The endless fascination of right typical atrial flutter: can we predict its occurrence?

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Abstract

The chance for the development of right AFL is strictly related to changes in functional conduction properties of the atrial myocardium which are greatly influenced by fibrotic/scar tissue and increased atrial volume. If these circumstances take place, reduction in conduction velocity can favor a macro-reentry circuit with the wavefront that does not meet its refractory tail and perpetuate the arrhythmia. Therefore, the time required to traverse the entire circuit is related to the circuit's functional properties. With pacing from the coronary sinus os the right atrial collision time (RACT) of the two wavefronts traveling the circuit in counterclockwise and clockwise direction is calculated. in this prospective study, a cut-off of 115.5 ms of RACT showed a sensitivity and specificity of 92.7% and 93.0% respectively for diagnosis of AFL and an ROC curve indicated an AUC of 0.96 (95% CI: 0.93-1.0, p<0.01). Based on these premises, RACT could be utilized as new marker for the propensity of developing typical AFL.

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In this issue of the Journal, Rickman et (1) al. take us into a new journey across the landscape of the cavo-tricuspid dependent atrial flutter (AFL) by enriching the old concepts with a novel parameter, i.e. right atrial collision time (RACT), whose role is to predict the occurrence of right common atrial flutter. Cavo-tricuspid dependent AFL has been extensively investigated in the last few decades and it is well also known its association with atrial fibrillation (AF). (2-5). The chance for the development of right AFL is strictly related to changes in functional conduction properties of the atrial myocardium which are greatly influenced by fibrotic/scar tissue and increased atrial volume. If these circumstances take place, reduction in conduction velocity can favor a macro-reentry circuit with the wavefront that does not meet its refractory tail and perpetuate the arrhythmia. Therefore, the time required to traverse the entire circuit is related to the circuit's functional properties. Constant pacing from the coronary sinus os is a well-recognized surrogate for the calculation of the collision time of the two wavefronts traveling the circuit in counterclockwise and clockwise direction.

Rickman et al. (1) have used this modality of pacing for investigating whether the collision time of the two wavefronts on the lateral wall of the right atrium could predict the occurrence of typical atrial flutter. For this purpose, the authors have designed a prospective study including a sample size of 98 patients, 41 having AFL and 57 as controls. The main findings were that patients with AFL were older (64.7±9.7 vs 52.4±16.8 years) and more often male and with significantly longer mean RACT (132.6±17.3 ms vs 99.1±11.6 ms) (p<0.001).

Moreover. a RACT cut-off of 115.5 ms had a sensitivity and specificity of 92.7% and 93.0% respectively for diagnosis of AFL and an ROC curve indicated an AUC of 0.96 (95% CI: 0.93-1.0, p<0.01). Based on these premises, RACT could be utilized as new marker for the propensity of developing typical AFL Needless to remind that patients with AFL also showed longer P wave duration, larger right and left atrium volumes and higher rate of comorbidities. Really and truly, these findings are not unexpected. On the other hand, on multivariate analysis only RACT was independently associated with atrial flutter patients (OR 1.6 (1.1 – 2.4) p = 0.03).

So, is that simple?

P wave duration is often considered an expression of longer atrial conduction time (6,7) and one could raise the question if this "easy-taken" parameter might simply used for the same purpose:*identifying those patients at risk to develop AFL.* P wave duration is an expression of the entire atrial conduction time between the two atria and, thus it is not comparable to RACT which marks the conduction time only within the right atrium.(8). P wave is the phenotypic expression of the more complex atrial conduction process and it is specifically influenced by the local conduction time in the left atrium. Therefore, we may observe longer P wave duration which is not always associated to longer RACT. Actually, it would have been useful to explore in this study population, if patients who exhibited AF over the follow up also had longer P wave at the time of index electrophysiologic procedure.

More unexpected is the finding that larger right/left atrial volumes were not associated to the development of AFL on multivariate analysis; I suspect that the limited number of patients included in the study prevents from a more detailed analysis that could yield a picture more adherent to the clinical reality. Assigning a precise cut-off value of RACT for the future development of typical AFL is a "debatable" action, just in terms of "let's do cavo-tricuspid ablation" as empirical and prophylactic maneuver (9). In this regard, I'd be cautious, awaiting more robust and consistent data from larger population studies. On the contrary, I give credit to the authors that longer RACT can be found in patients undergoing AF ablation and without clinical history of typical AFL. Under these circumstances, I'd be in favor to add a "prophylactic" AFL ablation to left atrial ablation for AF, since the chance to have typical AFL afterwards is not negligible (2,10).

Therefore, despite the actual limitations of the role played by RACT, I support the estimation of this parameter in patients candidate to AF ablation as to reduce the likelihood of AFL occurrence over the follow up, even though, up to now there is no universal consensus of a preventive AFL ablation. What is lacking is the information about the potential influence on RACT of drugs used in patients with AFL. Did any antiarrhythmic medications affect the calculation of the conduction time across the right atrium? Do the antiarrhythmic drugs influence RACT at the same degree in patients with and without AFL? These questions deserve to be fulfilled in the near future including a higher number of patients as to corroborate the true value of RACT as predictive parameter of future risk of AFL occurrence

Functional or structural?

Least but not last is the evaluation of the underlying substrate that can lead to the clinical appearance of AFL. *Atrial myopathy* is the phrasing to highlight the presence of a progressive atrial disease which promotes areas of fibrosis and scars. If one considers that patients in AFL group were older and with larger atrial volume, it is conceivable that the myopathic process influences the conduction properties of the atrium and, thus it is not surprising to find prolonged RACT. Comorbidities such as systemic hypertension, OSA, ischemic disease can greatly affect the cardiac electrophysiology and specifically of the atrium; these pathologic clinical aspects were significantly associated to the AFL patients suggesting an undoubtable role in favoring the formation of the ideal pathophysiological substrate for reentry circuits.

After several decades of investigational studies and a great wealth of data collected about the typical atrial flutter, we are still here to be nicely fascinated by the basic electrophysiology and surprised of how much is still to be learned as to improve our understanding of this common cardiac arrhythmia. In the era dominated by novel technologies and sophisticated softwares supporting 3D mapping systems used in many EP labs, the continuous investigation of the electrophysiologic properties of cardiac tissue should not be disregarded.

but carefully stewarded.

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