

Catheter ablation in the interventional era of arrhythmogenic syndromes

Ablación por catéter en la era intervencionista de los síndromes arritmogénicos

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ARTICLE INFORMATION

Acronyms

RFA: radiofrequency ablation

RF: radiofrequency

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The worst times for antiarrhythmic drugs came in the late '80s, as a result of their own problems and the emergence of radiofrequency (curative treatment) and the implantable cardioverter-defibrillator (life-saving treatment). There was a revolution in the treatment of cardiac arrhythmias and a development of nonpharmacologic options, which were remarkably successful¹⁻⁴.

There was, and still is, a rapid worldwide growth of laboratories practicing interventionism in Arrhythmology: radiofrequency ablation (RFA) and implantable cardioverter-defibrillator. This is reflected in the numerous publications on these topics¹⁻⁶.

Callans said: *"Many of us began practicing electrophysiology before its interventional era, when this*

field was intensely intellectual but less successful at protecting patients from future harm". So it was in Cuba, since the creation of the clinical electrophysiology laboratory at the Institute of Cardiology and Cardiovascular Surgery, in December de 1984, until the first RFA was performed in January 1996.

The bases for interventionism in Arrhythmology, until reaching the RFA, were experimental electrophysiology, electrocardiography and clinical electrophysiology. There was a move from classical arrhythmology to the interventional one, and then diagnosis became therapeutics. Programmed electrical stimulation of the heart, surgery and fulguration with direct current, were the foundation for the development of the new procedure. All this also happened in our country, changing dramatically the role of the clinical laboratory, from a diagnostic and artistic role to a therapeutic function. Clinical electrophysiology emerged in 1967, and therapeutic electrophysiology, in 1987, with the emergence of radiofrequency (RF). There is an absolute two-way connection between them: the understanding of the arrhythmic substrate

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and its ablation; there cannot be a divorce between clinical electrophysiology and the electrotechnology, they enrich each other^{1-4,7}.

Rapid changes occurred in this emerging subspecialty, turning it into a major subspecialty that has faced, since then, great challenges.

Zipes said that: "No other cardiovascular subspecialty has undergone a more radical transformation than the study and treatment of cardiac arrhythmias". And that "...thanks to advances in radiofrequency catheter ablation it is the only subspecialty that can claim the ability to actually cure a patient of disease"². This illustrates the importance of the RF.

The purpose of the ablation is to cure the arrhythmia by destroying small areas of myocardial or conduction tissue which are critical for the initiation and maintenance of cardiac arrhythmia. This procedure results in well-demarcated, progressive lesions, and intentionally destroys the arrhythmogenic tissue in the myocardium, the atrioventricular connections, or in certain parts of the specialized conduction system. It aims to cure or control arrhythmias, and constitutes a safe and highly effective therapeutic option that requires no anesthesia, creates no barotrauma and causes a highly localized tissue damage^{1-4,8}.

The RF causes heat energy dissipation through the near myocardium from the end of the catheter, and the hyperthermia produced suppresses the arrhythmia and creates areas of cell inexcitability and blocked conduction. Its precise and directed nature, and detailed mapping, combine structural and functional aspects of the arrhythmogenesis, helping to understand these mechanisms. It seeks to create a local lesion with discrete edges, large enough to include the target but small enough to minimize collateral damage. The RFA is based on that: precise, small (4-7 mm in diameter and 5 mm deep) and selective lesions, aimed to a target and therefore requiring a detailed mapping. Its effects are superior compared to the more diffuse results of surgery (which it virtually eliminated) and the nonspecific effects of the anti-arrhythmic drugs. Research and education were not eclipsed, but were rather enriched by RF^{1-4,8}.

For any arrhythmia, there is a critical anatomic region that generates the impulse or allows its propagation, and that is required to sustain it clinically. If that substrate is irreversibly altered or destroyed, the arrhythmia will not occur, either spontaneously or caused.

The RF has allowed, among other things: a better understanding of arrhythmogenic mechanisms, with the progressive changes that occur when destroying a substrate; the development of new classifications to locate accessory pathways and the recognition of multiple pathways, dormant, arborized, neighboring, oblique, decremental, inaccessible pathways, those with anterograde and retrograde selectiveness, and those with poor function of the normal conduction system; as well as the presence of several fast and slow pathways in atrioventricular nodal reentrant tachycardia⁹⁻¹¹.

Growth was very fast worldwide, and laboratories conducting the most complex ablations (atrial fibrillation and others not yet performed in Cuba), developed rapidly. Publications also increased. Today, almost all arrhythmias are treated by RF: atrioventricular nodal reentrant tachycardia, accessory pathways, atrial tachycardia and flutter, ventricular tachycardia (in subjects with or without structural heart disease), extrasystoles, and atrial fibrillation.

The RFA can intervene on the substrate (slow pathway of atrioventricular nodal reentrant tachycardia, accessory pathways, among others), on the trigger (ventricular extrasystole in ventricular fibrillation), on the substrate and the modulator (atrial fibrillation through accessory pathways), and on the element that initiates the arrhythmia or on the element that sustains it, that is not always the same one (pulmonary vein extrasystoles, reentry, Purkinje foci).

Innovations have been made in catheters (irrigated, deflectable), the energy (ultrasound, laser, cryoablation), fluoroscopic anatomy and refined mappings (fluoroscopic or not)^{1-4,7,12,13}.

Sometimes RF combines with other interventional procedures: pacemaker and ablation, and implantable cardioverter-defibrillator and ablation.

Major international records were created, such as the Multicentre European Radiofrequency Survey (MERFS), the Heart Rhythm Society and the NASPE (North American Society of Pacing and Electrophysiology)^{5,6}.

Then, the danger that electrophysiology was transformed into electrotechnology and the electrophysiologist into "ablationist" emerged, but it has not happened in general; on the contrary, RFA has allowed to understand many aspects of the pathophysiological mechanisms of arrhythmias and their substrates. In the past, arrhythmias were diagnosed and treated;

today, arrhythmogenic substrates are diagnosed and treated.

There are guidelines to direct the practice, but it is necessary to consider the benefits and hazards, the clinical judgment for the final decision, and remember that such varied arrhythmogenic mechanisms must have also variable treatment options.

Initially, as with all new procedures, there was a lot of fantasy, euphoria and excitement; then, there was a tendency to equilibrium and balance.

Increasingly complex ablations were performed, requiring long execution periods. Sometimes, it is difficult to deter patients and physicians from using RFA, due to the excellent results of the procedure and the information available in Internet. However, there are real risks to be considered. Sometimes RFA is not a first-line option, but it is not always the last choice. It is necessary to take into account what type of ablation is going to be used, in whom, and with what diseases it is associated. The big lesson is to optimize the selection of patients. As Klein says: "We must ask ourselves not can we apply a technology but should we in a given individual"¹⁴⁻¹⁶.

If ablation was completely free of risk, no one would discuss its indications. Some of the problems it may cause are: atrioventricular blocks of varying degrees, stroke, coronary artery dissection, pulmonary embolism, cardiac tamponade, valvular damage, myocardial infarction, coronary spasm, pneumothorax, perforation of the coronary sinus, femoral artery laceration, pericardial effusion, hematoma, hypotension, pleural or pericardial effusion, vasovagal reaction, fever, respiratory depression, incessant arrhythmias, increased dispersion, repolarization abnormalities, proarrhythmia, and death. They are rare but they exist. Moreover, there are ablations that are not successful, and others in which the circuits recover their arrhythmogenic capacity^{17,18}.

Josephson says that the approach "Learning before you burn" should be tried first than "Learning while burning". And, with regard to the ablation of atrial fibrillation, he believes there is loss of critical thinking and that sometimes there is a total dependence on technology without understanding its limitations. What is published is accepted, forgetting the pros and cons and the risk-cost-ineffectiveness of therapies. These are times of use and abuse of RFA in atrial fibrillation, without remembering that positive results are very much publicized, while complications and

failures receive less coverage. Arrhythmia mechanisms differ, and not all atrial fibrillations are the same nor respond to similar procedures. There is an attempt to find a fast and universal cure, although this is not always possible. It must be known what is going to be ablated, and how much, in the various types of atrial fibrillation. It is necessary to standardize the monitoring and the statistical studies, because good results are obtained with varied methods. Furthermore, it must be remembered that this arrhythmia is silent in 75% of patients, that the lesions caused by RF may be excessive and that in 20-40% of them it recur in the form of atrial tachyarrhythmias. Sometimes, the earliest sites of activation during the mapping are misunderstood, additional lines can generate proarrhythmia, and 30-50% of the cases must undergo a second procedure. Not to mention some possible complications such as stroke, paralysis of the phrenic nerve, coronary occlusion, perforation, atrio-oesophageal fistula, and death. Furthermore, these are progressive diseases. Some interpretations may be erratic in the RFA of fibrillation: fractionated electrograms that may not be required to maintain the arrhythmia; asynchronous conduction and not by reentry; and overlapping of the activation wavefronts in a three dimensional structure. It is necessary to know how the arrhythmia is started, how it is perpetuated, if it is a persistent, chronic or permanent type and if ablación is indicated^{13,14}.

As for inherited arrhythmogenic syndromes, ablation is not useful in the long QT syndrome, and there are insufficient data on the short QT. It is performed in some associated arrhythmias and in ventricular fibrillation triggers in Brugada syndrome. It may decrease the frequency of events in arrhythmogenic right ventricular dysplasia that continues to occur because it is a progressive disease. It is used in the destruction of an accessory pathway in familial hypertrophic cardiomyopathy; and it is not useful in catecholaminergic polymorphic ventricular tachycardia¹⁹. Better results will have to be seen in familial atrial fibrillation.

There are unresolved and controversial issues, for example, whether ablation is performed in asymptomatic patients with accessory pathways or not, and its real role in atrial fibrillation²⁰⁻²³.

Epilogue: RFA has been of much help and much can be expected of it. There has been a transition from diagnostic to therapeutic electrophysiology. You can

learn while “burning” but it is better to learn before you “burn”. Electrophysiology and electrotechnology must go hand in hand. Like every procedure, it has limitations; therefore, it is necessary to know what to expect from it and what not, and in which patients it must be applied and in which it must not.

REFERENCES

1. Josephson ME. Catheter and surgical ablation in the therapy of arrhythmias. En: Clinical cardiac electrophysiology: techniques and interpretations. 4th ed. Philadelphia: Lippincott Williams & Wilkins; 2008. p. 746-887.
2. Issa ZF, Miller JM, Zipes DP. Ablation energy sources. En: Clinical arrhythmology and electrophysiology. A companion to Braunwald's Heart Disease. Philadelphia: Saunders Elsevier; 2009. p. 100-17.
3. Atiga WL, Calkins H. Catheter ablation of supraventricular tachycardias. En: Ganz LI. Management of cardiac arrhythmias. New Jersey: Humana Press; 2002. p. 51-73.
4. Tracy CM, Akhtar M, DiMarco JP, Packer DL, Weitz HH, Creager MA, *et al.* American College of Cardiology/American Heart Association 2006 update of the clinical competence statement on invasive electrophysiology studies, catheter ablation, and cardioversion: a report of the American College of Cardiology/American Heart Association/American College of Physicians Task Force on Clinical Competence and Training: developed in collaboration with the Heart Rhythm Society. *Circulation*. 2006; 114(15):1654-68.
5. Scheinman MM. Patterns of catheter ablation practice in the United States: results of the 1992 NASPE survey. *North American Society of Pacing and Electrophysiology. Pacing Clin Electrophysiol*. 1994;17(5 pt 1):873-5.
6. Hindricks G. The Multicentre European Radiofrequency Survey (MERFS): complications of radiofrequency catheter ablation of arrhythmias. The Multicentre European Radiofrequency Survey (MERFS) investigators of the Working Group on Arrhythmias of the European Society of Cardiology. *Eur Heart J*. 1993;14(12):1644-53.
7. Wellens HJ. Forty years of invasive clinical electrophysiology 1967-2007. *Circ Arrhythmia Electrophysiol*. 2008;1(1):49-53.
8. Haines DE. The biophysics and pathophysiology of lesion formation during radiofrequency catheter ablation. En: Zipes DP, Jalife J, editors. *Cardiac electrophysiology: from cell to bedside*. 4th ed, Philadelphia: WB Saunders Co; 2005. p. 1018-27.
9. Callans DJ, Schwartzman D, Gottlieb CD, Marchlinski FE. Insights into the electrophysiology of accessory pathway-mediated arrhythmias provided by the catheter ablation experience: "learning while burning, part III". *J Cardiovasc Electrophysiol*. 1996;7(9):877-904.
10. Callans DJ, Schwartzman D, Gottlieb CD, Marchlinski FE. Insights into the electrophysiology of atrial arrhythmias gained by the catheter ablation experience. "Learning while burning, part II". *J Cardiovasc Electrophysiol*. 1995;6(3):229-43.
11. Callans DJ, Schwartzman D, Gottlieb CD, Marchlinski FE. Insights into the electrophysiology of ventricular tachycardia gained by the catheter ablation experience: "Learning while burning". *J Cardiovasc Electrophysiol*. 1994;5(10):877-94.
12. Erdogan A, Grumbrecht S, Neumann T, Neuzner J, Pitschner HF. Microwave, irrigated, pulsed, or conventional radiofrequency energy source: which energy source for which catheter ablation? *Pacing Clin Electrophysiol*. 2003;26(1 Pt 2):504-6.
13. Erdogan A, Walleck E, Rueckleben S, Neumann T, Tillmanns HH, Waldecker B, *et al.* Companion between pulsed and continuous radiofrequency delivery. *J Interv Card Electrophysiol*. 2007;20(1): 21-4.
14. Josephson ME. Electrophysiology at a crossroads. *Heart Rhythm*. 2007;4(5):658-61.
15. Klein GJ. Electrophysiology at crossroads: a time of great opportunity. *Heart Rhythm*. 2007;4(5):662-4.
16. Modi S, Skanes AC. Complex problems require complex solutions... but may result in other complex problems. *Heart Rhythm*. 2011;8(11):1667-8.
17. Calkins H, Yong P, Miller JM, Olshansky B, Carlson M, Saul JP, *et al.* Catheter ablation of accessory pathways, atrioventricular nodal reentrant tachycardia, and the atrioventricular junction: final results of a prospective, multicenter clinical trial. The Atakr Multicenter Investigators Group. *Circulation*. 1999;99(2):262-70.
18. Bohnen M, Stevenson WG, Tedrow UB, Michaud GF, John RM, Epstein LM, *et al.* Incidence and predictors of major complications from contemporary catheter ablation to treat cardiac arrhythmias. *Heart Rhythm*. 2011;8(11):1661-6.
19. Stephenson EA, Berul CI. Electrophysiological in-

- terventions for inherited arrhythmia syndromes. *Circulation*. 2007;116(9):1062-80.
20. Freedberg NA. Learning while burning revisited. *J Cardiovasc Electrophysiol*. 2008;19(1):7-9.
21. Cohen MI, Triedman JK, Cannon BC, Davis AM, Drago F, Janousek J, *et al*. PACES/HRS expert consensus statement on the management of the asymptomatic young patient with a Wolff-Parkinson-White (WPW, ventricular preexcitation) electrocardiographic pattern: developed in partnership between the Pediatric and Congenital Electrophysiology Society (PACES) and the Heart Rhythm Society (HRS). Endorsed by the governing bodies of PACES, HRS, the American College of Cardiology Foundation (ACCF), the American Heart Association (AHA), the American Academy of Pediatrics (AAP), and the Canadian Heart Rhythm Society (CHRS). *Heart Rhythm*. 2012;9(6):1006-24.
22. Obeyesekere MN, Leong-Sit P, Massel D, Manlucu J, Modi S, Krahn A, *et al*. Risk of arrhythmia and sudden death in patients with asymptomatic pre-excitation: a meta-analysis. *Circulation* 2012; 125(19):2308-15.
23. Raviele A, Natale A, Calkins H, Camm JA, Cappato R, Ann Chen S, *et al*. Venice chart international consensus document on atrial fibrillation ablation: 2011 update. *J Cardiovasc Electrophysiol*. 2012; 23(8):890-923.