Catheter ablation has rapidly gained acceptance as a mainstay of therapy for many symptomatic patients with atrial fibrillation since the original publication by the Bordeaux group. Early on it became apparent that in most patients with paroxysmal AF, the arrhythmia was initiated by focal firing in the pulmonary veins. Ablation focused on elimination of such triggers and was largely limited to patients who would stay in sinus rhythm long enough to allow successful mapping within the pulmonary veins. As this was time consuming and was associated with high risk of developing pulmonary vein stenosis, ablation lesions were moved further and further away from the sources of focal firing with co-development of Circumferential Pulmonary Vein Ablation (CPVA) aiming to encompass pulmonary vein ostia with circular lesions without verification of conduction block and Segmental Pulmonary Vein Isolation evolving into Pulmonary Vein Antrum Isolation with the targeted area similar to that in CPVA but with requisite documentation of entry and / or exit block of conduction. While successful in the majority of patients with paroxysmal AF, these approaches were lacking in patients with persistent and permanent AF. A concurrent approach addressing the fibrillatory substrate was developed and proved to be a successful standalone alternative to lesions encircling the pulmonary veins at one center. This approach had the advantage of better tailoring the lesion set to the individual patient and potentially limiting energy delivery and associated risks. Addressing fibrillatory conduction as an adjunct to ablating triggers had since become incorporated into the lesion set targeting persistent and permanent atrial fibrillation in a stepwise approach popularized by the Bordeaux group. At the same time better tools have enabled clinicians to probe deeper into the complex interaction of the mechanisms initiating and maintaining atrial fibrillation. So in experimental models of atrial fibrillation “drivers” or areas of microreentry were seen surrounded by areas of fibrillatory conduction. Research has pointed to the possibility of the “drivers” or “triggers” to be in close proximity to areas of atrial myocardium innervated by autonomic ganglionated plexi with areas of fibrillatory conduction 1-2 cm remote from these.
This is an important contribution to the literature on the mechanisms of atrial fibrillation highlighting frequency distribution differences between paroxysmal and chronic AF, where patients with persistent AF have a more uniform distribution of the areas with high DF and faster global AF cycle lengths. These differences may be responsible for the difference in ablation success rates between AF subtypes. The investigators demonstrated a close spatial relationship between areas of high DF and surrounding areas of high fractionation, suggesting that the two are indeed related and as was seen in experimental optical mapping studies, fibrillatory activity represented by CFAEs surrounds more organized “drivers” with high DF. Such local frequency gradients were further supported by limited activation mapping. No conclusions could be drawn on whether or not ablation of the high DF sites could help improve outcomes since DF analysis occurred offline and DF mapping was not used to guide ablation. There is emerging evidence that ablation guided using DF or CFAE mapping may result in higher success rates, but the question of whether CFAEs or DF is a more important ablation target remains to be answered. While development of tools which will allow rapid high density activation mapping to better delineate the relationship between DF and CFAE and hopefully fine-tune the ablation strategy is ongoing, ablation targeting anatomical substrate of the pulmonary veins with tailored adjunctive defragmentation will likely remain at the core of invasive treatment for AF.

References