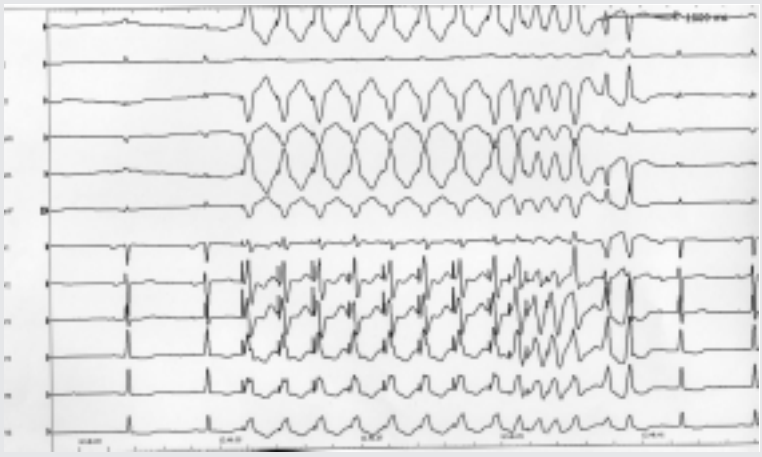

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Figure 3. *Ablation of epicardial ventricular tachycardia in a patient with idiopathic dilated cardiomyopathy guided by endocardial and epicardial electroanatomic mapping.* Panel G: *ablation site.* Panel H: *ventricular tachycardia termination by radiofrequency.* Panel I: *prevention of ventricular tachycardia induction after ablation.*

this area of abnormal endocardial electrograms and the site of origin of the VT was demonstrated in the majority of VTs (88%).

Deep intramural or epicardial isthmus. The second specific feature of the arrhythmogenic substrate for IDCM-VTs is the extensive involvement of the epicardial layers, that can be demonstrated in > 30% of the patients undergoing simultaneous endo- and epicardial mapping by the use of electroanatomic techniques, as recently demonstrated by Soejima et al.[17]. An epicardial scar was documented in 7 patients undergoing epicardial electroanatomic mapping after endocardial ablation had failed, and in 6/7 a critical isthmus responsible for VT was demonstrated by pace-mapping/entrainment and successfully ablated. For this reason, a deep intramural or epicardial isthmus of the circuit may be considered the target for ablation in many forms of IDCM-VTs, thus differentiating these arrhythmias from post-infarction VTs, in whom the scar is more frequently involving the endocardium and can be successfully ablated from this side. In patients with IDCM-VTs, advanced mapping may facilitate the detection of a deep substrate and guide the need for a direct epicardial approach in selected cases, as shown in one presented case.

Case presentation. A typical example of the epicardial involvement in a patient with IDCM is shown in figure 3. A 47-year-old woman, suffering from recurrent palpitations for 2 years, had a monomorphic sustained VT documented upon an emergency-room admission. Following DC-shock cardioversion, therapy with amiodarone was instituted. During hospitalization, an echocardiogram revealed a dilated left ventricle with a uniformly depressed contractile function, with a left ventricular ejection fraction of 40%. A coronary angiogram was normal and, given the absence of inflammatory or autoimmune disease as a cause for myocarditis, the diagnosis of IDCM was made. The patient experienced repeated VT recurrences in spite of the antiarrhythmic treatment, and for this reason she was referred to our Institute for electrophysiologic evaluation and ablation. A first procedure was performed using a conventional single catheter approach that revealed a small area of diastolic activation during VT. Radiofrequency ablation on this site resulted in the termination of the arrhythmia and in the prevention of its induction. Shortly after, however, the patient experienced recurrence of the same VT, and a second procedure was planned, involving simultaneous endo- and epicardial CARTO mapping. Access to the epicardium was achieved through a subxyphoid puncture, according to the technique described by Sosa et al.[18]. As shown in figure 3, epicardial mapping during sinus rhythm revealed an extensive area of the inferior, lateral and anterior walls toward the base of the left ventricle characterized by low-amplitude (< 1.0 mV) electrograms; in contrast, the corresponding areas on the endocardial surface were normal, with the exception of a small endocardial area corresponding to the site of the previous ablation. During VT, diastolic activity was recorded epicardially; delivery of radiofrequency current (temperature control, 50°C; 15 W) led to early VT interruption. An extensive series of lesions was delivered on the epicardial surface, at sites surrounding the VT termination site, and again at corresponding endocardial sites. Prevention of the induction of any VT was achieved by this ablation procedure and the patient has remained free from any arrhythmia since then.

Conclusions

The definite role of catheter ablation, for specific subsets of VT presentation, is an evolving one. Although the placement of a back-up ICD seems to be reasonable in most patients with structural heart disease, the role of catheter ablation in eliminating VT recurrences is becoming more and more relevant, since it provides undoubtedly a significant improvement of quality of life in patients with frequent interventions of the device. Furthermore, the avoidance of electrical storms by catheter ablation is likely to improve the prognosis in these patients.

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