Journal of the American College of Cardiology
Vol. 35, No. 6, 2000

ISSN 0735-1097/00/$20.00
Published by Elsevier Science Inc.
PII S0735-1097(00)00606-9

Nonsurgical Transthoracic Epicardial Catheter Ablation to Treat Recurrent Ventricular Tachycardia Occurring Late After Myocardial Infarction

Eduardo Sosa, MD, Mauricio Scanavacca, MD, André d'Avila, MD, Flávio Oliveira, MD, José Antônio F. Ramires, MD, FACC

São Paulo, Brazil

OBJECTIVES We sought to evaluate feasibility, safety and results of transthoracic epicardial catheter ablation in patients with ventricular tachycardia occurring late after an inferior wall myocardial infarction.

BACKGROUND Transthoracic epicardial catheter ablation effectively controls recurrent ventricular tachycardia (VT) in patients with Chagas' disease in whom epicardial circuits predominate. Epicardial circuits also occur in postinfarction VT.

METHODS Fourteen consecutive patients aged 53.6 ± 14.5 years with postinfarction VT related to the inferior wall were studied. The VT cycle length was 412 ± 51 ms. Two patients had previously undergone unsuccessful standard endocardial radiofrequency energy (RF) ablation. The VT was incessant in one patient. Left ventricular angiography showed inferior akinesia in 13 patients and an inferior aneurysm in 1 patient. Ablation was performed with a regular steerable catheter placed into the pericardial sac by pericardial puncture.

RESULTS The pericardial space was reached in all patients. Electrophysiologic evidence of an epicardial circuit was present in 7 of 30 VTs. Due to a high stimulation threshold, empirical thermal mapping was the only criterion used to select the site for ablation. Three VTs were interrupted during the first RF pulse. Two pulses were necessary to render it noninducible in 3 patients (1 VT per patient). In the remaining 4 VTs, 3, 3, 4 and 5 RF pulses, respectively, were used. The overall success was 37.14% (95% confidence interval, 11.83% to 62.45%). Patients are asymptomatic for 14 ± 2 months.

CONCLUSIONS Postinfarction pericardial adherence does not preclude epicardial mapping and ablation to control VT related to an epicardial circuit in postinferior wall myocardial infarction. (J Am Coll Cardiol 2000;35:1442–9) © 2000 by the American College of Cardiology

Nonsurgical transthoracic epicardial catheter ablation has been introduced recently as an alternative approach to treat patients with Chagas' disease and recurrent ventricular tachycardia (VT) related to a subepicardial circuit (1,2). Briefly, this procedure consists of introducing a standard 4-mm tip ablation catheter into the pericardial space by a transthoracic pericardial puncture (1–3), similar to the procedure Krikorian and Hancock (4) described to drain pericardial effusions.

This novel approach may be instrumental in some patients because possible subepicardial target sites have been implicated in the failure of standard endocardial catheter ablation (5,6,11–16). Although intraoperative mapping suggests that epicardial circuits are common in postinfarction VT related to the left ventricular inferior wall (7–11), to our knowledge, transthoracic catheter epicardial ablation has never been attempted in post–myocardial infarction (MI) VT. Given the initial successful results obtained with the transthoracic epicardial catheter ablation approach in patients with Chagas' disease in whom epicardial circuits predominate (1–3), we speculated that this technique could be useful in patients with postinfarction VT related to an inferior MI. However, the presence of incidental pericardial adherence associated with transmural MI could impose a

See page 1450

From the Heart Institute (InCor), University of São Paulo Medical School, São Paulo, Brazil. Presented in abstract form at the 19th Scientific Sessions of the NASPE, San Diego, California, May 1998.

Manuscript received July 26, 1999; revised manuscript received November 11, 1999, accepted February 2, 2000.

Abbreviations and Acronyms

MI = myocardial infarction
RF = radiofrequency energy
VT = recurrent ventricular tachycardia

limitation for this approach in postinfarction patients (17,18). Thus, we sought to evaluate the feasibility, efficacy and safety of transthoracic epicardial catheter ablation in patients with postinfarction VT.

METHODS

Patient population. From October 1996 to August 1998, 14 consecutive patients with recurrent and drug-refractory postinfarction VT were referred to the Group of Arrhythmia of the Heart Institute of the University of São Paulo Medical School in Brazil. All patients underwent diagnostic electrophysiologic study. Recurrent ventricular tachycardia was defined as well tolerated to allow adequate mapping when systolic pressure was >70 mm Hg with no signs of low cerebral flow or cardiac output. These patients were selected to undergo radiofrequency energy (RF) ablation. The VT was incessant in one patient and two other patients had previously undergone unsuccessful standard endocardial RF ablation. An implantable cardioverter-defibrillator had been implanted in one of them.

Mean age was 53.6 ± 14.5 years and 10 patients were male. All patients had an old inferior MI. At least two episodes of recurrent monomorphic VT had been documented by 12-lead ECG in all patients prior to hospital admission. Clinical VT had a right bundle branch block QRS configuration with left axis deviation in five patients and a left bundle branch block with left axis deviation in nine patients. Mean VT cycle length was 412 ± 51 ms. Recurrences had occurred in 10 patients despite the use of several antiarrhythmic drugs.

Time elapsed between acute MI and the first episode of VT varied from 8 to 108 months. Clinical manifestations during such episodes included precordial palpitations in nine patients and syncope in five patients.

Mean left ventricular ejection fraction determined by two-dimensional echocardiogram was 50 ± 12%. All patients underwent coronary artery and left ventricular angiography. Total occlusion of right coronary artery was observed in 11 patients. In three patients, the right coronary artery was patent. The left coronary artery was free of lesions in five patients. Two patients had a 50% reduction of coronary lumen: one in the left circumflex artery and one in the left anterior descending coronary artery, respectively. Left ventricular angiography showed inferior akinesia in 13 patients and a large inferior aneurysm in 1 patient. All other left ventricular wall segments were normal in these patients.

Mapping and RF ablation. The study protocol was approved by the Scientific and Ethical Committee of the University of São Paulo in Brazil. Informed consent was obtained from all patients before ablation. The procedure was performed in the electrophysiology laboratory with patients in a fasting state. Deep sedation was achieved with intravenous midazolam and fentanyl.

Mapping catheters were introduced in the following sequence: two multipolar catheters were positioned in the coronary sinus and right ventricular apex through femoral venous puncture. Programmed ventricular stimulation was performed at twice the diastolic threshold with a 1-ms pulse, with as many as three extrastimuli from the apex and outflow tract of the right ventricle, at two-drive cycle lengths. If induced VT was hemodynamically tolerated, a subxiphoid puncture was made to introduce a conventional deflectable quadripolar RF catheter into the pericardial space (1–4).

Electrophysiologic signals were recorded (using a PC Electrophysiologic Measurement System; EMS 4.2; University of Limburg, The Netherlands) at a paper speed of 100 to 300 mm/s. Gain applied to the endocardial and electrocardiographic signals was 10 mm/mV, while the epicardial signals (i.e., coronary sinus and epicardial signals) were registered at 20 mm/mV and at a filter setting between 50 Hz and 500 Hz.

Approaching the pericardial space. Transthoracic puncture was done after proper asepsis of the subxyphoid area. A regular needle that is used to administer epidural anesthesia (a Tuohy 17-Gr needle with a 79.4-mm effective length, a 101.6-mm overall length, and a 1.5-mm O.D. Abbott I/N# E622, Abbott Ireland Ltd.; Sligo, Republic of Ireland) was used for this procedure. The needle was introduced at a 45° angle toward the left scapula. Guided by fluoroscopy, the operator gently advanced the needle until it was close to the cardiac silhouette, where a light negative pressure was felt (Fig. 1).

Needle angle was adjusted according to the region the operator wished to evaluate. This region was most frequently the medial third of the right ventricle, where, based on the coronary angiography, no major coronary vessels can be found. Besides, the catheters placed at the right ventricular apex and in the coronary sinus are useful references to guide the needle tip.

In order to precisely demonstrate the site of the needle tip, 2 ml of contrast media was injected (loxitalamato de meglumina e sódio, Telebrix Coronar—Gulbert Produções Ltda, Rio de Janeiro, Brazil) as the needle approached the heart silhouette. If the needle was outside the pericardial space, contrast media accumulated in the mediastinum. However, when the needle tip was inside the pericardial space, contrast medium could be seen surrounding the cardiac silhouette (Fig. 2). This thin layer of contrast in the pericardial space confirmed that the needle was correctly positioned. Finally, a soft floppy tip guidewire was intro-

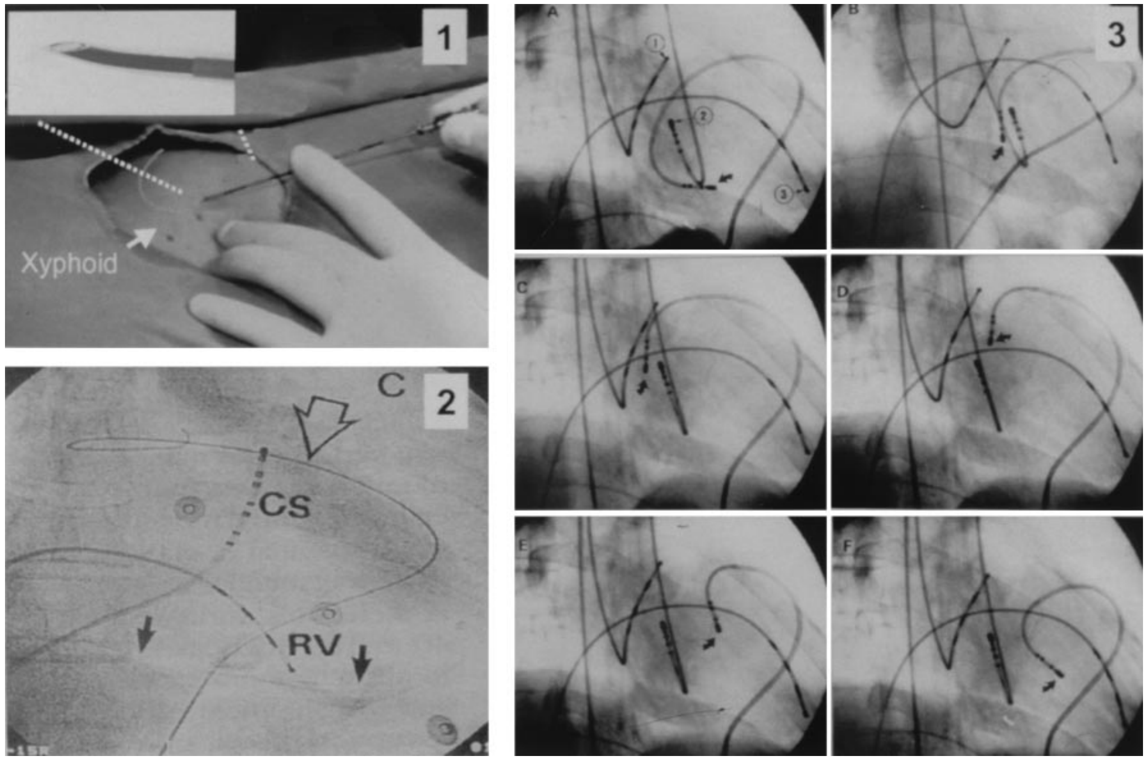

Figure 1. Technique used to insert the mapping and ablation catheter in the pericardial space. The needle used for epidural anesthesia (detailed in the **left superior corner**) being currently used for transthoracic pericardial puncture, according to the technique described by Kirkorian, to drain epicardial effusions **(panel 1)**. A soft floppy-tip guidewire is introduced into the pericardial space **(large arrow)**, where contrast is also present **(black arrows) (panel 2)**. Then, an 8F introducer is advanced, the guidewire is removed and a quadripolar, 4-mm tip, 7F regular steerable catheter is gently introduced into the pericardial sac to perform epicardial mapping and/or ablation. **Panel 3** demonstrates a right anterior oblique view at 60° obtained by fluoroscopy during epicardial mapping procedure. The epicardial catheter **(arrow)** is manipulated and placed in different locations of the epicardial space (**A** to **F**), where epicardial electrograms are obtained. RV = right ventricular apex catheter; CS = coronary sinus; 1 = coronary sinus catheter; 2 = endocardial left ventricular catheter; 3 = right apical endocardial catheter.

duced in the pericardial space through the puncture needle. As a general rule, the guidewire would easily slip into the pericardial space.

Guidewire position was also monitored with fluoroscopy. Eventually, an 8-F introducer was advanced, the guidewire was removed and a quadripolar deflectable catheter with a 4-mm tip was inserted into the pericardial space for mapping and ablation (Fig. 1) that could be easily mapped because there are no papillary muscle or thrombi in the pericardial sac to restrict catheter movement.

Whenever epicardial mapping failed to identify an epicardial circuit, a fourth regular deflectable catheter was advanced through a femoral artery and retrogradely across the aortic valve to allow endocardial mapping of the left ventricular cavity. Because transthoracic puncture ideally should be performed before giving the patient heparin, only patients in whom endocardial mapping became necessary underwent systemic anticoagulation, which was achieved with administration of an intravenous bolus of 5,000 U heparin followed by hourly doses of 1,000 U heparin. Both epicardial and endocardial catheters had a 4-mm distal tip electrode and 2-mm to 5-mm interelectrode spacing.

Selection and ablation of the target site. Adequate target sites were selected in a stepwise fashion. When fractionated electrograms or late potentials were found during sinus rhythm, VT was induced by programmed stimulation. After VT induction, the ablation catheter was moved until an early epicardial electrogram, an isolated middiastolic potential or continuous diastolic electrical activity was found. Bipolar pacing was attempted at the mapping site during VT at cycle lengths of 40 to 100 ms shorter than the VT cycle length to allow demonstration of concealed entrainment and measurement of the returning cycle after interrupting epicardial pacing. Unipolar stimulation was never used during this study. Criteria used to map and interpret electrograms related to a reentrant circuit were those suggested by Stevenson et al. (16).

If epicardial stimulation was not possible due to a high stimulation threshold, an empirical *thermal mapping* technique, which consists of applying RF pulses of 60°C for 10 s, was used to determine whether heat could terminate VT. If VT was interrupted within the 10 s, application was maintained for 30 s. If VT was not modified within the first 10 s, the catheter was manipulated until better electrograms

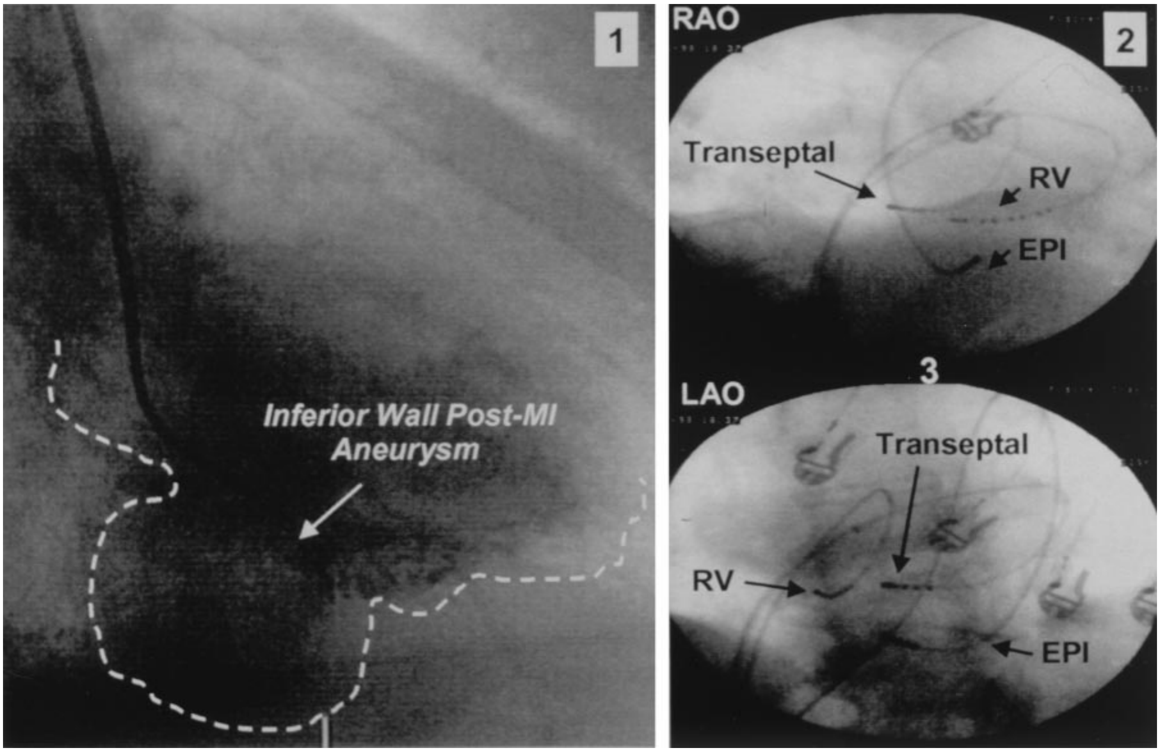

Figure 2. **Panel 1** shows a contrasted ventriculography in systole at 30° right anterior oblique view in a patient with postinfarction VT. Notice a large inferolateral aneurysm **(dotted lines)**. The epicardial catheter (EPI) positioned in the aneurysmatic area for mapping in the right (RAO) anterior oblique **(panel 2)** and left (LAO) anterior oblique **(panel 3)** views at 30° are shown. RV = right endocardial apical catheter; Transeptal = quadripolar catheter inserted in the left basal inferior wall through transeptal puncture due to advanced aortic disease.

could be obtained and new *thermal mapping* pulse was applied. Epicardial RF energy pulses were delivered between the distal tip of the epicardial catheter and an indifferent 110-cm^2 patch electrode located on the patient's left scapula. An application was defined as successful if the VT terminated during the energy application and was no longer inducible by programmed ventricular before and after the intravenous isoproterenol infusion.

In patients who underwent epicardial ablation, special attention was given to the distance between coronary arteries and the distal electrode of the ablating catheter. Theoretically, the risk of damaging epicardial vessels during application of RF energy increases as the catheter comes closer to the coronary artery. The distance between the vessel and the ablation catheter tip was estimated by the following: 1) analyzing the distance between the apex of the heart, where large epicardial vessels are unlikely to be present, and the ablating and coronary sinus catheters; 2) observing the distance between the ablating catheter and a coronary artery during left coronary arteriography obtained before the first pulse was applied (subsequent pulses were also delivered taking this angiography as a reference); or 3) observing the distance between the ablating catheter and epicardial veins of the heart during retrograde injection of contrast media into the coronary sinus. It was assumed that an important coronary artery is close to a major epicardial vein.

Based on the size of the lesion produced by RF applications in experimental studies (19,20), we have established empirically that the smallest acceptable distance between the catheter tip and a major coronary artery should be 12 mm (3 times the length of the catheter tip). If the ablation catheter was closer to an artery or vein than 12 mm, RF pulse was not delivered. Moreover, the ST-T segment was simultaneously monitored in the 12-lead electrocardiogram after VT interruption to detect any possible coronary artery lesion during RF application.

Postablation follow-up. An echocardiographic examination of the heart was performed after the procedure and on the day the patient was discharged from the hospital. Creatine kinase and its MB fraction were measured every 6 h during the first 24 h after the procedures. Patients were discharged from the hospital seven days after the procedure when clinical condition allowed it and followed up in outpatient visits to the clinic weekly for the first month after the procedure and every three months thereafter.

Statistical analysis. Statistical analysis was performed by software (Statistical Analysis System program; SAS Institute Inc.; Cary, North Carolina). Descriptive data are presented as mean ± SD and *t* test was used to assess differences between continuous variables.

RESULTS

The pericardial space was reached in all patients with no complications. The epicardial catheter could be moved easily inside the pericardial sac, including the infarcted area. Resistance to the catheter movement was not found to be more important in the area of MI in comparison to other areas. In one patient with a large inferior wall aneurysm and therefore a transmural MI, pericardial adherence, if present, did not impose any limitation to catheter manipulation since the entire epicardial surface of the aneurysm could be covered by epicardial mapping (Fig. 2).

A total of 30 VTs were induced in the 14 patients. Seven VTs were interrupted by an epicardial application (39% of all mappable VTs), whereas 3 VTs were interrupted by endocardial ablation (17% of all mappable VTs). Neither epicardial nor endocardial ablation could interrupt the other 8 VTs (46% of all mappable VTs). Due to poor hemodynamic tolerance, 12 VTs could not be mapped at all. Analysis of the 12-lead ECG did not reveal any useful finding capable of distinguishing between an epicardial and an endocardial VT.

Due to a high stimulation threshold (>15 mA and with a 3.5-ms pulse width), empirical thermal mapping was the only criterion used to select the site for ablation in all VTs. Three VTs were interrupted during the first RF pulse while two pulses were necessary to render it noninducible in three patients (one VT per patient). Interruption was obtained only in the remaining 4 VTs with 3, 3, 4 and 5 RF pulses, respectively. The overall success of ablation therapy in these patients was 37.14% (95% confidence interval, 11.83% to 62.45%).

The earliest epicardial activation site occurred 87 ± 13 ms before the onset of the QRS complex. During all applications, the mean power used was 35 ± 16 W; temperature, 56 ± 10°C; and impedance, 118.5 ± 15 omega. When all patients were considered, mean procedure duration was 210 ± 16 min and mean radiation exposure time was 22 ± 4 min of pulsed fluoroscopy at seven squares per second. Figures 3 and 4 illustrate examples of successful epicardial RF applications.

The procedure was well tolerated in all patients, and none of the patients experienced CK-MB elevation (4.2 ± 2 U/liter [normal value <10 U/liter]). Echocardiographic examination on the day of hospital discharge did not reveal pericardial effusion. No complications occurred during hospitalization. The seven patients with successfully ablated epicardial VTs were asymptomatic 14 ± 2 months after the procedure. One patient is taking oral amiodarone, one patient continues to take oral sotalol and five patients are not taking antiarrhythmic drugs.

DISCUSSION

The present study suggests that possible pericardial adherence after MI does not preclude epicardial mapping and ablation in patients with postinfarction VT related to an epicardial circuit. Additionally, it suggests that there is a high prevalence of epicardial circuits in patients with postinfarction VT related to an old inferior MI, reinforcing previous data obtained during surgical procedures. These results support the concept that transthoracic epicardial mapping and ablation may help controlling postinfarction epicardial VT.

Prior studies. It was not so long ago that epicardial mapping could be performed only during cardiac surgery, thus restricting it to the surgical theater. More recently, Arruda et al. (21) described the insertion of a multipolar catheter into the epicardial veins to obtain epicardial signals. However, mapping by this method is limited by the cardiac anatomy, and the technique is not helpful when the site of origin is not close enough to the epicardial vessels to be mapped and/or ablated.

The possibility of mapping an extensive epicardial surface permits electrical activity to be assessed at very close sites and therefore display more data about electrical activity of the heart. This is not usually possible with endocardial mapping because the presence of papillary muscles and chordae and repetitive ventricular systoles prevent free manipulation of the endocardial catheter.

Epicardial RF applications. Creation of small lesions produced by empirical use of *thermal* pulses of 60°C for 10 s instead of using pacing techniques to distinguish a bystander site from the common pathway is a limitation of this approach. Although VT interruption during application indicates that site participated in the reentrant circuit, lesions may not be large enough to interrupt wide pathways. Therefore, potential adequate sites for ablation might not be identified. The use of pulses at higher temperatures for longer periods in patients in whom ventricular capture is not possible might identify a larger number of epicardial circuits.

The necessity for multiple applications raises questions on how hazardous this procedure might be for the coronary arteries. However, because these pulses are applied in areas where scar tissue tends to predominate, applications will be delivered in the vicinity of occluded and previously damaged coronary arteries. It has been possible to identify and catheterize small coronary arteries responsible for blood supply to the site of origin or pathway of ventricular tachycardia in as many as 84% of patients with postinfarction VT (22). In the past years, occlusion of these vessels by transcoronary chemical ablation has not been found to produce deleterious consequences to the ventricular function (23,24). Similarly, applications of RF pulses close to these vessels should not be expected to give rise to serious complications. Actually, preliminary data suggest that it is

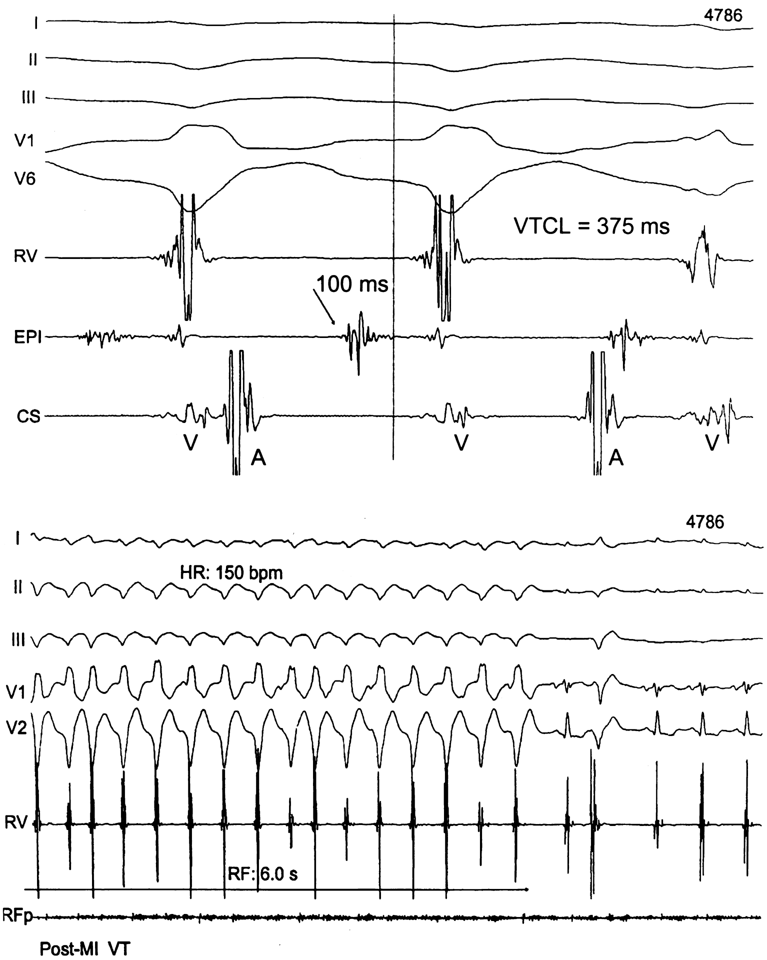

Figure 3. Activation mapping of ventricular tachycardia obtained during a transthoracic epicardial ablation procedure. The epicardial electrogram found in EPI precedes the onset of the QRS complex by 100 ms **(upper panel)** and it was used to guide ablation. Due to a high stimulation threshold, ventricular capture was not obtained during bipolar pacing from the epicardial catheter. At these sites, an epicardial application of RF interrupted VT within 6 s **(lower panel)** and rendered it noninducible. Electrocardiographic I, II, III, V_1, and V_6 leads are shown. VTCL = ventricular tachycardia cycle length; RV = right endocardial apical catheter; Epi = the epicardial mapping and ablation catheter; CS = coronary sinus catheter; p = proximal par of electrodes; V = ventricular electrogram; A = atrial electrogram; HR = heart rate; bpm = beats per minute.

safe to apply RF pulses close to the coronary arteries (25) and that selective coagulation necrosis of canine adventia and media induces extracellular matrix accumulation without neointima formation (26). However, it remains unclear what the long-term effects of RF pulses delivered in the vicinity of epicardial coronary arteries are in human beings. Further investigation is still needed to clarify this specific point. Thus, given the potential hazards of this approach, more investigational testing is needed before transthoracic epicardial catheter ablation is implemented clinically.

Possible complications. The nonsurgical transthoracic procedure seems to be safe. Although no complications were observed in the present study, further comments are necessary in this regard. The presented series of 14 patients with post-MI VT belong to a larger group of 53 patients in whom transthoracic epicardial mapping and/or ablation was carried out between June 1995 to March 1999 (34 patients with Chagas' disease, 4 with dilated cardiomyopathy and 1 pediatric patient). In this larger series, accidental right ventricular perforation occurred in four patients. In three of them, there was a small hemopericardium (50 ml), drained at the electrophysiology laboratory with a pig-tail catheter placed in the pericardial space and connected to a vacuum bottle. Only three patients complained of precordial discomfort; two of these had a pericardial rub. They were successfully treated with anti-inflammatory drugs. Neither phrenic nerve lesion nor clinically significant pulmonary lesion was observed after the procedure. Besides, all patients were submitted to an echocardiogram shortly after the procedure, and that was repeated on the day of hospital discharge and none of the patients had pericardial effusion on the day of the hospital discharge. No early or late complaints suggesting the presence of pericardial or acute coronary artery disease were observed in these patients.

JACC Vol. 35, No. 6, 2000
May 2000:1442–9

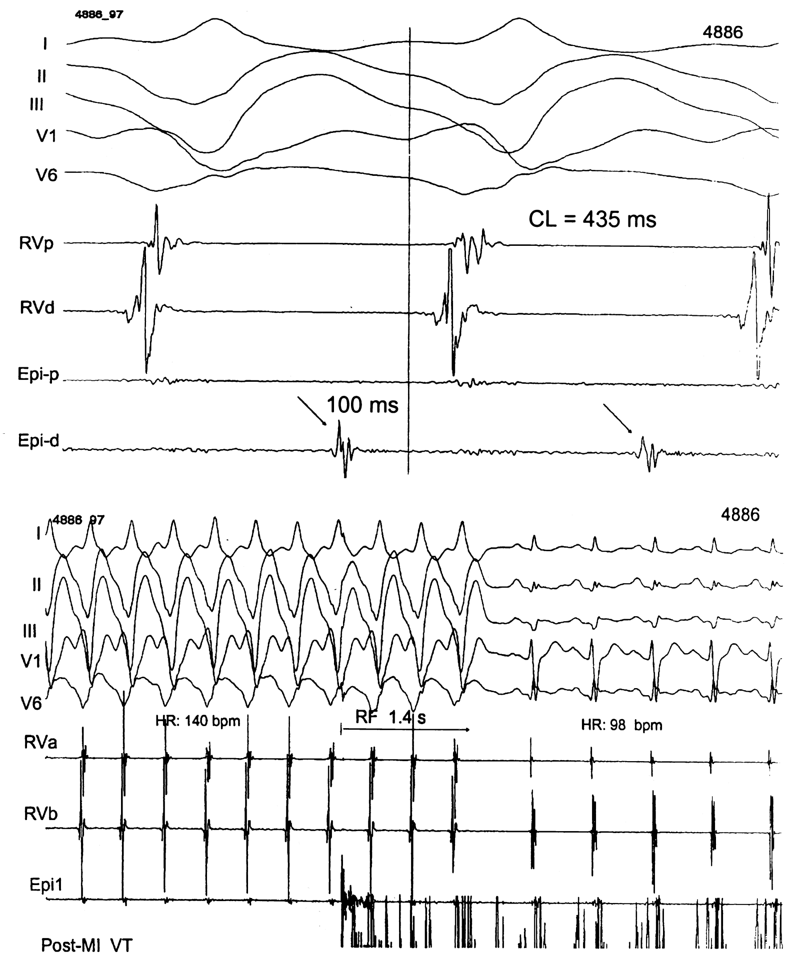

Figure 4. Activation mapping of ventricular tachycardia obtained during a transthoracic epicardial ablation procedure. A midiastolic signal found in the epicardial catheter distal bipole (Epi-d) precedes the onset of the QRS complex by 100 ms **(upper panel)**. Application at this site interrupted VT after 1.4 s which could not be reinduced. Abbreviations as in Figure 3. b and 1 = distal par of electrodes; a = proximal par of electrodes.

Study limitations. Although we have reported a consecutive series of patients with postinferior MI VT, the left ventricular ejection fraction is unexpectedly well preserved in all of them (50 ± 12%) and only one patient had an inferior aneurysm. This observation may suggest that most of the patients in our series did not have transmural infarction. Therefore, the incidence of epicardial circuits may be more prevalent in small or nontransmural inferior infarction. Besides, the incidence of pericardial adhesions, which could limit epicardial mapping, is less important in a small infarction, which might have increased success rate for cannulation of the pericardial sac in these series. Consequently, the results of this technique in larger MIs may not be the same.

In the present series, two patients had undergone successful epicardial ablation after failing to respond to standard endocardial procedures. However, results obtained with these patients do not allow us to know which VTs could be ablated by endocardial RF applications. Some studies suggest that standard endocardial pulses may produce transmural lesions, but this is not the case for most RF endocardial applications (27). However, it is clear that irrigated catheters produce deeper endocardial lesions, but it is not known whether they could be used to ablate epicardial circuits. Further investigation is needed to establish the limits and usefulness of transthoracic epicardial ablation to treat patients with recurrent VT. In this regard, it will be crucial to settle criteria to distinguish an endocardial from an epicardial VT before the electrophysiologic evaluation.

Assuming that epicardial circuits cannot be destroyed by standard endocardial applications, it remains unclear whether a technique using lower RF power ablation (transthoracic epicardial ablation) is safer than other technique using high power RF ablation (irrigated tip endocardial ablation). This question could be addressed only by comparing safety and efficacy of endocardial ablation with irrigated catheter and transthoracic epicardial ablation in patients with epicardial VTs. Besides, the utility of these procedures is clearly related to prevalence of epicardial circuits in a given patient. Patients without an epicardial circuit cannot benefit from this approach as demonstrated by a mean success rate of 37.14% (95% confidence interval from 11.83% to 62.45%).

CONCLUSIONS

Possible postinfarction pericardial adherence does not preclude transthoracic epicardial mapping, which can be utilized to guide epicardial RF catheter ablation to control ventricular tachycardia related to an epicardial circuit occurring late after an inferior wall myocardial infarct. However, further studies on safety of transthoracic epicardial catheter ablation are needed before it is implemented clinically.

Reprint requests and correspondence: Dr. Eduardo Sosa, Unidade Clinica de Arritmia, Instituto do Coração—HC FMUSP, Av. Enéas de Carvalho Aguiar, 44, CEP = 05403-000/São Paulo/SP/Brazil. E-mail: sosa@incor.usp.br.

REFERENCES

1. Sosa E, Scanavacca M, d'Avila A, Pilleggi F. A new technique to perform epicardial mapping in the electrophysiology laboratory. J Cardiovasc Electrophysiol 1996;7:531–6.
2. Sosa E, Scanavacca M, d'Avila A, et al. Endocardial and epicardial ablation guided by nonsurgical transthoracic epicardial mapping to treat recurrent ventricular tachycardia. J Cardiovasc Electrophysiol 1998;9:229–39.
3. Sosa E, Scanavacca M, d'Avila A, et al. RF catheter ablation of ventricular tachycardia guided by nonsurgical epicardial mapping in chronic chagasic heart disease. Pacing Clin Electrophysiol 1999;22:128–30.
4. Krikorian JG, Hancock EW. Pericardiocentesis. Am J Med 1978;65:808–16.
5. Kaltenbrunner W, Cardinal R, Dubuc M, et al. Epicardial and endocardial mapping of ventricular tachycardia in patients with myocardial infarction: is the origin of the tachycardia always subendocardially localized? Circulation 1991;84:1058–71.
6. Blanchard SM, Walcott GP, Wharton JM, Ideker RE. Why is catheter ablation less successful than surgery for treating ventricular tachycardia that results from coronary artery disease? Pacing Clin Electrophysiol 1994;17:2315–24.
7. Svenson RH, Littmann L, Gallagher JJ, et al. Termination sequence of ventricular tachycardia with epicardial laser photocoagulation: a clinical comparison with patients undergoing successful endocardial photocoagulation alone. J Am Coll Cardiol 1990;15:163–70.
8. Littman L, Svenson RH, Gallagher JJ, et al. Functional role of ventricular tachycardia: observations derived from computerized epicardial activation mapping, entrainment, and epicardial laser photoablation. Circulation 1991;83:1577–91.
9. Svenson RH, Littmann L, Colavita PG, et al. Laser photoablation of ventricular tachycardia: correlation of diastolic activation times and photoablation effects on cycle length and termination: observations supporting a macroreentrant mechanism. J Am Coll Cardiol 1992;19:607–13.
10. Hadjis TA, Stevenson WG, Harada T, et al. Preferential locations for critical reentry circuit sites causing ventricular tachycardia after inferior wall myocardial infarction. J Cardiovasc Electrophysiol 1997;8:363–70.
11. Pfeiffer D, Moosdorf R, Svenson RH. YAG laser photocoagulation of ventricular tachycardia without ventriculotomy in patients after myocardial infarction. Circulation 1996;94:3221–5.
12. Trappe HJ, Klein H, Auricchio A, et al. Catheter ablation of ventricular tachycardia: role of the underlying etiology and the site of energy delivery. Pacing Clin Electrophysiol 1992;15:411–24.
13. Kottkamp H, Hindricks G, Chen X, Brunn J, et al. RF catheter ablation of sustained ventricular tachycardia in idiopathic dilated cardiomyopathy. Circulation 1995;92:1159–68.
14. Kim YH, Sosa-Suarez G, Trouton TG, et al. Treatment of ventricular tachycardia by transcatheter RF ablation in patients with ischemic heart disease. Circulation 1994;89:1094–1102.
15. Gonska BD, Cao K, Schaumann A, et al. Catheter ablation of ventricular tachycardia in 136 patients with coronary artery disease: results and long-term follow-up. J Am Coll Cardiol 1994;24:1506–14.
16. Stevenson WG, Khan H, Sager P, et al. Identification of reentry circuit sites during catheter mapping and RF ablation of VT late after myocardial infarction. Circulation 1993;88:1647–70.
17. Strickberger SA. Pericardial space exploration for ventricular tachycardia mapping: should the countdown begin? J Cardiovasc Electrophysiol 1996;7:537–8.
18. Sosa E, Scanavacca M, d'Avila A, et al. Long-term results of visually-guided left ventricular reconstruction as single therapy to treat ventricular tachycardia associated with postinfarction anteroseptal aneurysm. J Cardiovasc Electrophysiol 1998;9:1133–43.
19. Petersen HH, Chen X, Pietersen A, et al. Temperature-controlled irrigated tip RF catheter ablation: comparison of in vivo and in vitro lesion dimensions for standard catheter and irrigated tip catheter with minimal infusion rate. J Cardiovasc Electrophysiol 1998;9:409–14.
20. Nakagawa H, Wittkampf FH, Yamanashi WS, et al. Inverse relationship between electrode size and lesion size during RF ablation with active electrode cooling. Circulation 1998;98:458–65.
21. Arruda M, Chandrasekaran K, Reynolds D, et al. Idiopathic epicardial outflow tract ventricular tachycardia: implications for RF catheter ablation (abstr). Pacing Clin Electrophysiol 1996;19:611.
22. Saxon LA, Sherman CT, Stevenson WG, et al. Ventricular tachycardia after infarction: sources of coronary blood flow to the infarct zone. Am Heart J 1992;124:84–6.
23. Brugada P, de Swart H, Smeets JL, et al. Transcoronary chemical ablation of ventricular tachycardia. Circulation 1989;79:475–82.
24. Kay GN, Epstein AE, Bubien RS, et al. Intracoronary ethanol ablation for the treatment of recurrent sustained ventricular tachycardia. J Am Coll Cardiol 1992;19:159–68.
25. Arruda M, Otomo K, Pitha J, et al. Epicardial left ventricular recordings and RF catheter ablation from the coronary veins: a potential adjunct approach for mapping and ablation of ventricular tachycardia. Pacing Clin Electrophysiol 1997;20:1075.
26. Hehrlein C, Thompson M, Chuang CH, et al. Selective coagulation necrosis of canine adventitia and media induces extracellular matrix accumulation without neointima formation. Atherosclerosis 1995;113:109–15.
27. Bartlett TG, Mitchell R, Friedman PL, Stevenson WG. Histologic evolution of RF lesions in an old human myocardial infarct causing ventricular tachycardia. J Cardiovasc Electrophysiol 1995;6:625–9.