

INTRODUCTION





Atrial fibrillation ablation. Unsolved questions, many possible answers



Josep Brugada^{a,b}

^a Hospital Clínic, Barcelona, Spain ^b Pediatric Hospital Sant joan de Déu, University of Barcelona, Esplugues de Llobregat, Spain

Received 3 June 2017; accepted 16 September 2017 Available online 1 November 2017

Atrial fibrillation (AF) is the most frequent sustained arrhythmia and probably the most complex one. We are far from understanding all mechanisms involved in the occurrence and persistence of AF. Many theories have been developed and each one accounts for a certain number of patients and cases, but definitively none of them is inclusive for all cases of AF seen in the clinical practice. This variability makes the therapeutical approach also very complex. Which arrhythmia represents which mechanism and what is the best option for treatment remains very hypothetical.

Many developments have occurred in the knowledge and approach to this extremely relevant medical problem. If you look 30 years ago, our understanding of the disease was very limited. Today, thousands of investigations have clarified a lot the physiopathology, the epidemiology, the basic mechanisms and the best therapy for each case. However, due to the complex nature of this disease, many aspects remain unsolved and the real answers are still lacking.

In this issue, the contributions of extremely important authors are devoted to understand some basic concepts on AF, but coming from the therapeutical experience. The groups have used different techniques to modify a very specific aspect of the electrical/anatomical substrate and from these results some theories about the physiopathology of this arrhythmia can be obtained.

The fact that many and very different aspects are treated is already a confirmation that this is a complex disease, having a variable origin, several possible triggers and obviously partial solutions.

From the initial concept of multiple wavelet theories by Allessie et al.¹ we moved to the presence of foci mainly located in the pulmonary veins described by Haissaguerre et al.² Know, the theory of rotors, involving specific anatomical areas of the atria introduces the concept that the abnormal electrical activity anchors in specific zones and maintain AF.³ Pappone et al. was the first to suggest that isolation of the pulmonary veins would be the basis for AF elimination.⁴ In fact, this is the main final objective in the majority of centres around the world when an ablation procedure is undertaken. Now, mapping persistent AF is one of the major challenges. Which part is related to foci in the pulmonary veins, and can be eliminated by isolation and which part is far from the pulmonary veins and relates to other substrates and has to be treated accordingly?

Mapping and understanding persistent AF might give some clues to this discussion. What is the role of ganglionic plexi in triggering and maintaining AF? Can we modify the behaviour of AF by ablating the ganglionic plexi? How would this affect the rest of functions of the myocardium? Do we need single

E-mail address: jbrugada@clinic.ub.es

shoot techniques like cryoablation to eliminate the substrate? Are these techniques safer than others? How can we avoid complications? Are we simplifying the message about easiness of the procedure of ablation in AF while we are manipulating the left atrium with all complications related to it?

In the very last years a lot of information has become available about the role of anatomical substrate, basically fibrosis in the left atrium and how this modifies the expected results in AF ablation. Some information suggests that when the left atrium is too ill (significant amount of fibrosis present) the results of ablation are so poor that probably it should not be proposed as a reasonable alternative. Also, the role of the left atrial appendage, not only as a contributor to the prothrombotic risk in AF, but also as a source of origin of foci or rotors that initiate/perpetuate AF has been questioned.

For all these elements, it is clear that AF is a very life problem. We are still very far from having the real answers to all questions. We are still far from having a magic solution to this problem. In fact, probably the only way to tackle the problem is to consider many problems having the same final manifestation that will require many solutions, some of them will have to be very creative and are still to be imagined. Researchers, clinicians, interventionists have a lot to do before AF will be a solved problem, as it is today the case with the vast majority of other supraventricular arrhythmias.

Conflicts of interest

The authors have no conflicts of interest to declare.

References

- Allessie M, Lammers W, Bonke F, et al. Experimental evaluation of Moe's multiple wavelet hypothesis of atrial fibrillation. In: Zaipes D, Jalife J, editors. Cardiac electrophysiology and arrhythmia. Orlando, FL: Grune and Stratton; 1985. p. 265–75.
- Haissaguerre M, Jais P, Shah DC, et al. Spontaneous initiation of atrial fibrillation by ectopic beats originating in the pulmonary veins. N Engl J Med. 1998;339:659–66.
- Narayan SM, Krummen DE, Shivkumar K, et al. Treatment of atrial fibrillation by the ablation of localized sources: the Conventional Ablation for Atrial Fibrillation With or Without Focal Impulse and Rotor Modulation (CONFIRM) trial. J Am Coll Cardiol. 2012;60:628–36.
- Pappone C, Rosanio S, Augello G, et al. Mortality, morbidity, and quality of life after circumferential pulmonary vein ablation for atrial fibrillation: outcomes from a controlled nonrandomized longterm study. J Am Coll Cardiol. 2003;42: 185–97.