Research Highlight

Heart team approach for left atrial appendage therapies: in addition to stroke prevention—is electrical isolation important?

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Background

The left atrial appendage (LAA) has been identified as a culprit for thromboembolic complications in the setting of atrial fibrillation (AF). LAA amputation has been integral to surgical treatment of AF since its early inception. As such, strategies to eliminate blood flow within the LAA have been developed and have proven their efficacy in preventing thromboembolic complications (1). These techniques are based on creating a mechanical barrier eliminating the LAA from blood flow, thereby preventing stasis that will cause thrombus formation. This mechanical barrier can be placed either endocardially (using either catheter-based techniques or sutures during open heart surgery) or epicardially. The main difference between these two approaches is that epicardial techniques involve the external application of mechanical force at the base of the LAA, hence truly closing the neck of LAA at its orifice, which over time leads to irreversible LAA fibrosis and subsequent disappearance (2). Only recently has the focus shifted towards the LAA, due to thromboembolic complications after oral anticoagulation in patients with elevated CHADS-VASC scores (1). But more importantly, the negative electrophysiological properties of the LAA might become a game changer as endocardial and epicardial LAA exclusion strategies produce different electrophysiological results.

LAA source of AF

Since Haïssaguerre's landmark work (3), the pulmonary veins have become the most important target for ablation of paroxysmal AF. However, lesion pattern and extent in the setting of non-paroxysmal AF remain under heavy debate. Many other cardiac structures play a role in the initiation and perpetuation of non-paroxysmal AF. Effective resolution of non-paroxysmal AF requires ablation of additional targets, most commonly the superior vena cava, ligament of Marshall, coronary sinus, crista terminalis and left atrial (LA) posterior wall. Recent studies have shown the LAA to be the site of triggers that can induce episodes of paroxysmal AF and of re-entrant drivers that participate in the maintenance of persistent AF (4,5). Furthermore, an interesting report demonstrated that the LAA can often be a potential trigger for AF recurrences after catheter ablation (6). The potential importance of electrical isolation of the LAA is not surprising, considering that the LAA has the same embryological origin as the entire posterior LA and that its tissue characteristics may lead to AF initiation in a similar way as the pulmonary veins (7).

LAA occlusion

Surgical LAA strategies are divided into epicardial and endocardial approaches. Endocardial surgical techniques, applied during open heart surgery, are suture-based and have not proved to be an effective LAA therapeutic strategy. In addition, long-term occlusion is debatable (8) and the impact on LAA electrical activity is presumably non-existent. This is attributed to the survival of the LAA due to continual myocardial blood supply, and moreover, as the LAA base is not subjected to mechanical occlusion. Epicardial approaches are composed of suture excision, stapler excision, suture ligation and device-enabled exclusion [Atriclip® (Atricure) and Tigerpaw® (Maquet)]. Electrical isolation is established in the setting of excision, as LAA amputation ensures its electrical silence. As for suture ligation, we can only assume that a tight ligation will lead to LAA fibrosis and death over time. However, this has never been documented, and the

acute effect of this therapy remains to be proven. Device-enabled LAA exclusion results in electrical isolation, as demonstrated in one case report (9) and a case series using the Atriclip® device (10). In the latter, 100% successful exclusion and acute electrical isolation of the LAA were demonstrated in the surgical setting after application of the Atriclip device. Evidence that this isolation persists comes from pre-clinical work, with this device showing complete LAA fibrosis and scarring six months post application in the baboon (2). While electrical isolation has yet to be formally documented with application of the Tigerpaw® device, there may be potential for similar benefit with this device, given similarities in mechanical properties and footprint.

In contrast, the endocardial catheter-based approach with devices such as the Watchman® (Atritech, Plymouth, Minnesota, USA) and Amplatzer® cardiac plug (AGA medical, Plymouth, Minnesota, USA) may possibly prevent LAA clots from entering the left atrium, but have not documented electrical isolation of the LAA. In fact, these devices in the LA will make it literally impossible to ablate an LAA source after its placement, due to the external disc covering the targeted area to be ablated.

Is LAA amputation the reason why surgical ablation is more effective than catheter ablation?

Catheter ablation is an effective therapy for the treatment of drug-refractory paroxysmal AF, and as such, pulmonary vein isolation (PVI) is considered a mandatory cornerstone in any AF ablation procedure. When comparing catheter-based PVI to stand-alone surgical PVI, the FAST trial demonstrated a difference in favor for the surgical approach (11). At 12-month follow-up, freedom from AF was 36.5% for catheter ablation and 65.6% for surgical ablation (P=0.0022). The main difference between these groups was that the LAA was amputated during surgical ablation in nearly every case, whereas the LAA was not at all addressed or occluded in the catheter-treated group. Obviously two different techniques for ablation are used, but assuming that entrance and exit block was demonstrated according to the guidelines in the periprocedural setting, the difference between these two similar populations raises further questions. Thus, the role of the LAA as an electrophysiological target becomes more apparent than ever.

Discussion

The primary reason for occluding the LAA in the setting of AF is to eliminate stroke risk due to clot formation within the LAA. This has been demonstrated by the Protect AF trial (1) and though the percutaneous devices are far from perfect, the results obtained with them are excellent and clinical acceptance is wide and growing. However, recent reports suggest that the LAA could be a source of AF triggers in up to 30 percent of patients after catheter PVI alone (6). As electrical isolation is the cornerstone of any ablation, silencing the LAA may be crucial in certain patients. Considering the number of first time failures after catheter ablation due to LAA triggers, the additional benefit of isolation of the LAA provided by epicardial surgical approaches, such as the Atriclip (10), becomes apparent, in addition to stroke prevention. It is our belief that once more, the heart-team approach will enable a tailored therapy in the setting of an invasive rhythm control strategy and lead to better outcomes by offering not only innovative ablation strategies but also effective LAA therapies.

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