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IS IT POSSIBLE TO TERMINATE ATRIAL FIBRILLATION BY PACING?

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Atrial fibrillation (AF) is an atrial tachyarrhythmia (ATA) with uncoordinated electrical activation of the atria and consequent ineffective atrial contractions. AF is characterized by irregular RR intervals (if there is no atrioventricular conduction disturbance), no distinct repetitive P waves and irregular atrial activation [1]. A more detailed definition can be found in 2003 [2]. It states that instead of P waves, frequent oscillations or fibrillation waves varying in shape, size and time are recorded. In besides these very important additions, there is a section on AF-related arrhythmias. It considers atrial flutter as a more organized arrhythmia than AF. It is indicated that the typical F-waves frequency of the atrial flutter is between 250 and 350 bpm. The description of atrial tachycardia (AT) emphasizes the presence of an isoline between P waves, which can follow a frequency of 100 to 300 bpm or more. It has been noted that AF can occur either in isolation or transforming into atrial flutter or AT, most often atrial flutter can degenerate into AF and conversely, AF can initiate atrial flutter.

It must be emphasized that atrial rate (AR) does not allow the differentiation between atrial flutter and AT, especially in the setting of antiarrhythmic drugs (AAD). AT originating from pulmonary vein (and other veins flowing into the heart) can have a very high AR and be resistant to the effects of AAD. On the other hand, the atrial flutter AR may decrease to 200 bpm or less during AAD action. It is also not always possible to detect areas of isoline between atrial activation waves. Furthermore, it is important to consider that the appearance of such isoelectric areas may be caused using electrocardiograph filters or Holter monitors.

In recent years, there has been increased interest in the electrophysiological mechanisms underlying AF. The 60-year prevailing theory of anisothoropne re-entry by G.K.Moe [3] as the main mechanism of AF maintenance has been questioned. A group of researchers repeated experiments on dogs using a model of AF based on vagus stimulation [4]. The experiments were, of course, carried out on a state-of-the-art technological level. S.Lee et al. showed that in addition to anisotropic re-entry, ectopic activity plays an important role in the maintenance of AF. The editorial for this publication analyses the limitations of both the G.K.Moe study, due to imperfect mapping techniques, and the S.Lee et al study (only epicardial mapping, small number of animals) [5]. The authors of the editorial congratulate S.Lee et al. for obtaining such 'provocative' data and write about the need to translate them into everyday clinical practice.

However, despite changing perceptions of AF and its underlying mechanisms, the position that 'true' AF cannot be terminated by pacing is undisputed. A review of the MINERVA study data shows that although only 20% of patients had a history of atrial flutter and 17% of AT, many ATA episodes had a baseline low AR, with a median of 244 bpm [6]. It is emphazised that an antitachycardia pacing is not able to arrest true AF, but only ATA (even if the initial AR is high and the activation waves are irregular) when the rhythm stabilises and/or the AR decreases.

The supplement to the Non-pharmacological Guidelines for the Treatment of Arrhythmias devotes an entire section to the role of implantable devices in preventing and controlling atrial tachyarrhythmias [7]. It has been pointed out that the pacing is capable of arresting AT and atrial flutter, but in some cases contributes to their transformation into AF. It is noted that second generation atrial antitachycardia pacing devices are capable of applying therapy against a background of decreased AR and rhythm 'organisation', i.e. spontaneous transformation of AF into atrial flutter or AT.

In the example of ATA stopping, presented in Lukin's article [8], the intervals between P or F waves vary in a

rather narrow range of 220-280 ms (the intervals of 180 and 300 ms are measured incorrectly), and there are no pronounced changes of atrial complex shape (as far as the endogram can tell). The mean interval between P or F waves before antitachycardia pacing is 244 ms, corresponding to AR of 246 bpm. This allows the ATA to be treated as AT or atrial flutter, but not as AF. Thus, without questioning the role of algorithms to prevent the onset of AF and to control ATA in reducing the burden of AF and the risk of it becoming permanent, we would like to emphasize that statements about the possibility of controlling "true" AF with pacing do not seem quite correct to us.

REFERENCES

1. Kotalczyk A, Lip GY, Calkins H. The 2020 ESC Guidelines on the Diagnosis and Management of Atrial Fibrillation. *Arrhythm Electrophysiol Rev*. 2021;10(2): 65-67. https://doi.org/10.15420/aer.2021.07.

2. Lévy S, Camm AJ, Saksena S, et al. International consensus on nomenclature and classification of atrial fibrillation: A collaborative project of the Working Group on Arrhythmias and the Working Group of Cardiac Pacing of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology. *J Cardiovasc Electrophysiol.* 2003;14(4): 443-5. https://doi.org/10.1046/j.1540-8167.2003.00003.x.

3. Moe GK. On the multiple wavelet hypothesis of atrial fibrillation. *Arch Int Pharmacodyn Ther.* 1962;140: 183.

4. Lee S, Khrestian CM, Sahadevan J, Waldo AL. Reconsidering the multiple wavelet hypothesis of atrial fibrillation. *Heart Rhythm*. 2020;17(11): 1976-1983. https://doi.org/10.1016/j.hrthm.2020.06.017.

5. Ganesan P, Narayan SM. Re-evaluating the multiple wavelet hypothesis for atrial fibrillation. *Heart Rhythm*. 2020;17(12): 2219-2220. https://doi.org/10.1016/j.hrthm.2020.07.009.

6. Padeletti L, Pürerfellner H, Mont L, et al. MINERVA Investigators. New-generation atrial antitachycardia pacing (Reactive ATP) is associated with reduced risk of persistent or permanent atrial fibrillation in patients with bradycardia: Results from the MINERVA randomized multicenter international trial. *Heart Rhythm.* 2015;12(8): 1717-25. https://doi.org/10.1016/j.hrthm.2015.04.015.

7. Nogami A, Kurita T, Kusano K, et al. JCS/JHRS 2021 guideline focused update on non-pharmacotherapy of cardiac arrhythmias. *J Arrhythm.* 2022;38(1): 1-30. https://doi.org/10.1002/joa3.12649.

8. Lukin IB. Algorithms for the prevention and treatment of supraventricular tachycardia in patients with implanted pacemakers: case series. *Journal of Arrhythmology.* 2022;29(4): e9-e14 (In Russ). https://doi.org/10.35336/VA-2022-4-12.