Epicardial Ablation for Ventricular Tachycardia in Chronic Chagas Heart Disease

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The Chagas Heart Disease, described for the first time in 1909 by Carlos Chagas, is caused by *Trypanosoma cruzi*, a flagellated protozoa transmitted to humans, mostly by the feces of Triatoma infestans in endemic areas. Estimates are that fifteen million people are infected in Latin America, leading to 45,000 deaths per year, 90% owing to heart disease.

The cardiac involvement is the most prevalent and severe manifestation of Chagas Heart Disease. After the infection, most individuals remain without manifestation of the disease along their lives, but at least 30% develop heart rhythm disorders, severe symptoms of heart failure and thromboembolic events, typically after 10-30 years after infection. Chronic myocarditis predisposes to cardiac dilation and to the formation of ventricular aneurysms. Thrombi are more prevalent in apical ventricular aneurysm, which is typical of the disease, and the cause for thromboembolic events in the systemic and pulmonary circulation. Segmental myocardial fibrosis is the anatomical substrate for ventricular arrhythmias and atrio-ventricular and intraventricular conduction abnormalities. Sudden death occurs in 55-65% of the patients, at times in the absence of prior cardiac symptoms. Death as a result of heart failure happens in 25-30%, and cerebral or pulmonary embolism, in 10-15%[1-3].

Ventricular extrasystoles are rather common in patients with Chagas Heart Disease, and its prevalence and complexity are associated with the extent of myocardial injury, particularly with left ventricular dysfunction and dilation[4]. Non-sustained ventricular tachycardia (NSVT) has been recognized as an independent risk factor for death and included in score for risk stratification[5]. Sustained ventricular tachycardia (SVT) is considered the main cause of sudden death, and may happen in different stages of the disease and even in patients without important ventricular dysfunction[6].

Amiodarona is the most commonly used antiarrhythmic drug in Brazil to treat ventricular arrhythmias in patients with Chagas Heart Disease. Cohort studies involving patients with sustained Chagas Heart Disease and VT revealed 5-11.9% of annual mortality, with sudden death representing 61-78% of the cases, mostly with important ventricular systolic dysfunction[7-10]. On account of this, the implantable cardioverter defibrillator (ICD) has been recommended to patients with sustained VT and ventricular dysfunction[11-14]. However, patients with Chagas Heart Disease seem to receive more ICD shocks compared to patients with coronary heart disease. A relevant finding was the annual mortality of 16.6% in a cohort of 90 patients with Chagas Heart Disease subject to ICD implantation due to sustained VT. Although the patients presented low rate of sudden death, they had a significant rate of total mortality. Patients with more than four shocks over a period of 30 days presented higher mortality compared to patients with no shock or a smaller number[11]. Since these patients mostly died from heart failure, one can speculate that excessive shocks applied by ICD may depress the ventricular function and increase non-sudden mortality. This data corroborate the indication of catheter ablation as the logical strategy to reduce the recurrence of sustained VT and prevent ICD shocks[15].

Catheter Ablation of Sustained VT in Chagas Heart Disease

The main mechanism of SVT of chronic Chagas Heart Disease is the reentry located in a left ventricle inferolateral and baseline scar in more than 70% of the cases[16,17]. The reentrant circuit of SVT may involve subendocardial, intramyocardial and subepicardial fibers. In some patients, the reentrant circuits have their genesis in places with very thin walls and, on account of that, conventional pulses of RF, emitted from the endocardium, may cause transmural injury and reach all the structures potentially involved in the circuit. Nevertheless, in others, the segmental injury is intramural and the circuit is predominantly kept by subepicardial fibers. Insofar as the contralateral subendocardial tissue is very thick, it may prevent RF applications from reaching the causative intramyocardial and subepicardial fibers and be the reason for an unsuccessful procedure. This was the initial hypothesis for resorting to the transthoracic subxiphoid percutaneous approach to explore the pericardial space and identify patients with possible subepicardial circuits[15].

Epicardial Ablation of Sustained VT in Chagas Heart Disease

The transthoracic epicardial approach has been used since 1995 for mapping and ablation of sustained VT in patients with Chagas Heart Disease[18] and subsequently also applied in patients with other heart diseases[19]. The experience acquired over time confirmed the initial findings[20-23], and consensus documents issued by different medical societies report

Keywords

Chagas Cardiomyopathy; Tachycardia, ventricular; Catheter ablation.

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that it is necessary in at least 20% of the patients subject of SVT ablation in tertiary centers, mainly in patients with non-ischemic heart diseases[24,25]. Recent review underscores the current importance of the subxiphoid percutaneous approach in several intrapericardial procedures[26].

The technique of access to the periocardial space has been kept virtually unchanged over time, with nearly no technological advance for its performance[27]. The Tuohy needle is the main tool to reach the pericardial space (Figure 1). It is curved at one end, which makes it easier to penetrate pericardial membranes (Figure 2). A risk of 10% of pericardial bleeding is forecasted when applying this technique, usually a minor and transient one. The risk of severe bleeding demanding surgical repair is around 1-2%. In most patients subject to this approach, the epicardial mapping widens the exploration area. Pericardial adhesions are not common after prior ablations either.

The electrophysiological signs obtained during the epicardial mapping show patterns similar to those obtained with the endocardial mapping, whether in patients with Chagas Heart Disease or with other heart diseases. Delayed potentials are most predominantly found in the target area during mapping in sinus rhythm and pre-systolic activity. Meso-diastolic and continuous activities are also frequent in the original place of VT. The critical isthmus of the reentrant circuit in the subepicardial tissue may be confirmed by entrainment maneuvers or interruption of VT during the application of RF in these places, as can be observed by the endocardial approach. The prevalence of epicardial VT in 257 consecutive patients was higher in patients with Chagas Heart Disease (37%) compared to patients after myocardial infarction (28%) and patients with idiopathic dilated cardiomyopathy (24%)[28].

Three anatomical aspects of the epicardial surface may hamper the efficiency of epicardial ablation: the presence of epicardial coronary artery, a thick layer of fat and the risk of causing injury in neighboring tissues, such as the phrenic nerve[29]. For all these reasons and more, a substantial number of patients still present clinical recurrences after endocardial and epicardial ablation, whether due to limitations found during the procedure or to the evolution of the disease. The recurrence rate seems to decrease after the introduction of electroanatomic mapping to establish the scar extension and limits (Figures 3 and 4), and irrigated-tip catheter ablations (deeper injuries, even in the presence of fat) may bring on wider substrate injuries[30]. Alternatively, wider epicardial injuries may increase the risk of damaging the coronary arteries and extracardiac structures[31,32].

Prospective randomized study is required to evaluate the role and risks of different strategies during catheter ablations in patients with Chagas Heart Disease[33-38]. However, there is a special group of patients to whom catheter ablation presents arguable benefits: patients with ICD receiving multiple shocks, regardless of the adjuvant therapy with antiarrhythmic drugs. In these cases, the clinical outcomes are very clear in short and medium-term evaluations[39]. It is worth noting that the Chagas Heart Disease has a progressive nature, and it is not uncommon for patients with good left ventricular function to have favorable SVT ablations, but return 5-10 years afterwards presenting left ventricular dysfunction and recurrence of new SVT.

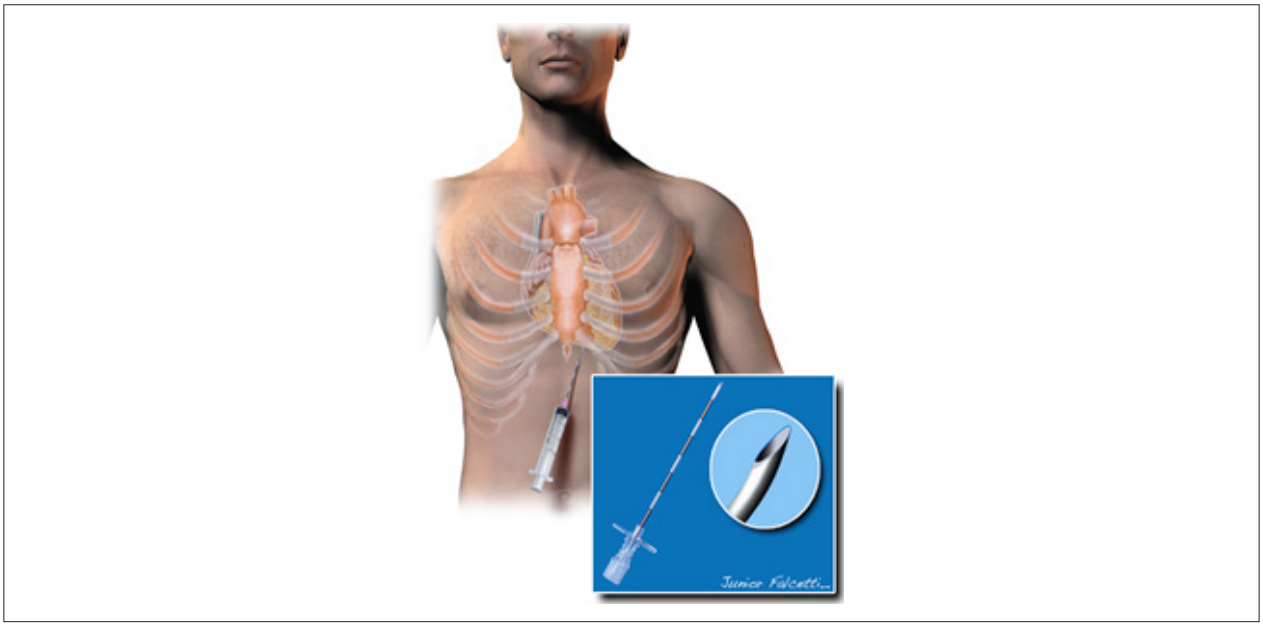

Figure 1 – *Access to the normal pericardial space for epicardial mapping and ablation by subxiphoid puncture. In detail, the Tuhoy needle drawn for epidural anesthesia and used in this procedure to mitigate the risk of cardiac perforation.*

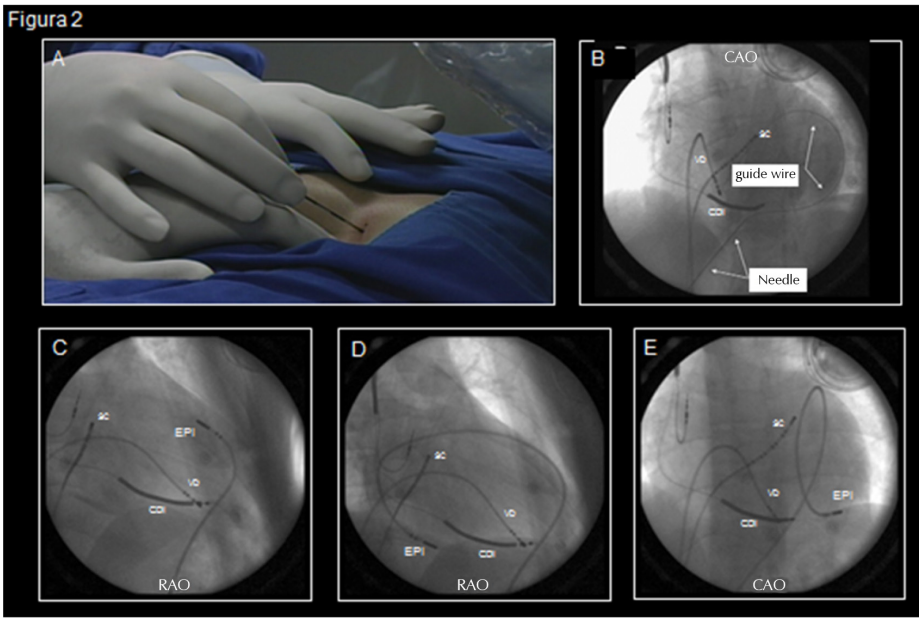

Figure 2 – *Technique of access to the normal pericardial space. A: Subxiphoid puncture with Tuhoy needle. The epigastric compression makes it easier to introduce the needle in the pericardial space, lowering the risk of intra-abdominal organ perforation, mainly the liver. B: Heart fluoroscopy in left anterior oblique projection (LAO) showing the right positioning of the guide wire in the pericardial space. C, D, E: Fluoroscopic aspects in right anterior oblique (RAO) and LAO of the positioning of the exploratory catheter on the heart epicardial surface.*

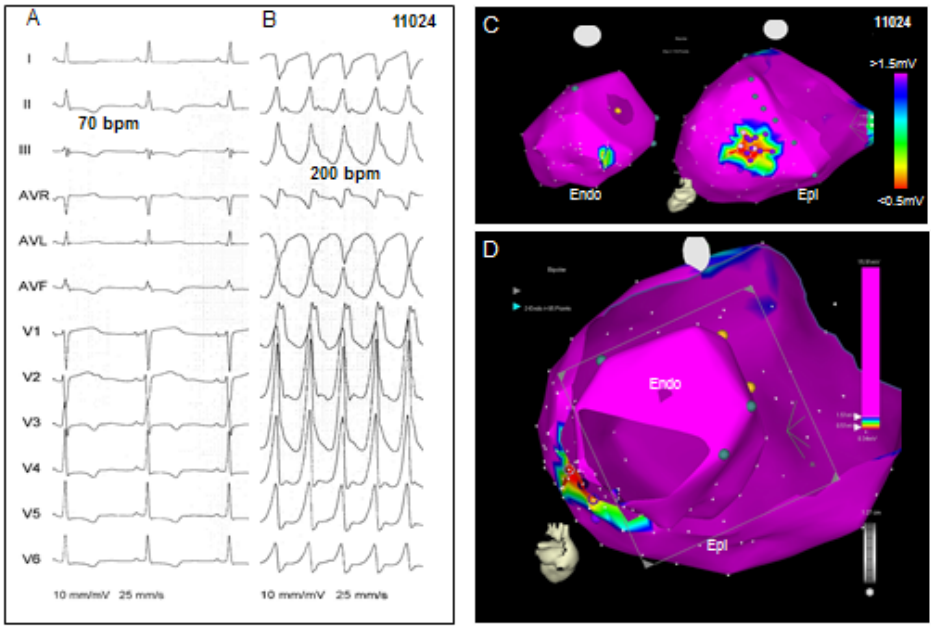

Figure 3 – *Electroanatomic mapping in patients with Sustained Ventricular Tachycardia (SVT) secondary to Chronic Chagas' Cardiomyopathy (CCC). A: ECG in sinus rhythm. Note that there are no disturbances of the atrioventricular and intraventricular conduction or electrically inactive areas, but only change of repolarization of the left ventricle (LV) inferior and lateral walls, secondary to the segmental scar located in the same regions (panels C and D). B: ECG of SVT with electrocardiographic pattern suggesting origin in the LV baseline region (positive QRS of V1 to V6), with onset of ventricular activation on the lateral wall (negative QRS in DI and aVL and positive in D2, D3 and aVF). C: 3D electroanatomic mapping of the LV endocardial and epicardial surfaces in sinus rhythm with the Carto system. The colors represent the amplitude of the ventricular electrograms in the investigated regions. Note that the low-voltage area, suggesting the presence of scar, is predominantly epicardial. D: Integration of the endocardium and epicardium maps showing the LV inferior lateral and baseline segmental injury, anatomical substrate for SVT in this patient.*

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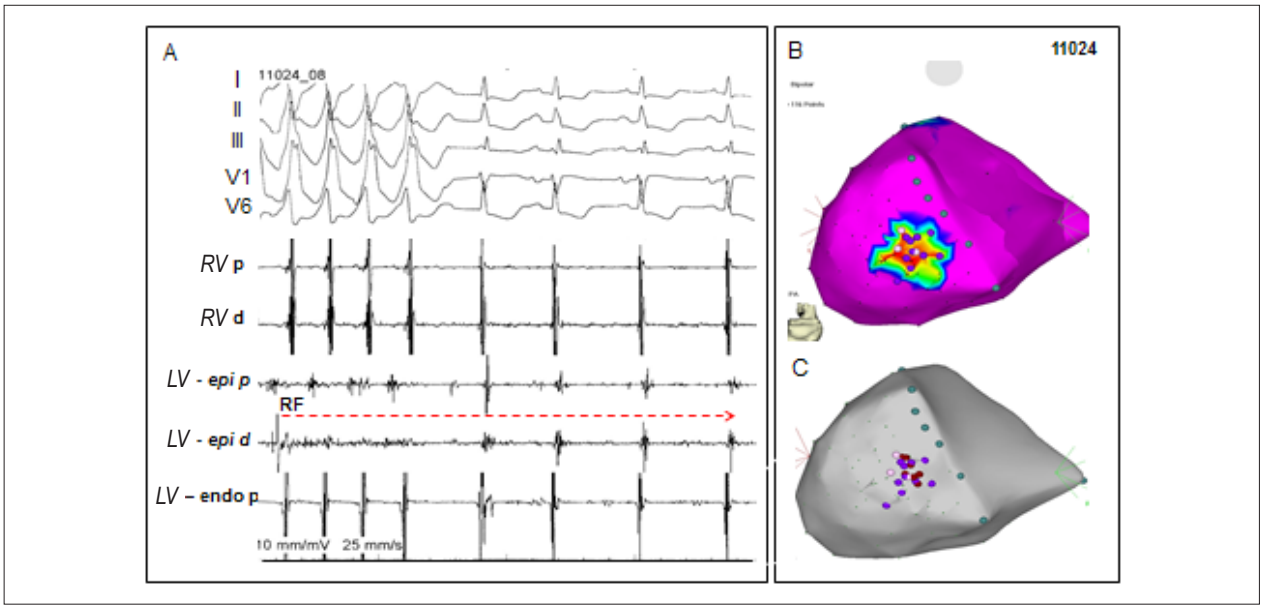

Figure 4 – *SVT ablation in the same patient of Figure 3. A: Electrophysiological scan documenting the moment of application of RF pulse by epicardial catheter with interrupts SVT and restores the sinus rhythm. I, II, III, V1 and V6: ECG leads synchronized with intracavitary electrograms. RV p and RV d: bipolar electrograms obtained by proximal and distal electrode placements of the right ventricle, respectively. LV epi p and LV epi d: bipolar electrograms obtained, respectively, by proximal and distal electrodes of the epicardial catheter positioned on the LV inferior and lateral scar. VE endo p: bipolar sign of the catheter introduced in the LV. RF: moment of application of radio frequency power, which interrupts SVT. The application is kept by 60 seconds. Speed of registration: 25 mm/s. B: Posterior view of the voltage electroanatomic map showing the limits of LV inferior, lateral and baseline scar. The purple and pink spots indicate places with fractionated and delayed electrograms during the sinus rhythm. The green spots indicate the transition between the left ventricle and left atrium. C: Same electroanatomic mapping view showing the RF applications (red spots) in the SVT origin site.*

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