Published in final edited form as:

Heart Rhythm. 2019 April ; 16(4): 562–563. doi:10.1016/j.hrthm.2018.11.018.

Predictability in complex atrial arrhythmias: The N/N-1 algorithm to guide ablation of atrial tachycardias

Daniel W. Kaiser, MD, FHRS^{*}, Albert J. Rogers, MD, MBA[†], and Sanjiv M. Narayan, MD, PhD, FHRS[†]

^{*} St Helena Hospital, St Helena, California

[†] Department of Medicine, Stanford University, Stanford, California.

Macroreentrant atrial tachycardias (ATs) are some of the most challenging cases to accurately map and successfully ablate.¹ Because patterns of scar and wavefront propagation are unique to each patient, the ablation procedure tends to be complex, time-consuming, and sometimes frustrating. Complex and detailed activation maps are painstakingly created, targets identified, and the tachycardia circuit successfully ablated. but frequently this results in the induction of a second, slower AT that requires repeating the entire process.²

In this issue of *HeartRhythm*, Takigawa et al³ report an ingenious clinical algorithm to better understand, anticipate, and potentially expedite such cases. To create the algorithm, the authors retrospectively examined high-resolution electroanatomic maps of 52 ATs in 45 patients with previous atrial fibrillation (AF) ablation to identify the index AT circuit, the site of likely successful ablation, and the regions of wavefront collision. Depending on the location of the ablation site, atrial wavefronts were labeled as being generated distal to (the N beat) or proximal to (N-1 beat) that site. If 2 N beats or 2 N-1 beats collide, the authors predicted that the tachycardia should terminate after ablation of the identified region. However, if an N beat meets an N-1 beat, the authors predicted that after ablating the index tachycardia, a new tachycardia will be unmasked. The tachycardia cycle length (TCL) of the unmasked AT should then be the TCL of the original AT plus double the time from when the wavefront leaves the index AT circuit to when the wavefront of N/N-1 collides in the second AT circuit. The authors tested this algorithm prospectively in 172 patients with 194 reentrant ATs in whom, after ablation of the first AT, a second slower AT developed in 23% (45/194). In this prospective cohort, the algorithm predicted AT termination or conversion to a subsequent AT with 96% accuracy and accurately predicted TCL of unmasked tachycardias with a median error of 6 ms.

The authors should be congratulated for developing this algorithm and prospectively demonstrating its accuracy. By considering the impact of AT ablation in terms of global atrial activation, subsequent ATs can be anticipated by the N/N-1 algorithm to expedite mapping and ablation. One of the most satisfying aspects of the study is that it solidifies the predictability of multiple potential arrhythmic pathways even in previously ablated atria.

Intuitively, one may have expected changes in rate or new ablation lesions to alter activation patterns, functional block, and anatomic obstacles, and to create unpredictable tachycardias. However, the accuracy of predictions highlights the fact that seemingly complex phenomena typically follow relatively simple principles. Marine et al⁴ noted in classic typical atrial flutter that wavefronts may collide in the left atrium, yet, of course, cavo-tricuspid isthmus ablation rarely unmasks left atrial flutters. This is now predictable from the study by Takigawa et al³ because collisions arise distal to (N to N collision; if counterclockwise) or proximal to (N-1 to N-1 collision, if clockwise) the ablation site. Similarly, we recently demonstrated during overdrive pacing that the timing to entrainment of atrioventricular nodal reentrant tachycardia and atrioventricular reentrant tachycardia was highly predictable, enabling creation of an algorithm that predicted how many cycles of pacing would be needed to advance and then entrain tachycardia.⁵ Extensions of this work account for measurable decremental conduction.⁶ Such concepts may help refine mapping and ablation procedures, and may improve antitachycardia pacing for ventricular tachycardias.

As with all good studies, however, the work of Takigawa et al³ presents some limitations. First, of the 273 ATs included in the study, 79 (29%) were excluded, primarily because of premature AT termination or unclear flutter circuits. Thus, although the algorithm predicts characteristics of a second AT, many may terminate prematurely or be difficult to map even in experienced hands. Second, the authors note that most left atrial ATs were caused by iatrogenic obstacles or regions of slow conduction from previous ablation. Accordingly, the accuracy of the algorithm may vary at centers at which AF ablation of the posterior wall, complex fractionated atrial electrograms, rotational or focal drivers,^{7,8} or ganglionic plexuses or other targets varies from the Bordeaux practice. This requires additional validation. Third, Yamashita et al⁹ and Baykaner et al¹⁰ recently reported that ATs after AF ablation may be microreentrant or focal sites related to residual unablated AF driver sites. This algorithm, in theory, may not identify those tachycardias. Thus, although it may be tempting to use the algorithm to predict ablation sites of secondary ATs, one should still currently demonstrate that they are manifest.

The findings of this study suggest that multiple complex tachycardias after ablation for AF are predictable and operate in the “newtonian world,” where cycle lengths are additive and secondary circuits can be anticipated and characteristics predicted. Given the increasingly defined interactions between atrial macroreentry with AF, it is intriguing to speculate whether mathematical relationships can be defined that govern transitions of macroreentry to fibrillatory activity. Such unified concepts could revolutionize our thinking of complex arrhythmias, their interaction with structural and functional substrates, and the approach to therapy for fibrillation. We again congratulate Takigawa et al³ and await future mathematical formulations that may further improve our understanding of complex arrhythmias.

Acknowledgments

Dr Kaiser reports consulting income from Abbott, AtriCure, Medtronic, and Terumo Medical; and is inventor of patents related to anti-tachycardia pacing algorithms. Dr Rogers is supported by grants from NIH (F32 1F32HL144101-01) and Stanford University (Stanford Society of Physician Scholars). Dr Narayan is supported by grants from NIH (R01 HL83359, HL122384, HL103800); is co-inventor of patents owned by the University of California and Stanford University; and reports consulting income from Abbott, UpToDate, and the American College of Cardiology.

References

1. Gerstenfeld EP, Marchlinski FE. Mapping and ablation of left atrial tachycardias occurring after atrial fibrillation ablation. *Heart Rhythm* 2007;4:S65–S72. [PubMed: 17336888]
2. Arantes L, Klein GJ, Jaïs P, Lim KT, Matsuo S, Knecht S, Hocini M, O'Neill MD, Clémenty J, Haïssaguerre M. Tachycardia transition during ablation of persistent atrial fibrillation. *J Cardiovasc Electrophysiol* 2011;22:506–512. [PubMed: 21114705]
3. Takigawa M, Derval N, Martin C, et al. A simple mechanism underlying the behavior of reentrant atrial tachycardia during ablation. *Heart Rhythm* 2019; 16:XXX-XXXX.
4. Marine JE, Korley VJ, Obioha-Ngwu O, Chen J, Zimetbaum P, Papageorgiou P, Milliez P, Josephson ME. Different patterns of interatrial conduction in clockwise and counterclockwise atrial flutter. *Circulation* 2001;104:1153–1157. [PubMed: 11535572]
5. Kaiser DW, Hsia HH, Dubin AM, Liem LB, Viswanathan MN, Zei PC, Wang PJ, Narayan SM, Turakhia MP. The precise timing of tachycardia entrainment is determined by the postpacing interval, the tachycardia cycle length, and the pacing rate: theoretical insights and practical applications. *Heart Rhythm* 2016;13:695–703. [PubMed: 26611239]
6. Kaiser DW, Nasir JM, Liem B, Brodt CR, Motonaga K, Ceresnak S, Turakhia M, Dubin A. A novel pacing maneuver to verify the post-pacing interval minus the tachycardia cycle length while adjusting for decremental conduction: using “dual chamber entrainment” for improved supraventricular tachycardia discrimination. *Heart Rhythm* 2018; S1547–5271(18)31166–4.
7. Narayan SM, Krummen DE, Shivkumar K, Clopton P, Rappel W-J, Miller JM. Treatment of atrial fibrillation by the ablation of localized sources: CONFIRM (Conventional Ablation for Atrial Fibrillation With or Without Focal Impulse and Rotor Modulation) trial. *J Am Coll Cardiol* 2012;60:628–636. [PubMed: 22818076]
8. Haïssaguerre M, Hocini M, Denis A, et al. Driver domains in persistent atrial fibrillation. *Circulation* 2014;130:530–538. [PubMed: 25028391]
9. Yamashita S, Hooks DA, Shah A, et al. Atrial tachycardias: cause or effect with ablation of persistent atrial fibrillation? *J Cardiovasc Electrophysiol* 2018; 29:274–283. [PubMed: 29072796]
10. Baykaner T, Zaman JAB, Rogers AJ, et al. Spatial relationship of sites for atrial fibrillation drivers and atrial tachycardia in patients with both arrhythmias. *Int J Cardiol* 2017;17:31816–31818.