Mini Review

Ablation of atrial fibrillation (AF) is our most frequently performed complex ablation procedures. Our approach to ablation of persistent AF represents a confluence of the recent literature with evolving technical capability. Pulmonary vein isolation (PVI) may be considered the cornerstone of AF ablation. However, recent studies identify the atrial substrate, rather than PVs, as a major source of persistent AF [1]. This finding is consistent with identification of rotors as drivers of human AF [2]. Recognition of the potential importance of substrate and rotors was easily anticipated. Given that wavebreak occurs constantly during AF as high-frequency wavefronts encounter structural and functional heterogeneities, with fractionation resulting in localized conduction failure and the tendency for rotation [3]. It seems self-evident that certain rotors may persist, acting as drivers of AF.

We recognize that the resolution of available mapping systems continues to be the Achilles heel of rotor mapping and ablation. Nevertheless, complex fractionated atrial electrograms (CFAEs) should be found in the core region of rotors [3], although CFAEs may also arise from other processes (discharging ganglionatedplexi, heterogenous conduction, wavel collision). In this regard, ablation of CFAEs would likely interrupt the drivers of AF, with the most dense and tenacious CFAE nests being most important to AF maintenance [4]. Recognition of the potential importance of substrate and rotors was easily anticipated, given that wavebreak occurs constantly during AF as high-frequency wavefronts encounter structural and functional heterogeneities, with fractionation resulting in localized conduction failure and the tendency for rotation [3]. It seems self-evident that certain rotors may persist, acting as drivers of AF.

We routinely acquire a pro-procedure CT of the LA and PV insertions. Unless patients have a CIED with monitoring ability for AF, I also strongly prefer to implant a Medtronic Reveal XT or LINQ insertable cardiac monitor at least 4 weeks prior to ablation to obtain baseline AF burden, and subsequently to monitor the success of each ablation. This serves as an invaluable guide to discontinuation of anti-arrhythmic medications, symptom correlation with any AF recurrence, and anticoagulation. We perform ablation with therapeutic INR, however prefer to hold NOACs (Xarelto, Eliquis, Pradaxa) pre-ablation, with goal to resume full dose therapy with the evening dose post ablation. We do not use Pradaxa immediately post ablation due to the lack of a reversal agent. All AF ablations are performed under general anesthesia, thereby minimizing patient motion for stable CARTO mapping. Antiarrhythmic drugs are held at least 7 days prior to ablation, with amiodarone held as long as clinically reasonable. These are resumed immediately post ablation, and continued during the 3 month blanking period post ablation.

We use four Biosense Webster catheters, including Sound star ICE, CS, Pentarray, and Surround Flow (SF) ablation catheters, with SL1 transseptal sheaths. Patients with persistent AF typically present in AF, otherwise AF is induced with burst pacing and maintained with low-dose isuprel as needed. The Pentarray catheter is first used to create RA geometry using fast activation mapping (FAM). During FAM, the CARTO software is used to perform CFAE analysis of all EGMs recorded by the Pentarray, with simultaneous generation of an atrial scar map (<0.05 mV) [7]. The ablation catheter is advanced within the CS to incorporate this geometry, with the Pentarray subsequently used for CFAE mapping within the CS when proximal poles of the CS catheter display complex signals. Ablation targeting the most complex signals within the RA and CS is then performed (10-20W within the CS, 20-40W within the RA, 15 cc/min irrigation) with high-output pacing (10 mV, 10 m sec) to assess for phrenic nerve capture along the lateral RA. Heparin bolus is given during RA studies, such that ACT >300 is achieved prior to transseptal nerve capture along the lateral RA. Heparin bolus is given during RA studies, such that ACT >300 is achieved prior to transseptal puncture, minimizing the risk of thrombus formation within the arterial circulation. Finally ICE is used to create a preliminary LA geometry, localize the esophagus, and to mark the LPV carina.

As illustrated in Figure 1, the FAM of the CS and reference point at the LPV carina (Panel A, all obtained from within the RA prior to transseptal puncture) allows the CT image of the LA to be positioned precisely relative to the RA (Panel B). Following transseptal catheterization, generating the LA FAM (Panel C) is then like manipulating the Pentarray within a known geometry.
provided by the CT image. By preventing blind mapping of the LA, this workflow allows LA geometry to be obtained quickly and with certainty. We find the resulting geometry is often hardly distinguishable from the CT, likely due to less deformation artifact compared to traditional lasso catheters and the ability of the Pentarray splines to better align with LA contours. CT merge is therefore not needed and not performed. An added benefit of the Pentarray is less concern for entrapment in the mitral valve apparatus.

Figure 2 shows CARTO displaying all LA EGMs meeting CFAE criteria (high [red dot] and medium [blue dot] confidence, default settings). We find CFAEs are typically scattered around the PV antra, with clusters found in 2-4 areas throughout the LA, which typically represents a combination of sites posterior to the LPVs, along the LA ridge, anterior to the LAA or RPVs, the interatrial septum, or along the LA floor adjacent to the CS. Comparison to the associated voltage map (not shown) indicates how CFAEs may correspond to regions of scar, serving as a further guide to effective ablation. After creation of the CFAE maps, the distribution of complex signals, as determined by the CARTO software, suggests where antral PVI (Figure 3) and linear lesion sets (LA roof, mitral isthmus, CS isolation) may be placed to maximally interrupt CFAEs. By performing such CFAE-guided AF ablation, with creation of standard lesion sets as would otherwise be indicated by clinical circumstances, patients may also benefit from CFAE ablation without increased risk for macro-reentrant tachycardias following CFAE ablation, as may sometimes occur after targeting CFAEs [6].

AF frequently terminates during CFAE-guided ablation, allowing assessment of block across ablation lines. Otherwise, ibutilide may be infused to simplify AF dynamics, allowing identification of remaining critical CFAE sites. Figure 4 shows a paroxysmal AF pattern in the presence of an anti-arrhythmic (Figure 4C). On this basis, AF ablation was performed October 2, 2013, with termination of AF occurring during ablation of CFAEs anterior to the LAA following CFAE-guided PVI (Panel A). Following termination, AF was completely non-inducible with atrial burst pacing, both under control conditions and following isuprel infusion. Loop recorder monitoring demonstrated no AF recurrence post ablation (Figure 4C).
Pulmonary Vein Isolation (PVI) and Ablation of Atrial Fibrillation Guided by Complex Fractionated Atrial Electrograms (CFAEs)

Figure 4: Termination of AF during CFAE ablation anterior to the LAA (arrow, Panel A). Following CFAE-guided PVI and AF termination, AF was non-inducible with atrial burst pacing ± isuprel (Panel B). Continuous AF monitoring with a Reveal XT loop recorder demonstrates no AF recurrence post ablation (Panel C).

References


