

Feasibility of Catheter Ablation of Atypical Atrial Flutters

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Atypical atrial flutters (AAFL) are difficult-to-manage atrial arrhythmias, yet potentially amenable to effective radiofrequency catheter ablation (CA). However, data on CA feasibility are only sparingly reported in the literature in different clinical settings, such as AAFL related to surgical correction of congenital heart disease.

atypical atrial flutter

atrial fibrillation

catheter ablation

1. Introduction

Atypical atrial flutters (AAFL) are complex cardiac arrhythmias that often involve re-entrant circuits related to atrial scarring and areas of slow conduction ^[1]. They may arise from the right as well the left atrial chamber ^[1]. Differently from the typical atrial flutter (AFL) where the electrical wavefront revolves around the cavotricuspid isthmus, the key to diagnosing AAFL is the reconstruction of the re-entry course by mapping and clear identification of the area critical to the re-entrant circuit.

AAFL may be observed in different clinical scenarios ^[1] spanning from patients who have undergone surgical correction of congenital ^[2] and acquired valvular heart disorders ^[3] to patients that have underwent non-surgical ablation of atrial fibrillation (AF) ^[4] and may even occur in apparently normal hearts ^[5].

Whatever the underlying structural heart disease, the greater the complexity of the pathophysiological substrate, such as in case of intra-atrial reentrant tachycardias (IART) in patients with congenital heart disease (CHD), the more difficult the clinical management. In fact, by promoting slow conduction through ion channel blockade, antiarrhythmic drugs (AAD) may even paradoxically enhance the risk of arrhythmia maintenance with potentially troublesome clinical consequences in the affected patients ^[6]. For this reason, catheter ablation (CA) has emerged as a potentially definitive treatment option for palpitations, heart failure, and even sudden cardiac death ^[2]. However, the associated long-term maintenance of sinus rhythm (SR) may be disappointing despite the implementation of cutting-edge technologies in this field ^[7].

2. Clinical Settings Associated with Atypical Atrial Flutters

2.1. Surgical Correction for Congenital Heart Disease

Macro-reentrant atrial arrhythmias or post-incisional IART represent common complications after surgical correction for congenital heart disease [8]. IART generally develops in adulthood several years after surgery and is often poorly tolerated in these patients [2]. Cavo-tricuspid isthmus-dependent AFL is seen in at least 58% of patients after cardiac surgery [2][9], whereas IART occurs in up to 25% of cases [2]. On the one hand, anatomical position of surgical scars deeply influences IART location. In patients with a history of atrial septal defect (ASD) and Tetralogy of Fallot repair, the observed macro-reentrant circuits revolving around areas of dense scar or through electrical gaps along double potential lines are generally consistent with the right-sided location of surgical atriotomies [2]. Re-entry around septal patch and left-sided IART have been also observed in rarer cases after ASD correction [2][10]. The electrophysiology substrate is even more complex when Fontan procedure for univentricular hearts is considered [11]. Due to the major hemodynamic abnormalities in these patients, the anatomical location of IART is difficult to predict and depends on the combination of iatrogenic areas of conduction block in heavily remodeled right atrial chambers [11]. However, the classic Fontan (i.e., right atrial to pulmonary artery anastomosis) and the intracardial lateral tunnel were more recently replaced by the so-called extracardiac Fontan where completely external conduits are used. Thanks to a total cavopulmonary connection created through right atrial bypass, the extracardiac Fontan operation has progressively led to a significant reduction in IART occurrence in these patients [12]. In this complex scenario, the implementation of three-dimensional electroanatomic mapping systems proved invaluable in the better understanding of the pathophysiological substrates of these cardiac arrhythmias [11].

2.2. Cardiac Surgery for Acquired Heart Disease

Cardiac surgery for the correction of mitral valve (MV) disease is common and associated with the development of complex, macro-reentrant arrhythmias revolving around iatrogenic scars [13][14]. In this setting, AAFL is observed in up to 55% of cases [3] and their anatomical location is greatly influenced by atriotomies and cannulation sites performed at the time of surgery [13]. Three major atriotomies have been described for surgical correction of MV disease, as follows: (1) left atrial atriotomy as an incision between the right pulmonary veins and the interatrial septum (Waterston's groove) (**Figure 1A**); (2) Guiraudon's approach or superior trans-septal access involving a vertical right atriotomy extended over the superior right atrium, the septum, and the dome of the left atrium (**Figure 1B**); and, finally, (3) combined trans-septal approach consistent of a vertical right atriotomy parallel to the atrio-ventricular sulcus (**Figure 1C**) followed by a separate incision in the interatrial septum (**Figure 1D**) [15].

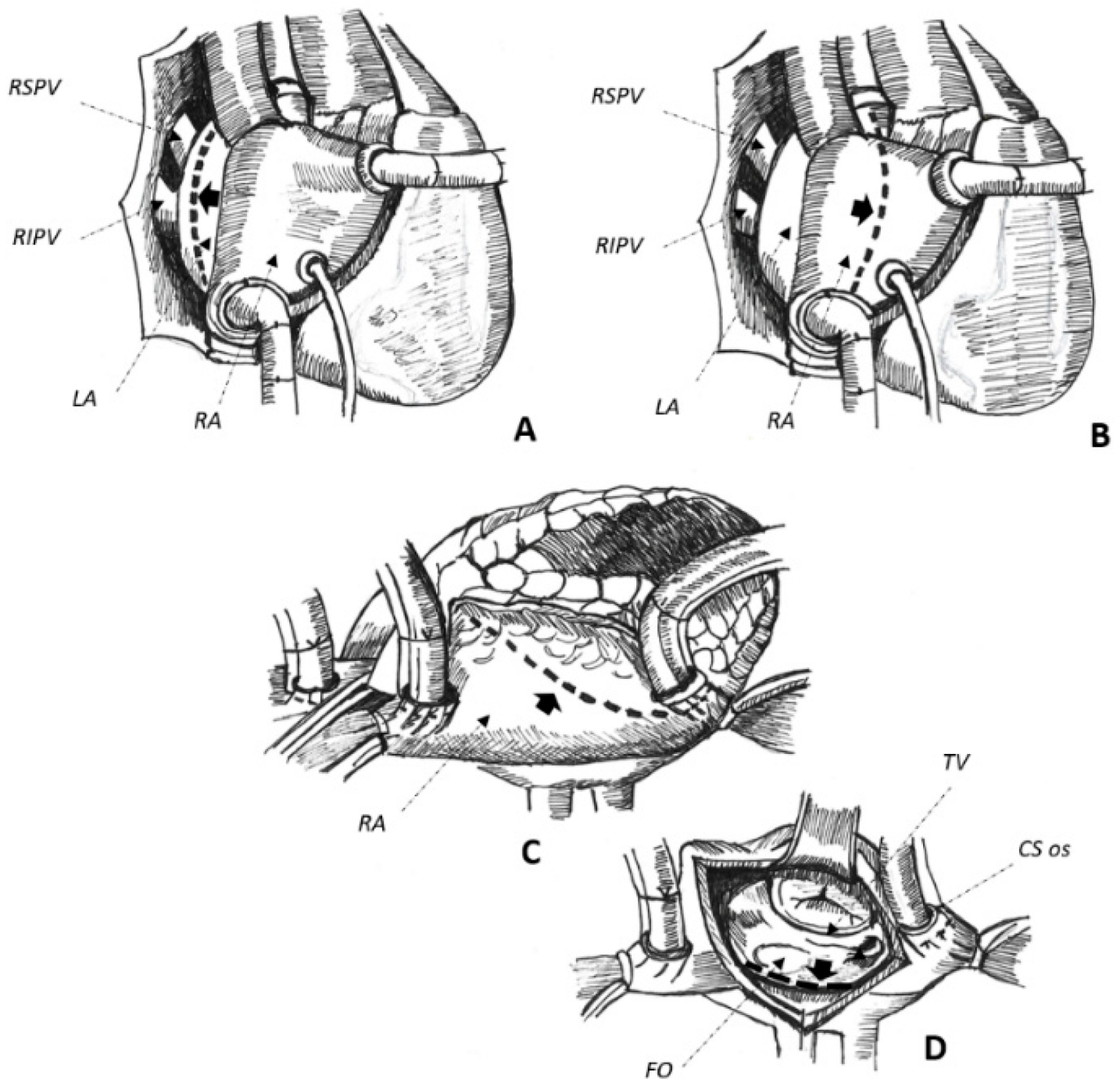


Figure 1. (A–D). Schematic representation of surgical approaches to get access to the mitral valve. Left atrial approach (A), superior trans-septal (B), and combined trans-septal approaches, the latter combining a right lateral atriotomy (C) with a trans-septal one (D) to expose the mitral valve. Atriotomies are reported by interrupted lines marked by thick black arrows in the figure. See text for further details. CS coronary sinus, FO fossa ovalis, LA left atrium, RA right atrium, RIPV right inferior pulmonary vein, RSPV right superior pulmonary vein, TV tricuspid valve.

2.3. Non-Surgical Pulmonary Vein Isolation

AAFL are common after non-surgical PVI [16]. Independent of the energy source used [17][18], the incidence of AAFL after PVI varies depending on the chosen ablation strategy: from less than 4% after ostial or antral PVI [19][20] to

31% for circumferential pulmonary vein ablation (CPVA) technique [21]. Deployment of ablation lesions in the left atrium is associated with an even greater incidence of AAFL occurrence [4]. Although macro-reentry is the predominant mechanism including left atrial roof and mitral-isthmus-dependent circuits [20], a non-negligible cause of post-PVI AAFL is represented by “small loop” or localized re-entry [4] due to gap-related mechanisms involving reconnected PV [4][20][22]. Although true focal arrhythmias have been rarely described in this setting [23], re-entry as small as 1 cm in diameter could nonetheless be observed after PVI [24]. These circuits usually display multiple slow-conducting channels along their course [25] that show remarkably long fractionated diastolic potentials lasting up to 140 milliseconds [4]. For this reason, at the end of the index CA, pulmonary veins should always be tested for persistent isolation, and ablation lines evaluated to identify conduction gaps. Broadly speaking, testing for electrical isolation requires, at a minimum, validation of an entrance conduction block [26], however, high voltage pacing in the PV may also be considered to evaluate a full bidirectional conduction block through the ablated PV [26].

2.4. Absence of Manifest Structural Heart Disease

In up to 6% of cases, AAFL occurs in patients with no evidence of structural heart disease [5]. Reasons for spontaneous atrial scarring are not clear. However, chronically increased atrial pressure overload in hypertension, occlusion of small coronary artery branches, isolated inflammation, and finally amyloid infiltration may explain an arrhythmogenic substrate in otherwise apparently healthy individuals [5][27]. Most of these circuits are right-sided and usually involve electrical silent areas located at the posterior or the lateral free wall of the right atrium, which can be effectively treated by radiofrequency energy applications delivered from these scars to the inferior vena cava ostium [5][28]. However, narrow and slow-conducting channels may also be found in left-sided, antero-septal circuits, as the result of the complex interweaving of epicardial fibers promoting AAFL [5]. Finally, even in normal hearts, transverse conduction across the crista terminalis [27] and the complex anatomy of the interatrial septum [29] may lead to macro-reentrant arrhythmias due to mechanisms of non-uniform anisotropy [27][30].

3. Overall Peri-Procedure Feasibility

Although pioneering works based their CA strategy on conventional mapping through the systematic evaluation of transient concealed entrainment and post-pacing intervals at different pacing sites [10][31][32], the feasibility of entrainment is known to be limited due to pacing-mediated arrhythmia termination, degeneration into AF, or increased pacing thresholds in patients on antiarrhythmic medications [28][33][34][35]. Moreover, AAFL are complex arrhythmogenic circuits sustained by double or multiple loops in up to 60% of cases, which would make an ablation strategy based on conventional mapping particularly challenging [10]. To overcome these issues, three-dimensional electroanatomic mapping systems have been progressively implemented in cardiac electrophysiology to guide mapping [33][35][36] and to achieve effective radiofrequency ablation of these complex circuits [7][9][34][35][37][38][39]. In fact, when these systems are used, the peri-procedure success rate spans from 65% [40] to 100% [9][37][39][41], with better results observed in patients with a history of non-surgical PVI or in case of no structural heart disease [36][41][42].

However, despite the implementation of the latest technologic developments in experienced hands, such as high-density mapping tools [43][44] or contact-force sensing catheters [44], peri-procedure failure is observed in up to 15–20% of cases [43][44], with a greater chance of acute failure in patients with history of surgical correction for CHD [40]. Difficult-to-ablate anatomical substrates [10], peculiar features of the targeted isthmi [45][46], and their anatomical locations [35][47], may explain failures. Further, a CA procedure may also be prematurely interrupted for safety issues to avoid right hemidiaphragm palsy [32], inadvertent block of the atrioventricular node [35], or atrial wall perforation with possible cardiac tamponade. Finally, the inherent complexity of CA of AAFL is proved by the reported long procedure [48] and fluoroscopy times [33].

As for the overall peri-procedure safety, local complications may occur in up to 7% of cases, including groin hematoma (up to 7%) [48], arteriovenous fistula (3–4%) [35][45], and femoral pseudoaneurysm (1.4%) [31] in generally anticoagulated patients. On the other hand, regarding systemic complications, cerebral [4][33] and peripheral [32] embolism could be as high as 4–6% with potentially life-threatening major bleedings only sparingly described, including retroperitoneal hemorrhage (2.2%) reported in one study only [3]. Finally, patients with mechanical valve prostheses may portend even a greater risk of peri-procedure thromboembolic or hemorrhagic complications. Therefore, particular attention should be paid to periprocedural antithrombotic regimens in this patient population to avoid potentially life-threatening events [14].

4. Maintenance of Sinus Rhythm after a Successful Procedure

AAFL recurrence is observed in up to 62% of cases after a single CA procedure with an overall SR maintenance as low as 38% on/off AAD after a variable follow-up duration, spanning from 7 ± 3 [43] to 37 ± 15 [5] months. Data on whether patients were on AAD before the procedure and at follow-up was not available in most of the studies, and the effect of AAD is therefore unclear in this setting.

The older the publication date, the greater the incidence of arrhythmia recurrence. This would suggest that the recent implementation of dedicated mapping tools [39] and irrigated-tip catheters [37][49] could help the cardiac electrophysiologist to achieve a greater long-term SR maintenance after an initially successful CA procedure [39][45]. The adoption of dedicated, tachycardia-oriented strategies for mapping and ablation of AAFL seem associated with even better results [35][45]. However, the greater the complexity of the atrial substrate to ablate, the higher the incidence of arrhythmia recurrence at follow-up. The worst long-term clinical outcome is commonly seen in patients with surgically corrected CHD (46–52% AAFL recurrence) [31][32], with better results observed after PVI (16–28%) [19][36] or in patients with apparently normal hearts (9–25% of tachycardia recurrence) [5][28].

5. The Winding Path to Improve the Procedure and the Overall Clinical Outcome

AAFL are typically sustained by critical *isthmi* anatomically [33] and functionally [4][35][48][50] defined. Regardless of the underlying structural heart disease and/or prior iatrogenic scars, these anatomical regions are bounded by anatomical/functional barriers and are associated with low bipolar voltages, fragmented electrograms [48][51], slow conduction velocity [45], and a typical mid-diastolic activation during ongoing tachycardia [10], which make these regions amenable to effective radiofrequency ablation [35].

For these reasons, as already described elsewhere [35][45], the integration of electro-anatomical information provided by these systems with surface and intracavitary signals would allow for the straightforward identification of the mid-diastolic isthmus amenable to radiofrequency ablation for effective arrhythmogenic substrate elimination. However, to avoid any misleading interpretation of the underlying circuit, accurate and high-density mapping of investigated atrial chambers is required to account for almost 90% of the tachycardia cycle length and thereby avoiding missing mapping segments potentially due to non-annotated low-amplitude and fragmented electrograms as low as 0.03–0.05 mV [25]. In this setting, the recent development of new tools, such as Octaray™ system (Biosense Webster Inc., Irvine, CA, USA) and the Ensite™ Omnipolar Technology (OT) (Abbott, Chicago, IL, USA) might allow for even better results [52][53][54], provided that the multitude of signals collected is correctly acquired and interpreted.

Although the implementation of these new technologies in CA procedures seems helpful in most cases, including mapping of re-entrant circuits with multiple loops [35][55], CA of the mid-diastolic isthmus may still be challenging due to its anatomical location and extension [47].

The ablation of roof-dependent circuits may be particularly challenging. The myocardial musculature surrounding the superior PV is generally thick and consistently displays adipose tissue separating the septopulmonary from the more endocardial septoatrial bundles [56]. This complex interweaving of myocardial fibers and their epicardial course may lead to non-transmural lesions and, thereby, to CA failure [57].

Regardless of the location of the tachycardia circuit or length of the ablative lesion, bidirectional conduction block does represent the essential endpoint of every CA procedure by evidence of detouring of the electrical wavefront around an anatomical barrier or scar through dedicated pacing maneuvers and/or demonstration of double potentials along the performed ablation lines. In some particular cases, demonstration of conduction block with the abovementioned criteria can be difficult and, therefore, complete disappearance of electrical signals at the target site can be considered a surrogate endpoint.

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