Hybrid Therapies for Ventricular Arrhythmias

PAOLO DELLA BELLA and STEFANIA RIVA

From the Arrhythmia Department, Institute of Cardiology, University of Milan, Centro Cardiologico Monzino, IRCCS, Milan, Italy

In recent years several trials demonstrated the efficacy of implantable cardioverter-defibrillation (ICD) therapy in reducing cardiac and total mortality in patients affected by rapid ventricular tachycardia (VT) and/or ventricular fibrillation. Nevertheless, ICD do not prevent arrhythmia recurrences, thus being a palliative and not a curative treatment modality. The tolerance to ICD therapy varies greatly, and within individuals, this leading to a nonuniform acceptance of this form of therapy. The very frequent occurrence of VT, defined as an arrhythmic storm, may be a life threatening condition. The majority of ICD patients is under antiarrhythmic drug therapy, to reduce episodes of VT or to make antitachycardia pacing more effective by slowing the tachycardia rate. Drug therapy, however, may cause additional problems, and does not represent the optimal solution. The prevention of VT and/or ventricular fibrillation episodes and excessive ICD therapy, remains a worthwhile goal. Radiofrequency catheter ablation (RFCA) is a curative approach, and can be expected to reduce the frequency of recurrent VT episodes in the majority of patients. The combination of these treatment modalities (ICD and RFCA) is often described as hybrid therapy, implying that the two treatments act providing some form of synergism. In experienced centers, RFCA is now performed, regardless of whether the VT rate is rapid and/or is hemodynamically unstable. Newer mapping and ablation techniques are now available, enhancing the acute success rate of the procedure. In this review the most recent application of VT catheter ablation and the use of advanced mapping and ablation techniques will be discussed. (PACE 2006; 29:S40–S47)

Until recently, antiarrhythmic drugs were the treatment of choice in the majority of patients with recurrent ventricular tachycardia (VT), whatever the underlying heart disease. However, within 2 years more than 40% of patients treated for sustained VT experience recurrences.[1]

Furthermore, the risk of sudden cardiac death remains high despite drug therapy, especially in patients with depressed left ventricular function and/or hemodynamically poorly tolerated VT.[2] Serious side effects frequently limit the efficacy of antiarrhythmic drugs. The most effective drug, amiodarone, produces adverse effects within 5 years in almost 75% of patients, although not always serious enough to require withdraw of the drug.

Alternative treatment options are surgical ablation, placement of an implantable cardioverter-defibrillator (ICD), and radiofrequency catheter ablation (RFCA). Antiarrhythmic surgery, e.g., endocardial resection, is highly effective in selected cases in limiting recurrences by removal of large segment of subendocardium (often more than 30 cm^2). However, it is a major intervention associated with increased incidence of heart failure in the follow-up period.[3–6]

During the last decade, ICDs have proved superior to antiarrhythmic drug therapy for prolonging survival and preventing sudden cardiac death in survivors of sustained ventricular arrhythmias.[7–9] Subsequently, ICDs were shown to be prognostically beneficial for primary prevention of ventricular arrhythmias in patients with coronary artery disease,[10,11] and their rate of implantation is rapidly growing all over the world.

Although ICDs prolong survival, they do not prevent VT recurrences, thus being a palliative and not a curative treatment. Within a year of implantation, 68% of patients experience arrhythmia recurrences[7]; sinus rhythm may be restored either by painless antitachycardia pacing or by a uncomfortable high voltage shock. Antitachycardia pacing is usually used for most monomorphic haemodynamically tolerated and slower VTs. In case of unstable VT or when pacing fails to restore sinus rhythm, a low energy cardioversion shock or defibrillation shock are required.

Individual tolerance of ICD shock varies, but frequent shocks are poorly tolerated virtually by all patients and significantly worsen the quality of life.[12] In some cases, though, recurrent or incessant VT causes multiple appropriate ICD shocks during a short period of time. This condition is known as arrhythmic storm, i.e., three or more episode of VT separated by more than 5 minutes during 24 hours that result in proper shocks by the ICD.[13] The incidence of this event ranges between 10% and 25%. It poses a significant threat on patient status and survival[14–16] A recent study by Mitchell et al.,[17] reports, indeed, that the mode of death in ICD patients is still sudden in 28% of cases, and that the most common mechanism is ventricular fibrillation or VT treated with appropriate shocks

Address for reprints: Paolo Della Bella, M.D., Institute of Cardiology, University of Milan, Centro Cardiologico, IRCCS, via Parea 4, 20138 Milan, Italy. Fax +390258002398; e-mail: paolo.della bella@ccfm.it

and followed by electromechanical dissociation. Many of these deaths may result from ICD shocks that are either too frequent, too closely spaced, or too large in magnitude. This hypothesis is supported by reports of severe haemodynamic deterioration after internal DC shocks during ICD implantation, particularly in patients with poor left ventricular function.[18]

Adequate adjustment of ICD programming and the use of antitachycardia pacing algorithms that reduce the number of shock to a tolerable level are important aspects of caring for this kind of patients. However, despite a multitude of programmable options, this is not an easy task. Many patients with episodes of VT terminated by antitachycardia pacing also receive shocks occasionally.[19]

At least 50% of patients with an ICD implanted receive concomitant antiarrhythmic drug therapy to reduce episodes of VT or to make antitachycardia pacing more effective by slowing the tachycardia rate.[20] Drugs therapy, however, may cause additional problems, and sometimes render antitachycardia pacing less effective rather than more effective. It is well known that some drugs, notably amiodarone, may increase the energy required for defibrillation. Thus, repeat defibrillation testing is necessary when drug therapy is instituted, to be sure that the maximal ICD output provides an adequate safety margin for defibrillation.

The decreased incidence of VT induced by antiarrhythmic drugs may have adverse effects: the tachycardia rate may slow below the detection rate of the ICD, or fall into the range where sinus tachycardia can also occur, making diagnosis difficult. Furthermore, drugs may slow the sinus rhythm, causing the patient to be paced, producing adverse haemodynamic effects from right ventricular pacing; or induce proarrhythmic effects.[21]

Thus, the development of treatment for the prevention of VT and/or ventricular fibrillation episodes and excessive ICD therapy, remains a worthwhile goal.

RFCA is a curative approach, and can be expected to reduce the frequency of recurrent VT in more than 70% of patients.[19,22–25] The procedure is indicated in the majority of cases in patients with an ICD previously implanted, in postinfarction cardiomyopathy as well in idiopathic dilated cardiomyopathy. The long-term efficacy of the ablation procedure in this setting is well established since it reduces or abolishes ICD discharges.

In experienced centers, RFCA is now performed, regardless of whether the VT rate is rapid and/or is hemodynamically unstable. Newer mapping and ablation techniques are now available enhancing the acute success rate of the procedure.

Conventional Mapping and RFCA of VT

Catheter ablation performed by a single catheter approach and conventional mapping technique achieves a high rate of acute and long-term success in patients with tolerated postinfarction VT.[24,26]

Optimal localization of the target area requires reproducible induction of the VT in the electrophysiologic laboratory and hemodynamic stability while endocardial mapping is undertaken to precisely localize the critical area(s) of re-entrant circuit. Targets for ablation are identified by the recording of isolated mid-diastolic potentials, earliest activation of the local electrograms, pace mapping in sinus rhythm, and/or the presence of concealed entrainment (Fig. 1).

The same mapping and ablation techniques, the same acute and long term success rate can be performed and obtained in patient with stable VT causing excessive ICD therapy.[24,25]

Silva et al., recently reported that conventional RFCA is highly effective in controlling arrhythmic storm of tolerated VT in patients with previously implanted ICD.[13] They performed 18 procedures in 15 patients, the success rate being 80%, using conventional and/or cooled-tip catheters. In two patients, with a failed endocardial approach and in one patient with left ventricular apex aneurysm and a thrombus, RFCA was successfully performed with epicardial approach. The extensive involvement of the epicardium in the genesis of VT, particularly in idiopathic dilated cardiomyopathy, can be documented in almost 30% of patients undergoing catheter ablation, as recently demonstrated by Soejima et al.[27]

Electrical storms of monomorphic and tolerated arrhythmias are, however, unusual as the situation usually involves rapid and hemodynamically unstable VT. Silva et al., demonstrated that kind of electrical storms in only 3% of patients with ICD. The majority of patients, particularly those with electric storms, presented with pleomorphic or untolerated forms of VT requiring three dimensional mapping systems or hemodynamic support that allows conventional point to point mapping techniques.

Conventional Catheter Ablation with the Aid of a Percutaneous Cardiopulmonary Support

Percutaneous cardiopulmonary support (pCPS) has proven to be a technique of value in high risk coronary patients undergoing percutaneous balloon angioplasty and in the management of cardiac emergencies characterized by acute lowering of cardiac output.[28–30]

RFCA coupled with pCPS system has been attempted in one emergency setting (incessant sustained untolerated VT) in a series of 17 consecutive

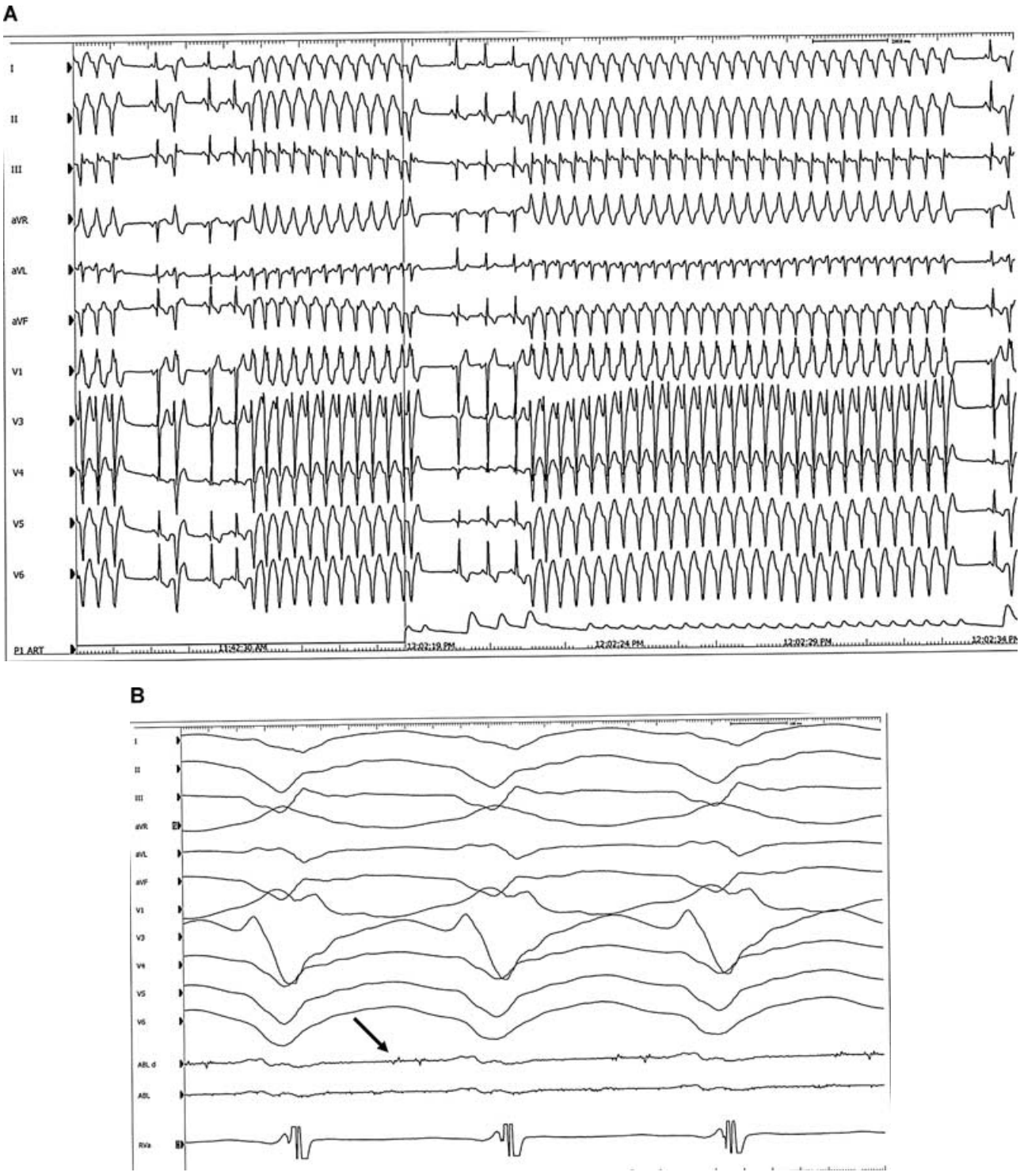

Figure 1. *Conventional catheter ablation of tolerated postinfarction ventricular tachycardia (VT). Panel A: 12-lead ECG showing the clinical arrhythmia presenting as iterative VT. Panel B: simultaneous recording of surface ECG and electrograms from the ablation site with low amplitude mid-diastolic potential indicated by arrow. Panel C: when the radiofrequency is delivered at the ablation site, termination of VT occurs in few seconds. RF = radiofrequency.*

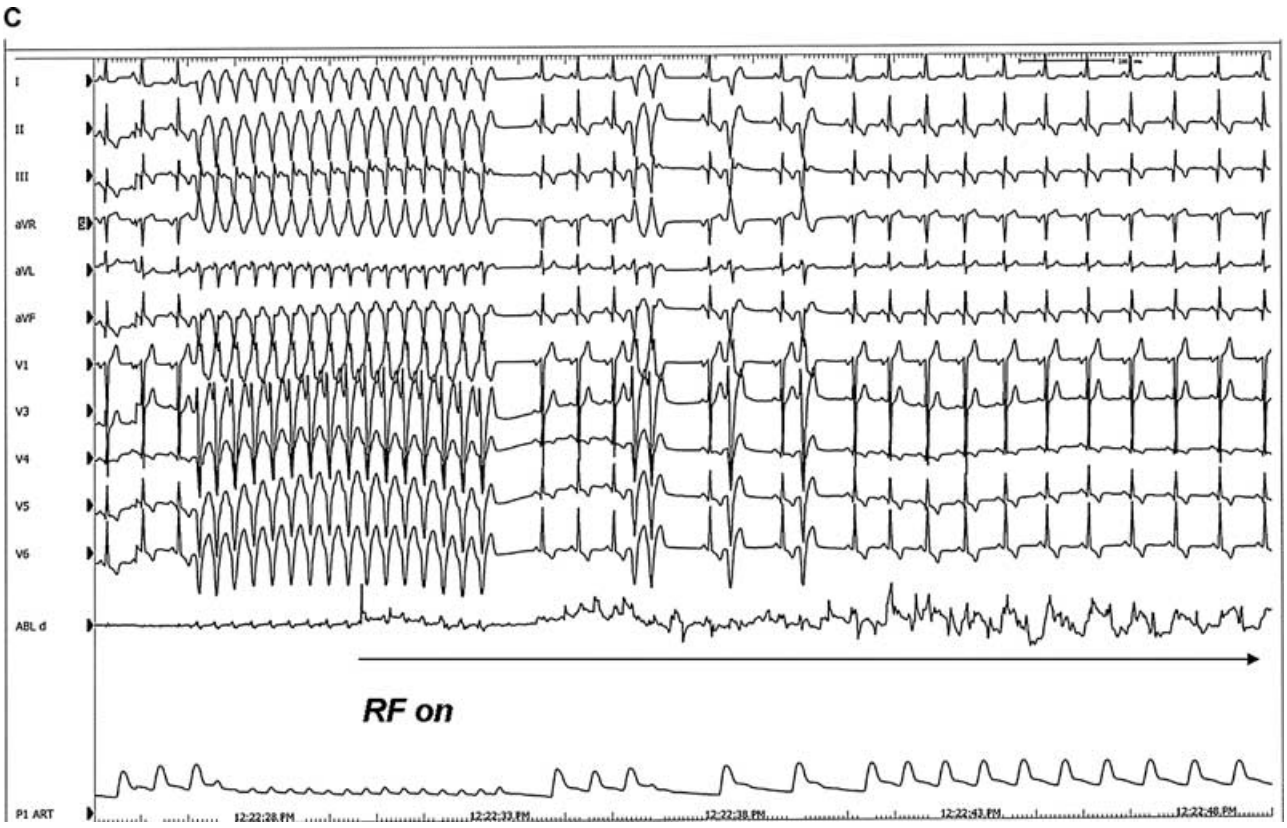

Figure 1. *Continued.*

patients at our institution. In all cases the arrhythmia was related to coronary artery disease, idiopathic dilated cardiomyopathy, or arrhythmogenic right ventricular dysplasia. (mean left ejection fraction 35%).[31] Fourteen of them experienced excessive ICD therapies (3–15 shocks/day), and three drug refractory VT associated with hemodynamic collapse. In the electrophysiologic laboratory we induced 58 episodes of sustained monomorphic VT (mean cycle length 317 ± 43 ms); 54/58 VTs were hemodynamically unstable requiring activation of pCPS to achieve and maintain a mean blood pressure of 79 ± 9 mmHg. Arterial and femoral sheats (15 and 17 Fr) were placed in the left femoral artery and vein, and connected to a pump coupled with an oxygenator and a heat exchanger; blood flow rates ranged from 1 to 6 L/min.

Termination and prevention of inducibility was obtained in 77% of VTs; acute clinical success was obtained in 59% of patients. We recorded few periprocedural complications: one patient experienced transient left ventricular stunning following pCPS interruption, one a periprocedural transient ischemic attack, and two a mild form of anemia.

At a mean follow up of 36 months (range 9–51) five patients (29%) remained free of recurrences of the ablated VTs; six experienced recurrences but with a significant reduction in the number of ICD therapies (1–5 shocks/month). No late adverse events related to the pCPS institution were observed.

In our experience pCPS has proved to be a safe and efficacy alternative strategy to map and ablate untolerated VT. It may not represent a procedural alternative to electroanatomic or noncontact mapping, but rather a possibility of performing catheter ablation, otherwise not feasible, in particular and emergency clinical setting like excessive ICD therapies or fast recurrent VT early after myocardial infarction or left ventricular surgery.

Noncontact Mapping (Endocardial Solutions)

Noncontact mapping allows an accurate reconstruction of the virtual three dimensional geometry of the left or right ventricle and the possibility to simultaneously map the entire endocardial surface, through the placement of a balloon mounted multielectrode array catheter.[32] This system offers the possibility of an off-line analysis of

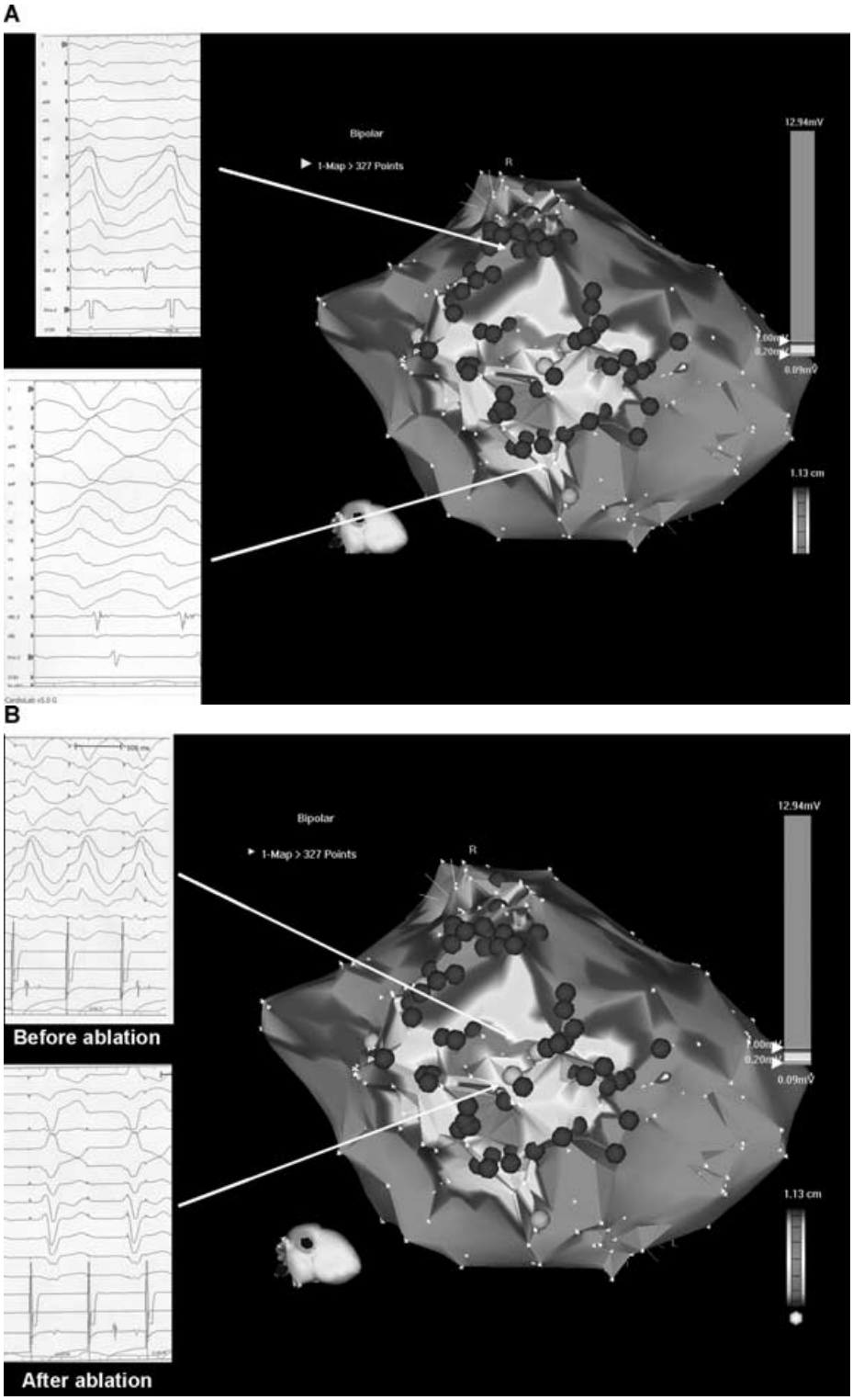

Figure 2. *Linear ablation of postinfarction ventricular tachycardia (VT) guided by CARTO mapping. Panel A: Bipolar voltage map obtained during sinus rhythm. VTs exit points are indicated by arrows. Red circles represent the lesion sites encircling the edge of the scar area. Panel B: successful linear ablation demonstrated by lack of capture by pacing from scar.*

each desired beat during either sinus rhythm or VT. Significant information can be obtained during VT on critical isthmus, and during sinus rhythm on the presence of areas of low voltage or scarring tissue by the analysis of activation pattern. Limitation of this mapping system relay on the need for two catheters in the left ventricle, and on the difficult encountered sometimes to attain a stable position of the balloon catheter. More over, the accuracy of the system decrease at increasing distance of the multi electrode array from the endocardium, as it may occur in dilated cardyomiopathy.

The use of noncontact system mapping allows RFCA of untolerated and nonsustained VT in different setting of underlying heart disease with high rate of acute an long-term success in patients with or without ICD previously implanted.[33–35]

Preliminary experience at our institution, reported in 2002, reported the use of noncontact mapping system to guide RFCA in 17 patients with poorly tolerated VT in prior myocardial infarction (11 patients), arrhythmogenic right ventricular dysplasia (3 patients), and idiopathic dilated cardiomyopathy (3 patients).[36] Four patients underwent to RFCA for excessive ICD therapy discharge. Twenty seven episodes of VT were induced during the procedures, and a satisfactory mapping could obtained in all episodes. Identification of the exit point or the diastolic pathway guide the ablation procedure. The overall acute success rate was 64%, being significantly greater when targeting the diastolic pathway (78%) as compared to the exit point (16%) of the VT. Over the long term the recurrence rate was lower when prevention of all VT was achieved at the postablation electrophysiological study.

Electro Anatomic Mapping System (CARTO)

Catheter ablation of unstable VT can be performed during sinus rhythm with the guide of electroanatomic mapping system (CARTO).[22,37,38] The system allows a three-dimensional reconstruction of the left ventricle coupled with both a voltage map (defining the scar area) and the activation map (localizing areas of latest activation during sinus rhythm) and displaying pattern of endocardial activation during VT[22] whenever the arrhythmia is tolerated.

Sra et al., recently show that electro anatomic mapping is helpful in identifying sites for catheter ablation in 19 patients with prior myocardial infarction and refractory VT causing multiple ICD shocks (4–62 shocks during the 12 week preablation period).[39] After the procedure, they observed that 66% of patients had no recurrence of VT during a mean follow up of 26 ± 8 weeks.

Table I.

Baseline Population Characteristics of Patients Undergoing VT Catheter Ablation

	Pts	ICD
Left VTs		
Post-MI	107	88 (82%)
IDCM	32	14 (44%)
Right VTs		
ARVD	40	35 (88%)

Pts = patients; VT = ventricular tachycardia; MI = myocardial infarction; ICDM = idiopathic dilated cardiomyopathy; ARVD = arrhythmogenic right ventricular dysplasia.

Catheter Ablation of VT at our Institution

From January 2002 to March 2005 we performed RFCA in 139 patients with left VT (107 pts with prior myocardial infarction; 32 patients with idiopathic dilated cardiomyopathy) and in 40 patients with right VT (arrhythmogenic right ventricular dysplasia, ARVD). About 80% of them have an ICD previously implanted and were referred to our Institution for VT causing excessive discharge of therapy (including true arrhythmic storm 28% of the cases) (Table I). The characteristics of the ventricular arrhythmias, clinical and induced during EP study, are shown in Table II.

RFCA guided by electroanatomic mapping system CARTO was performed in 23% of patients with previously implanted ICD and recurrent postinfarction VT. However, the percentage reach 100% in the last year; this data supporting the fact that majority of this kind of patients need nonconventional mapping system cause of the presence of untolerated and/or pleomorphyc VT. An example of RFCA of postinfarction VT in an ICD patients is shown in Figure 2: linear ablation was performed to encircle the edge of the

Table II.

Characteristics of Clinical and Induced VTs in EP Study

		VTs		
	Pts	Monomorphic	Pleomorphic	Incessant
Left VTs	102			
Post-MI	88	60 (68%)	29 (32%)	10 (11%)
IDCM	14	12 (85%)	2 (14%)	3 (21%)
Right VTs				
ARVD	35	26 (75%)	9 (25%)	3 (8%)

Abbreviations are as in Table I.

Table III.

Acute Procedural Outcome

	Pts	Results Pts (%)		
		Class A	Class B	Class C
Left VTs	102			
Post-MI	88	63 (71%)	18 (21%)	7 (8%)
IDCM	14	8 (57%)	4 (28%)	2 (14%)
Right VTs				
ARVD	35	23 (65%)	10 (29%)	2 (6%)

Abbreviations are as in Table I.

scar area during sinus rhythm; after effective ablation no capture can be obtained at the VT exit site.

Acute results of the procedures were shown in Table III. According to the results of the postprocedure electrophysiological study the acute outcome of the RFCA was defined as follows: Class A, interruption of the index VT and of all the induced VTs, and prevention of induction of any sustained ventricular arrhythmia; Class B, interruption of the index and/or other induced VTs, but persistent inducibility of other sustained VTs; Class C, failure at terminating the index VT.

Over the long term, prevention of any ICD therapy was obtained in 72% of patients with prior myocardial infarction, 64% of patients with idiopathic dilated cardiomyopathy, and 75% of patients with ARVD. In the remaining patients, however, a significant (more than 85%) reduction of the ICD discharge was obtained. Furthermore, when RFCA was guided by nonconventional mapping the acute success rate was still greater: 80% in patients with myocardial infarction and idiopathic dilated cardiomyopathy, and 72 in ARVD patients. Details are shown in Table IV.

Table IV.

Differences of Acute Procedural Outcome Related to Conventional and Nonconventional Mapping

	Pts	Results Success Pts (%)	
		All	Nonconventional Mapping
Left VTs	102		
Post-MI	88	64 (72%)	32/40 (80%)
IDCM	14	9 (64%)	9/11 (81%)
Right VTs			
ARVD	35	23 (65%)	18/25 (72%)

Abbreviations are as in Table I.

Conclusions

The large diffusion of ICD has dramatically reduced sudden cardiac death from rapid VT and/or ventricular fibrillation. Furthermore, it is contributing to the growth of a novel population of patients with frequently recurrent VTs, and originating a broad clinical spectrum ranging from unpleasant frequent ICD shock interventions to life threatening arrhythmia storms. In this setting the hybrid therapy may be viewed as a synergistic form of treatment characterized by a combination of: (1) a potentially curative catheter ablation aimed at the modification of the arrhythmia substrate to eliminate or decrease the potential for arrhythmia recurrences, (2) a backup ICD, as a protection against the risk of sudden death due to unpredictable or untreatable sudden events.

References

1. The ESVEM Investigators. Determinants of predicted efficacy of antiarrhythmic drugs in the electrophysiologic study versus electrocardiographic monitoring trial. Circulation 1993; 87:323–329.
2. Wyse DG, Talajic M, Hafley GV, Buxton AE, Mitchel LB, Kus TK, Packer DL, et al. Antiarrhythmic drug therapy in the multicenter unsustained tachycardia trial (MUSTT): Drug testing and as treated analysis. J Am Coll Cardiol. 2001; 38:344–351.
3. Krafchek J, Lawrie GM, Roberts R, Magro SA, Wyndham CRC. Surgical ablation of ventricular tachycardia; improved results with a map directed regional approach. Circulation 1986; 73:1239–1247.
4. Miller JM, Marchlinski FE, Harken AH, Hargrove WC, Josephson ME. Subendocardial resection for sustained ventricular tachycardia in the early period after acute myocardial infarction. Am J Cardiol. 1985; 55:980–984.
5. Niebauer MJ, Kirsh M, Kadish A, Calkins H, Morady F. Outcome of endocardial resection in 33 patients with coronary artery disease: Correlation with ventricular tachycardia morphology. Am Heart J. 1992; 124:1500–1507.
6. Van Hemel NM, Kingma JH, Defauw JJ, Hoogteijling-van Dusseldorp E, Kelder JC, Beukeme WP, Vermeulen FE. Continuation of antiarrhythmic drugs, or arrhythmia surgery after multiple drug failure. A randomized trial in the treatment of postinfarction ventricular tachycardia. Eur Heart J 1996; 17:564–573.
7. The Antiarrhythmics versus Implantable Defibrillators (AVID) Investigators. A comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from near fatal ventricular arrhythmias. N Engl J Med 1997; 337:1576–1583.
8. Connoly SJ, Gent M, Roberts RS, Dorian P, Roy D, Sheldon RS, Mitchell LB, Green MS, Kleibn GJ, O'Brien B. Canadian implantable defibrillator study (CIDS): A randomized trial of the implantable defibrillator against amiodarone. Circulation. 2000; 101:1297–1302.
9. Kuck KH, Cappato R, Siebels J, Ruppel R. Randomized comparison of antiarrhythmic drug therapy with implantable defibrillators in patients resuscitated from cardiac arrest. The cardiac arrest study Hamburg (CASH). Circulation. 2000; 102:748–754.
10. Moss AJ, Hall WJ, Cannom DS, Daubert JP, Higgins SL, Klein H, Levine JH, et al. Improved survival with an implanted defibrillator in patients with coronary artery disease at high risk for ventricular arrhythmia. Multicenter Automatic Defibrillator Implantation Trial Investigators. N Engl J Med. 1996; 335:1933–1940.
11. Moss AJ, Zareba W, Hall WJ, Klein H, Wilber DJ, Cannom DS, Daubert JP, et al., Multicenter Automatic Defibrillator Implantation

Trial II Investigators. Prophylactic implantation of a defibrillator in patients with myocardial infarction and reduced ejection fraction. N Engl J Med. 2002; 346:877–883.
12. Sears SF, Todaro JF, Urizar G, Lewis TS, Sirois B, Wallace R, Sotile W, et al. Assessing the psychosocial impact of the ICD: A national survey of the implantable cardioverter defibrillator health care providers. Pacing Clin Electrophysiol 2000; 23:939–945.
13. Silva RMFL, Mont L, Nava S, Rojel U, Matas M, Brugada J. Radiofrequency catheter ablation for arrhythmic storm in patients with an implantable cardioverter defibrillator. Pacing Clin Electrophysiol 2004; 27:971–975.
14. Villacastin J, Almendral J, Arenal A, Albertos J, Ormaetxe J, Peinado R, Bueno H, et al. Incidence and clinical significance of multiple consecutive, appropriate, high energy discharges in patients with implanted cardioverter defibrillators. Circulation 1996; 93:753–762.
15. Credner SC, Kingenheben T, Mauss O, Sticherling C, Hohnloser SH. Electrical storm in patients with transvenous implantable cardioverter defibrillators. J Am Coll Cardiol. 1998; 32:1909–1915.
16. Exner DV, Pinski SL, Wyse G, Rentroe EG, Follmann D, Gold M, Beckman KJ, et al. and the AVID Investigators. Electrical storm presages non sudden death- The Antiarrhythmics Versus Implantable Defibrillators (AVID) Trial. Circulation 2001; 103:2066–2071.
17. Mitchell LB, Pineda EA, Titus JL, Bartosch PM, Benditt DG. Sudden death in patients with implantable cardioverter defibrillators. The importance of post shock electromechanical dissociation. J Am Coll Cardiol. 2004; 39:1323–1328.
18. Steinbeck G, Dorwarth U, Mattke S, Hoffmann E, Morkewitz A, Kaulbach H, Tossani P. Hemodynamic deterioration during ICD implant: Predictors of high risk patients. Am Heart J. 1994; 127:1064–1067.
19. Stevenson WG, Friedman PL, Sweeney MO. Catheter ablation as an adjunct to ICD therapy. Circulation. 1997; 96:1378–1380.
20. Santini M, Pandozi C, Ricci R. Combining atiarrhythmic drugs and implantable devices therapy: Benefits and outcome. J Interv Card Electrophysiol. 2000; 4:65–68.
21. Soejima K, Stevenson WG. Ventricular tachycardia associated with myocardial infarct scar. A spectrum of therapies for a single patient. Circulation. 2002; 106:176–179.
22. Marchlinski FE, Callans DJ, Gottlieb CD, Zado E. Linear ablation lesion for control of unmappable ventricular tachycardia in patients with ischemic and non ischemic cardiomyopathy. Circulation. 2000; 101:1288–1296.
23. Soejima K, Suzuki M, Maisel WH, Brunckhorst CB, Delacretaz E, Blier L, Tung S, et al. Catheter ablation in patients with multiple and unstable ventricular tachycardias after myocardial infarction: Short ablation lines guided by reentry circuit isthmuses and sinus rhythm mapping. Circulation 2001; 104:664–669.
24. Della Bella P, De Ponti R, Uriarte JAS, Tondo C, Klersy K, Carbucicchio C, Storti C, et al. Catheter ablation and antiarrhyhmic drugs for haemodinamically tolerated post-infarction ventricular tachycardia. Long term outcome in relation to acute electrophysiological findings. Eur Heart J. 2002; 23:414–424.
25. Strickberger SA, Man Kc, Daoud EG, Goyal R, Brinkman K, Hasse C, Bogun F, et al. A prospective evaluation of catheter ablation of ventricular tachycardia as adjuvant therapy in patients with coronary artery disease and implantable cardioverter defibrillators. Circulation. 1997; 96:1378–1380.
26. O'Donnel D, Burke JP, Anilkumar R, Simeonidou E, Furniss SS. Radiofrequency ablation for post infarction ventricular tachycardia: Report of a single centre experience of 112 cases. Eur Heart J. 2002; 23:1699–1705.
27. Soejima K, Stevenson WG, Sapp JL, Selwyn AP, Couper G, Epstein LM. Endocardial and epicardial radiofrequency ablation of ventricular tachycardia associated with dilated cardiomyopathy: The importance of low voltage scars. J Am Coll Cardiol. 2004; 43:1834–1842.
28. Guarneri EM, Califano JR, Shatz RA, Morris NB, Teirstein PS. Utility of standby cardiopulmonary support for elective coronary interventions. Catheter Cardiovasc Interv. 1999; 46:32–35.
29. Suarez de Lezo J, Pan M, Medina A, Pavlovic D, Romero M, Segua J, Ruiz M, et al. Percutaneous cardiopulmonary support in critical patients needing coronary interventions with stents. Catheter Cardiovasc Interv. 2002; 57:467–475.
30. Kurusz M, Zwischenberger JB. Percutaneous cardiopulmonary bypass for cardiac emergency. Perfusion. 2002; 17:269–277.
31. Fassini G, Della Bella P, Trevisi N, Tondo C, Riva S, Carbucicchio C, Bartorelli A. Long term safety and outcome of hemodynamically-supported catheter ablation for the treatment of untolerated ventricular tachicardia. Pacing Clin Electrophysiol 2002; 25:533 (abs.)
32. Schilling RJ, Peters NS, Davies DW. Feasibility of a noncontact catheter for endocardial mapping of human ventricular tachycardia. Circulation 1997; 96:1525–1531.
33. Strickberg SA, Kinght BP, Michaud GF, Pelosi F. Morady F. Mapping and ablation of ventricular tachycardia guided by virtual electrograms using a noncontact, cumputerized mapping system. J Am Coll Cardiol 2000; 35:414–421.
34. Ciaccio EJ, Chow AW, Davies DW, Wit AL, Peters NS. Localization of the isthmus in reentrant circuits by analysis of electrograms derived from clinical noncontact mapping during sinus rhythm and ventricular tachycardia. J Cardiovasc Electrophysiol 2004; 15:27–36.
35. Segal OR, Chow AW, Markides V, Schilling RJ, Peters NS, Davies DW. Long term results after ablation of infarct related ventricular tachycardia. Heart Rhythm 2005; 2:474–482.
36. Della Bella P, Pappalardo A, Riva S, Tondo C, Fassini G, Trevisi N. Noncontact mapping to guide catheter ablation of untolerated ventricular tachycardia. Eur Heart J 2002; 23:742–752.
37. Stevenson WG, Delacretaz E, Friedman PL, Ellison KE. Identification and ablation of macroreentrant ventricular tachycardia with the CARTO electroanatomic mapping system. Pacing Clin Electrophysiol 1998; 21:1448–1456.
38. Kottkamp H, Wetzel U, Schirdewahn P, Dorszewski A, Gerds-Li JH, Carbucicchio C, Kobza R, et al. Catheter ablation of ventricular tachycardia in remote myocardial infarction: Substrate description guiding placement of individual linear lesions targeting noninducibility. J Cardiovasc Electrophysiol. 2003; 14:675–681.
39. Sra J, Bhatia A, Dhala A, Black Z, Deshpande S, Cooley R, Akhtar M. Electroanatomically guided catheter ablation of ventricular tachycardias causing multiple defibrillator shocks. Pacing Clin Electrophysiol 2001; 24:1645–1652.