Atrial flutter (AFL) is one of the most common supraventricular arrhythmias in clinical practice. A significant number of patients with AFL will develop atrial fibrillation (AF) afterwards. Moreover, a sizable proportion of patients who undergo AF ablation will develop AFL (atrial macroreentrant tachycardia is a more accurate term) as a secondary arrhythmia after the ablation procedure. Approaches to the management and use of anticoagulation therapy are considered equivalent for AFL and AF and the same stroke prevention strategies are therefore recommended. Appropriate management of AFL is not only important due to the symptoms, but also to the increased risk of complications, such as thromboembolism and stroke, which may lead to permanent disability or death. So far, different therapeutic strategies have been introduced for AFL, including rate control, cardioversion to sinus rhythm (principally electrical cardioversion or high-rate stimulation), and catheter ablation. Due to the low success rate of pharmacological antiarrhythmic approaches in AFL, long-term drug therapy is less acceptable nowadays, and is recommended when ablation is not feasible. Catheter ablation is a promising treatment method to maintain sinus rhythm, especially in the case of cavotricuspid isthmus-dependent AFLs. Catheter ablation for typical right atrial isthmus-dependent AFL has yielded a high success rate of 90-98% and a low recurrence rate of only 2-15%; however, successful ablation depends on the correct identification of the reentrant circuit responsible for the arrhythmia and its critical isthmus. For non-isthmus-dependent right or left atrial macroreentrant tachycardia (so-called atypical AFL), the precise identification of the critical isthmuses for successful catheter ablation procedures is certainly more complex as multiple re-entrant pathways in the right and/or left atrium may be involved. Fast and accurate identification and understanding of the re-entrant pathway and the critical conduction zone are crucial for the development and performance of a successful ablation strategy.

The recent study by Adragão et al. sought to help us better understand how to identify and localize the critical isthmus in left AFL and introduced a stepwise approach. They took advantage of a new feature of an electroanatomical mapping (EAM) system, which produces a histogram of local activation times (LAT), in addition to the activation and voltage maps.

An LAT histogram is actually a graphical illustration of the LAT values of all the points that contribute to the LAT col-
oring on active maps and provides a visual representation of the activation throughout the tachycardia cycle length (TCL). This tool helps electrophysiologists to identify the part of the cycle which needs to be mapped further. In principle, the range of the LAT histogram is determined based on the window of interest (WOI) and when a point is edited to be outside of the WOI, the range is expanded accordingly. The height of the histogram shows the number of points that fall within the bin range; while each bin is color coded based on the LAT values of the points associated with it. In Figures 2 and 3 of Adragão’s article, an example of an LAT histogram is presented.

In order to identify the critical isthmus in left AFL, Adragão et al. proposed a very logical stepwise approach. After identifying the LAT-valleys (defined as zones in the LAT histogram with 20% or less points relative to the highest bar [maximum LAT value]); they checked whether the identified LAT-valleys corresponded to slow conduction areas and heterogeneous low-voltage zones. They then quantified the LAT-valley atrial surface with the Carto® area measurements feature to identify whether the regions corresponded to the successful ablation site or not. Their initial findings showed that that all these areas corresponded to the primary LAT-valley identified in the global histogram analysis, which confirmed the accuracy of their method. In fact, they introduced a new electrophysiologic triad for identification and localization of the left critical isthmus in AFL which encompasses: (1) areas of low- voltage (0.05 to 0.3 mV); (2) sites of deep histogram valleys (LAT-valleys) with less than 20% density points relative to the highest density zone; and (3) a prolonged LAT-valley duration which included 10% or more of the TCL. Although it was a retrospective study in a small group of patients, their results may open new windows toward a new less complex approach that is less reliant on extensive ablation lesions. Their findings also helped us to gain a better understanding of the utility of three-dimension electroanatomic mapping in the identification of the critical isthmus and potential ablation sites in patients with left AFL. Is this new strategy powerful enough to replace all other mapping strategies for successful ablation of non-isthmus-dependent atrial macro re-entrant tachycardia? No, because some limitations related to the different modes of activation mapping remain. One is that these methods are not able to define precisely the active or leading re-entrant circuit. This can only be achieved with application of elegant classical electrophysiological strategies such as entrainment mapping. Overall, the introduction of the new LAT histogram strategy is a useful additional tool to improve the fast recognition of critical isthmus sites in patients with complex atrial macroreentrant tachycardia.

Conflicts of interest
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References