Progressive modification of rotors in persistent atrial fibrillation by stepwise linear ablation

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Introduction

While percutaneous pulmonary vein (PV) isolation has proved to be an effective treatment of paroxysmal atrial fibrillation (AF), radiofrequency ablation of persistent atrial fibrillation (PeAF) has been much less successful. However, improved outcomes are reported for catheter ablation of PeAF guided by real-time dominant frequency analysis and by dynamic 3-dimensional (3D) electroanatomic mapping, particularly when this is used to target the origin and trajectory of rotors. Here, we present a complete case study of a patient with PeAF who was treated successfully with stepwise linear ablation. A novel frequency analysis approach, in which a wavelet filter and multiscale peak detection methods are combined, was applied retrospectively to electrograms recorded during the procedure. This reveals progressive changes in the number, location, and form of reentrant sources during ablation.

Case report

The patient was a 58-year-old woman with a 7-year history of AF and no prior ablation and classified as having PeAF. A decapolar 6-F electrode catheter (St Jude Medical, St Paul, MN) was positioned in the coronary sinus. A 9-F 64-channel electrode balloon was positioned in the middle of the left atrium (LA) via a transseptal approach with the J-tip wire lodged in the left superior PV. An 8-mm-tip deflectable ablation catheter (St Jude Medical) was then introduced into the LA via a second transseptal puncture. Intravenous heparin was administered to maintain an activated clotting time of 300–350 seconds after transseptal puncture. LA endocardial geometry was acquired, and a well-defined linear ablation procedure was followed. This involved 2 ablation lesion sets delivered at upper limits of 60 W and 60°C. A figure 7 lesion set was delivered across the LA roof to the ridge between the left atrial appendage and the left superior PV and then downward to the anterior antrum of the left inferior PV. A second (figure 0) lesion set followed a path from the roof line along the anterolateral junction of the right PVs, and the circuit was closed by extending a line between the right inferior PV and the mitral valve (MV) annulus adjacent to the coronary sinus. Circumferential PV isolation was not performed. Noncontact electroanatomic maps were recorded for ~10 seconds at regular specified intervals throughout the ablation process by using an EnSite 3000 system (St Jude Medical; sampling rate 1200 Hz; bandwidth 2–600 Hz; sensitivity 10 μV).

We have exported 2048 3D data cloud coordinates of LA geometry from the EnSite system and created the triangle mesh based on the data cloud by using custom-written MATLAB applications (MathWorks, Inc, Natick, MA). By using the EnSite geometrical data, we estimated that the superior-to-inferior depth of LA geometry was ~38.0 mm, the anterior-to-posterior width was ~41.7 mm, and the lateral-to-lateral length (excluding PV sleeves) was ~50.1 mm. The distance from anywhere in LA geometry excluding the PV sleeves to the center of the EnSite array was <40 mm owing to the position (along the septum-to-ridge direction of the LA) and dimension (45×18 mm²) of the EnSite array. Virtual electrograms from each LA geometry location point were analyzed and linked to their anatomical structure (Figure 1A). Frequency analysis was performed on each recorded electrogram, and key steps involved are illustrated in Figure 1B for a 1-second virtual electrogram segment.
A novel frequency analysis approach is demonstrated to effectively process atrial unipolar electrograms acquired by a noncontact balloon. Frequency analysis reveals progressive changes in the number, location, and form of reentrant sources during linear ablation. 3D panoramic electroanatomic mapping is crucial for accurate dynamic description of atrial fibrillation and guidance of catheter ablation.

Before catheter ablation, specific regions on the LA roof and interatrial septum were characterized by high-frequency activity (Figure 2A). Typically activity died out after a few cycles, but interatrial septum were characterized by high-frequency activity 70 ms apart, the peak with the highest derivative gradient was identified as local atrial activation (black circles in panel IV) when ≥70 ms apart. Where maxima satisfied the gradient criterion, but were <70 ms apart, the peak with the highest derivative gradient was identified as local activation and others as fractionation (open circles in panel IV). Regional atrial frequency was estimated from the local activation count during the recorded time interval except the time windows (~120 ms) in which ventricular depolarization occurred. Activation time maps were constructed by tracking the propagation of local activation complexes across the LA surface.

Before catheter ablation, specific regions on the LA roof and interatrial septum were characterized by high-frequency activity (Figure 2A). Typically activity died out after a few cycles, but then recurred at the proximate regions. Figure 7 lesions altered this pattern, especially along the roof line (Figure 2B). High-frequency activity appeared to become more uniform and was also evident at the junction of the right superior PV and in the inferior septum. Partway through figure 0 lesions, a macroreentrant flutter circuit was established and the corresponding frequency map is shown in Figure 2C. The observation of 64 representative virtual electrograms confirmed a stable 2:1 atrioventricular rhythm with regions of high-amplitude fractionation in the roof and inferior septum indicative of local conduction delays. Activation was initiated in the anterior wall and spread slowly to the roof. Typical virtual electrograms from the site of initial activation (white circle) and the adjacent anterior septum are shown in Figure 2D.

We observed transient rotors before and during ablation. These were sustained for 2 cycles at most and originated near sites of high-frequency activity (in the LA roof, the interatrial septum, PV junctions, and the MV annulus). Figure 3A shows a rotor that started near the MV annulus and circulated clockwise on the anterior surface of the LA before ablation. A more short-lived rotor that originated in the interatrial septum after figure 7 lesions is presented in Figure 3B.

Sinus rhythm was established with the completion of figure 0 lesions. Postprocedural AF occurred several seconds later, but sustained sinus rhythm was restored shortly soon after consolidating the “box set” of linear ablation lines. Mapping and ablation took 49 minutes, and the patient was free from AF at 12 months.

Discussion

3D electroanatomic mapping over extended periods is beneficial for an accurate dynamic analysis of AF. This is difficult to achieve with point-to-point contact mapping systems because electrical activity in AF is nonstationary. However, 3D mapping can be carried out using atrial electrograms recorded simultaneously with multielectrode intracardiac basket catheters. The CONFIRM trial demonstrates the potential of this approach, but it requires electrodes to be in contact with the endocardial surface of the atria, which is not always possible. An alternative method is to deploy noncontact arrays and use inverse mapping to reconstruct 3D potential distributions.

In this case study, simultaneous virtual electrograms at multiple atria regions have been recorded at intervals throughout a successful PeAF ablation procedure. These were analyzed subsequently using novel wavelet-based signal analysis tools that enable regional activation times and frequencies to be determined. The findings reinforce the utility of real-time 3D electroanatomic mapping for identifying nonstationary reentrant sources during AF ablation and demonstrate that noncontact intracardiac mapping systems can be used for this purpose. A potential advantage of our approach is that it can be used to identify regions of unstable rotor activity.

A problem with virtual electrograms reconstructed with noncontact methods is that they are unipolar signals. They have greater spatiotemporal complexity than do bipolar contact recordings and can be more difficult to interpret, particularly during PeAF. We have adapted a wavelet-based technique reported by Houben et al7 to detect local activation times in the presence of far-field potentials. This has enabled us to track reentrant activation and to determine local activation frequency more directly than is possible with dominant frequency analysis.

In this work, we have demonstrated that originally and subsequently identified sources are located primarily in the atrial roof and septum. The first roof line ablation was ~10 mm from the closest high-frequency activity (Figure 2A). After the completion of figure 7 lesions, high-frequency activity in the roof was shifted by >20 mm from the roof line (Figure 2B) and disappeared after subsequent ablation. Figure 0 lesions (Figures 2A–2C) were ~10 mm away from the high-frequency region in the septum and sources in this
Figure 1  Three-dimensional endocardial surface of the left atrium using coordinates provided by the EnSite system (St Jude Medical) was reconstructed at the anterior view (A), and a typical atrial virtual electrogram was processed by the proposed signal processing tools (B). Wavelet-based peak atrial impulse detection (the far-field ventricular activation complex is indicated in grey). I: Typical virtual electrogram of the patient with persistent atrial fibrillation at the posterior inferior septum. II: The electrogram is attenuated during the ventricular activation time window. III: Wavelet scalogram of panel II, indicating the magnitude and duration of each of the 15 scales. White indicates a negative deflection and black a positive deflection. IV: Derivative signal constructed from 7 lowest wavelet scales. The maxima for which adjacent derivative gradients exceed a preset threshold are indicated by closed circles, with the peak-to-peak distance ≥ 70 ms. Where peaks are separated by < 70 ms, the peak with the highest derivative gradient was identified as local activation (closed circles) and others as fractionation (open circles). ASS = anterior superior septum; LAA = left atrium appendage; LIPV = left inferior pulmonary vein; LSPV = left superior pulmonary vein; MV = mitral valve; PIS = persistent atrial fibrillation; RIPV = right inferior pulmonary vein; RSPV = right superior pulmonary vein.
Figure 2  Regional frequency maps rendered on the anterior LA wall immediately before (A) and during (B and C) linear ablation. Intraprocedural AF shown in images C and D presents 2-ms electrogram segments for the highest-frequency region in the inferior interatrial septum (left panel) and the site at which activation originates (right panel; indicated by the circle in image C). Abbreviations as in Figure 1.
There are some limitations of this study. First, atrial electrograms recorded in the LA from 1 patient were analyzed; in the future, we will include more patients and biatrial data. Second, only activation maps were used instead of more robust phase analysis to characterize rotors, even though the work by Narayan et al12 concluded that both approaches led to similar qualitative results. Furthermore, in this study we directly processed unipolar electrograms which is a challenge to apply the existing phase analysis approach due to specific morphology of the atrial signals.13 Lastly, it is known that virtual electrograms in AF exhibit morphological artifact when compared with contact bipolar electrograms,14 particularly in some regions of the larger atria where the distance from the center of the mapping probe is > 40 mm.15

References
1. Brooks AG, Stiles MK, Laborderie J, Lau DH, Kuklik P, Shipp NJ, Hsu LF, Sanders P. Outcomes of long-standing persistent atrial fibrillation ablation: a systematic review. Heart Rhythm 2010;7:835–846.
2. Atienza F, Almendral J, Jalife J, Zlochiver S, Ploutz-Snyder R, Torrecilla EG, Areval A, Kalifa J, Fernández-Avilés F, Berenfeld O. Real-time dominant frequency mapping and ablation of dominant frequency sites in atrial fibrillation with left-to-right frequency gradients predicts long-term maintenance of sinus rhythm. Heart Rhythm 2009;6:33–40.
3. Yao Y, Zheng L, Zhang S, He DS, Zhang K, Tang M, Chen K, Pu J, Wang F, Chen X. Stepwise linear approach to catheter ablation of atrial fibrillation. Heart Rhythm 2007;4:1497–1504.
4. Wu L, Yao Y, Zheng L, et al. Long-term follow-up of pure linear ablation for persistent atrial fibrillation without circumferential pulmonary vein isolation. J Cardiovasc Electrophysiol 2014;25:471–476.
5. Narayan SM, Patel J, Mulpedu S, Krummen DE. Focal impulse and rotor modulation ablation of sustaining rotors abruptly terminates persistent atrial fibrillation to sinus rhythm with elimination on follow-up: a video case study. Heart Rhythm 2012;9:1436–1439.
6. Narayan SM, Crimean DE, Shivkumar K, Clopton P, Rappel WJ, Miller JM. Treatment of atrial fibrillation by the ablation of localized sources. J Am Coll Cardiol 2012;60:628–636.
7. Houwen RP, de Groot NM, Allessie MA. Analysis of fractionated atrial fibrillation electrograms by wavelet decomposition. IEEE Trans Biomed Eng 2010;57:1388–1398.
8. Zhao J, Yao Y, Huang W, Shi R, Zhang S, Legrice U, Lever NA, Small BH. Novel methods for characterization of paroxysmal atrial fibrillation in human left atria. Open Biomed Eng J 2013;7:29–40.
9. Haissaguerre M, Hocini M, Sanders P, et al. Localised sources maintained atrial fibrillation organised by prior ablation. Circulation 2006;113:616–625.
10. Ng J, Kadish AH, Goldenberger JH. Technical considerations for dominant frequency analysis. J Cardiovasc Electrophysiol 2007:18:757–764.
11. Pandit SV, Jalife J. Rotors and the dynamics of cardiac fibrillation. Circ Res 2013;112:849–862.
12. Narayan SM, Krummen DE, Enyeart MW, Rappel W-J. Computational mapping of cardiac fibrillation: evidence for localized mechanizations for ablation of atrial fibrillation. PLoS ONE 2012;7:e46034.
13. Umapathy K, Nar K, Masse S, Krishnan S, Rogers J, Nash MP, Nanthakumar K. Phase mapping of cardiac fibrillation. Circ Arrhythm Electrophysiol 2010;3:105–114.
14. Narayan SM, Wright M, Derval N, et al. Classifying fractionated electrograms in human atrial fibrillation using monophasic action potentials and activation mapping: evidence for localized drivers, rate acceleration, and nonlocal signal etiologies. Heart Rhythm 2011;8:244–253.
15. Earley MJ, Abrams DJ, Sporton SC, Schilling RJ. Validation of the noncontact mapping system in the left atrium during permanent atrial fibrillation and sinus rhythm. J Am Coll Cardiol 2006;48:485–491.